Characteristics of *Rhizoctonia solani* and Binucleate *Rhizoctonia* Species Causing Foliar Blight and Root Rot on Table Beets in New York State

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ABSTRACT

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A total of 107 isolates of Rhizoctonia solani and binucleate Rhizoctonia species were collected over a 2-yr period from naturally infected roots, petioles, and leaf tissues of table beets (Beta vulgaris) as well as directly from hymenia of the teleomorph, Thanatephorus cucumeris, on petiole and crown tissues. Ninety-eight of these isolates were found to be multinucleate, with diagnostic characteristics of R. solani (T. cucumeris), while the remaining nine isolates originating only from root tissues were binucleate Rhizoctonia species. About 88% of the R. solani isolates belonged to anastomosis group 2-2 (AG-2-2), and the remainder were either AG-5, AG-4, or AG-2-1. Binucleate isolates had a slower growth rate on PDA at 28 C than multinucleate isolates. Although isolates of AG-2-2 originated from different tissues, their average growth rate on PDA and colony characteristics were similar. The AG-2-2 isolates from roots and petioles were highly to moderately virulent to leaves and were highly virulent to root tissues of table beets. The AG-4 and AG-5 isolates were low to moderately virulent to leaves but highly virulent to roots. The binucleate isolates were weakly virulent to both leaves and roots. There was no apparent correlation between the growth rate of the R. solani isolates and their virulence to either roots or leaves of table beets. A widespread strain of R. solani and its teleomorph are causing both the foliar blight and the root rot diseases of table beets in central New York.

Table beets (Beta vulgaris L.) have been grown for many years in the vegetable production area of central New York, but the annual hectarage has been declining due largely to the economics of production and the prevalence of diseases. In 1990, table beets were grown on 931 ha, resulting in a yield of 36,674 t for a farm value of \$2.2 million (13). Most of the table beets produced are used

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for processing, and only limited quantities are sold for the fresh market.

Root rots are considered to be the most important diseases of table beets, as they occur frequently and reduce both yield and quality (1). Rhizoctonia solani Kühn and Pythium ultimum Trow are the main causal agents of root rots in New York. These pathogens are also capable of causing seed decay, pre- and postemergence damping-off, wire stem, and malformed fleshy roots with external or internal rot symptoms.

In addition, R. solani causes foliar infections on table beets, resulting in the pocket rot disease syndrome that has

become important in recent years throughout the table beet production areas in New York State (16,17). This disease appears as randomly scattered pockets (foci) of infected plants within a field. Infected plants eventually die, resulting in open areas within the planting row. Furthermore, the teleomorph of R. solani, Thanatephorus cucumeris (A.B. Frank) Donk, was observed under field conditions for the first time in New York on the crown and lower part of the petioles of table beets during August-September 1990 (17). The occurrence of the teleomorph under field conditions may explain the frequent, severe occurrence of foliar blight epidemics on table beets in New York (16). Both the teleomorph and anamorph states are capable of causing aerial blights under warm, moist conditions on several crops, including sugar beets, bean, soybean, cotton, and tobacco (3,6,7,11,27,31).

Rhizoctonia species are worldwide in distribution with an extremely wide host range, and considerable morphological, pathological, and physiological variability exists among the species as well as in the diseases on different host crops (2,5,14). Subspecies of Rhizoctonia are generally characterized by hyphal anastomosis and differences in morphology, pathogenicity, physiology, cultural appearance, and ecology (2,14,20,25). The objective of this study was to characterize the isolates of R. solani and binucleate Rhizoctonia species associated with foliar blight and other foliar infections on table beets in New York State and to compare them with isolates originating from roots.

MATERIALS AND METHODS

Collection, isolation, identification, and storage of isolates. Isolates of Rhizoctonia species were obtained from infected leaves, petioles, and roots of table beets collected from seven fields throughout the commercial production areas in central New York during a 2yr period (1988-1990). Small pieces of root or leaf tissue were cut from the margin of expanding lesions, surfacesterilized for 1-2 min in 5% sodium hypochlorite, rinsed with sterile distilled water for 1-2 min, and then placed on acidified potato-dextrose agar (APDA) or water agar (WA). Isolates were also obtained directly from hymenia of T. cucumeris (anamorph: R. solani) from the surface of the crown and lower petiole tissues (17). Small pieces of hymenial layers were excised from beet tissues and placed on petri dishes containing APDA. After 24-36 hr, hyphal tip transfers were made to APDA from colonies showing mycelial characteristics of Rhizoctonia species such as branching at right angles, constriction of the hypha at the point of origin or union with the main hyphae, and presence of dolipore septae (20). One set of isolates was stored in screw-capped glass tubes containing PDA and maintained at 5 C in the dark and transferred once every year. A second set was stored as dried colonized beet seeds placed in sterilized screw-capped glass tubes and maintained at 5 C in the dark.

Nuclear condition. Nuclei were stained by treating 2-day-old hyphal strands of *Rhizoctonia* growing on WA with a solution (1 μ g/ml) of 4',6'-diamidino-2-phenylindole (DAPI) (25). Fluorescent nuclei were counted at 400× magnification using a Zeiss photomicroscope equipped with epifluorescence equipment using an excitation filter G365, dichromatic beam splitter FT 395, and barrier filter LP 420 (9). The number of nuclei per isolate was based on the average of 15 observations.

Anastomosis groups. The anastomosis group of each isolate of *Rhizoctonia* was determined by pairing them with known tester strains (19). Isolates were paired on 0.6×1.0 cm pieces of cellophane on 2% WA in petri dishes. Mycelial disks 5 mm in diameter from a tester isolate and an unknown isolate were placed at opposite ends of the cellophane. Hyphal anastomosis was determined microscopically without staining after incubation for 24-48 hr at 21 C. Perfect fusion (fusion of cell wall and cytoplasm) and imperfect fusion (fusion resulting in plasmolysis) were considered criteria for a positive anastomosis reaction (4,15,25). At least five fusion sites were observed for each positive anastomosis.

Cultural characteristics. Color of colonies and sclerotia, production of

sclerotia, and growth rate of *Rhizoctonia* isolates were determined according to the following procedure (15). Mycelial disks 5 mm in diameter were transferred from the margin of 2-day-old colonies to the center of PDA plates. Five dishes were used for each isolate in each test. After 2 wk of incubation at 28 C in the dark, sclerotial formation and color of colonies and sclerotia were determined. Color was defined according to the color chart of the Royal Horticultural Society of London (21). A dissecting microscope was used to observe very small sclerotia.

The growth rate of the isolates was determined after 48 hr of incubation at 28 C in the dark using five dishes per isolate (15). The growth rate was measured again in a second test using only 10 isolates selected at random. A correlation analysis between data of the first and second tests of these 10 isolates was performed in order to predict if a repetition of the test using all *Rhizoctonia* isolates would show a similar behavior.

Pathogenicity and virulence. Foliar tissues. The table beet cultivar Ruby Queen was used throughout this study. Table beet seedballs were treated with the fungicides metalaxyl (Apron 25W, Ridomil) and thiram 42S (Arasan) at 1.4 and 3.8 g/kg of seeds, respectively. Twelve seedballs were planted per 10-cm clay pot filled with a pasteurized (70 C for 30 min) potting soil mix of field top soil (coarse sandy loam) and sand (builder's sand) (1:1, v/v). Six weeks after planting, seedlings were inoculated with an aqueous suspension of 1 g of mycelium per 100 ml of sterile distilled water (16). Controls were sprayed with sterile distilled water. All treatments were replicated five times (five pots). After 72 hr of incubation in a mist chamber at 28 C, seedlings were transferred to a greenhouse (22–28 C). The pathogenicity of each isolate to table beets was rated with a pretransformed disease rating scale of 1-9 (16) in which 1 = healthy plants with no visible disease symptoms, 1.1-3 = upto 10% of tissues affected (low disease severity), 3.1-6 = 11-40% of tissues affected (moderate disease severity), and 6.1-9 = 41-100% of tissues affected (high disease severity). Treatments were arranged in a completely randomized design. Data were submitted to analysis of variance, and differences among means were determined by least significant difference.

Root tissues. Inoculum of each isolate of Rhizoctonia species was prepared using the soil-potato medium of Ko and Hora (10), then mixed with potting soil mixture (field soil and sand, 1:1, v/v) at a rate of 2%, v/v. Twelve beet seedballs of the cultivar Ruby Queen were planted per 10-cm clay pot filled with pasteurized potting soil mixture and covered with a layer of 100 ml of Rhizoctonia-infested soil. Each isolate had five replications, and all treatments were arranged in a

randomized block design on greenhouse benches at 22-28 C. The number of emerged seedlings and the incidence of postemergence damping-off were recorded every day over a 2-wk period.

The pathogenicity and virulence of *Rhizoctonia* isolates to foliar and root tissues of table beets were measured again in a second test using only 10 isolates selected at random. A correlation analysis between data of the first and second tests of these 10 isolates was performed.

RESULTS

A total of 107 fungal isolates were collected from naturally infected table beet tissues over a period of more than 2 yr (Table 1). All the isolates showed the diagnostic mycelial characteristics described for Rhizoctonia (20,25). Seventy-four isolates (69.2%) were obtained from naturally infected foliage, while 33 isolates (30.8%) were obtained from infected root tissues (Table 1). Forty-eight of the Rhizoctonia isolates obtained from foliar tissues were recovered from sunken cankers on petioles, and 11 isolates were recovered from circular lesions on leaves. The remaining 15 isolates were directly transferred from hymenia of T. cucumeris present on the surface of petioles and crown tissues.

Nuclear condition and anastomosis groups. Ninety-eight isolates (91.6%) were multinucleate. The number of nuclei per vegetative cell varied from 4.6 to 13.7. The remaining nine isolates of Rhizoctonia, all of which were obtained from beet root tissues collected from three different commercial fields, were binucleate (Table 1). None of the R. solani isolates tested in this study failed to anastomose (Table 1). The majority of R. solani isolates from foliage (67 isolates) belonged to AG-2-2, whereas five were in AG-4 and two were in AG-2-1 (Table 1). Isolates of R. solani originating from hymenia of T. cucumeris on petioles and those isolated from lesions on leaves were all AG-2-2. Nineteen (58%), four (12%), and one (3%) of the table beet isolates of Rhizoctonia from roots were AG-2-2, AG-5, and AG-4, respectively (Table 1); the remaining nine binucleate isolates obtained from roots were not tested for anastomosis reaction against any of the reported binucleate tester isolates.

Cultural characteristics. The color of vegetative hyphae of the AG-2-2 isolates of *R. solani* from petioles varied from yellow-white to brown, but they were mainly yellow-white or gray-brown. AG-2-2 isolates of *R. solani* from roots were principally brown, and those from hymenia of *T. cucumeris* and lesions on leaves (AG-2-2) were mainly gray-brown. AG-2-1 and AG-4 isolates of *R. solani* from petioles were yellow-white to orange-white, while AG-5 isolates ranged

from gray-white to yellow-white. Binucleate isolates were yellow-white (16).

After 2 wk of incubation at 28 C, most R. solani isolates produced sclerotia, but AG-2-1 isolates from petioles and the binucleate isolates from roots did not. The size of the sclerotia of R. solani AG-2-2 isolates from petioles ranged from <1 mm to >2 mm, and sclerotial aggregates were commonly observed. AG-4 isolates of R. solani produced small sclerotia (about 1 mm) and did not form aggregates. Sclerotia of the AG-2-2 isolates from roots were similar to those of AG-2-2 from petioles. Sclerotia of isolates AG-5 of R. solani were usually small and sparse and formed aggregates (16).

Rhizoctonia isolates collected from table beets differed significantly (P =0.01) in their growth rate on PDA (Table 1). Isolate 549 (AG-2-2) from roots was the fastest growing isolate, with an average of 7.2 cm/48 hr, while isolate 586 (AG-2-2) from petioles was the slowest growing, with an average of 1.1 cm/48 hr. The overall growth rate average for the AG-2-2 isolates of R. solani was approximately 4.4 cm/48 hr and did not vary among the isolates obtained from petioles, roots, or leaf tissues or directly from hymenia. AG-4 isolates from beet petioles grew an average of 5.1 cm/48 hr. Binucleate isolates had a slower growth rate, averaging 3.5 cm/48 hr. AG-5 isolates from beet roots exhibited an intermediate to high growth rate, averaging 5.1 cm/48 hr (Table 1). Similar results were obtained in the growth rate of 10 Rhizoctonia isolates selected at random in a repetition of this test (R = 0.97). This high coefficient of correlation indicates that a repetition of the test with all the isolates of Rhizoctonia would show a similar behavior.

Pathogenicity to foliar and root tissues. Highly significant variation was observed in the virulence of the isolates to leaf and petiole tissues of table beets (Table 1). Although all of the isolates were pathogenic, disease severity ratings ranged from 1.6 (binucleate isolate 580) to 9 (AG-2-2 isolates 612, 621, 666, 667, and 676). All uninoculated plants remained healthy (Table 1). Isolates of AG-2-2 of R. solani obtained from beet petioles were mostly moderately to highly virulent to foliar tissues of table beets (disease severity rating from 3.1 to 9). The AG-2-2 isolates obtained from beet roots did not differ in virulence from those obtained from petiole tissues. Isolates of R. solani obtained directly from hymenia and lesions on leaves were highly virulent to foliar beet tissues (disease severity rating from 6.1 to 9). However, binucleate isolates were low in virulence to foliar beet tissues (disease severity ratings <3). There was no correlation between the growth rate of the isolates of R. solani and their virulence

Table 1. Characteristics of *Rhizoctonia solani* and binucleate *Rhizoctonia* obtained from table beets grown in central New York State during 1988-1990

Plant tissue Isolate	Nuclei per cell ^a	AG group	Disease severity rating		Mean growth rate ^t
			Foliage ^c	Root ^d	(cm/48 hr)
Petioles					
621	9.7	2-2	9.0	91.0	4.9
612	7.1	2-2	9.0	94.6	5.5
619	11.7	2-2	8.8	82.9	4.2
620	9.0	2-2	8.6	98.2	4.6
582	8.0	2-2	8.4	77.5	4.3
613	10.1	2-2	8.4	98.2	5.7
513	9.2	2-2	8.4	81.1	6.0
		2-2 2-2	8.2	86.5	6.3
520	9.2			88.3	6.1
514	9.1	2-2	8.2		
510	6.7	2-2	8.0	91.0	3.9
615	8.4	2-2	7.8	52.2	5.0
614	9.0	2-2	7.8	93.7	4.4
594	6.4	2-2	7.2	86.5	3.9
519	8.2	2-2	6.8	81.1	6.3
587	9.6	2-2	6.6	73.7	2.5
595	9.2	2-2	6.6	80.2	3.7
598	7.2	2-2	6.4	100.0	4.9
581	9.0	2-2	6.2	78.2	4.3
576	11.1	2-2	6.2	82.0	3.1
585	9.9	2-2	6.0	91.9	4.6
627	7.9	2-2	6.0	71.2	4.1
588	6.8	2-2	5.8	18.0	2.6
575	4.9	2-2	5.8	77.5	3.7
604	8.4	2-2	5.6	99.1	3.7
		2-2	5.6	100.0	5.9
600	9.1				4.2
618	11.7	2-2	5.6	91.0	
611	7.8	2-2	5.2	85.6	4.8
606	7.9	2-2	5.2	91.9	3.3
625	13.7	2-2	5.0	74.8	4.7
628	10.4	2-2	5.0	78.4	4.4
629	9.2	2-2	4.8	89.2	4.8
586	9.3	2-2	4.8	82.7	1.1
577	7.0	2-2	4.8	52.3	3.6
605	5.4	2-2	4.4	98.2	3.6
515	6.8	4	4.2	96.4	5.9
599	9.4	2-2	4.0	95.5	5.9
609	8.1	2-2	3.8	99.1	4.0
626	8.0	2-2	3.8	82.9	5.4
622	13.2	2-2	3.6	97.3	4.6
616	10.2	2-2	3.6	83.8	3.5
630	10.2	2-2	3.4	96.4	5.6
518	8.3	4	3.4	93.7	5.1
	7.3	4	3.2	92.8	4.3
608		4	2.6	94.6	6.0
517	10.5		2.6		4.4
623	7.4	2-1		86.5	
624	10.7	2-2	2.2	77.5	6.0
516	6.7	4	2.2	92.8	5.7
617	9.1	2-1	1.8	94.6	6.0
Roots					
589	6.0	2-2	8.6	92.8	3.3
597	8.8	2-2	8.4	100.0	5.9
521	8.8	2-2	8.2	97.3	5.9
511	8.7	2-2	7.6	91.9	3.0
610	6.9	2-2	7.4	81.1	4.9
578	8.2	2-2	7.0	74.6	3.4
601	9.7	2-2	7.0	96.4	4.3
584	7.0	2-2	6.8	82.0	3.3
	7.0 7.8	2-2	6.6	98.2	5.0
506					
596 512	7.3	2-2	6.6	86.5	4.0

^aDetermined in mature hyphae from active cultures growing on water agar. Each number is the average of 15 observations per isolate.

(continued on next page)

^bAverage of five replicates rounded off to one decimal number.

^cDetermined on a pretransformed scale of 1-9 in which 1 = healthy plants and 9 = 100% of tissue affected.

^dTotal percentage of root disease incidence (pre- and postemergence damping-off) recorded 14 days after inoculation.

^cIsolates obtained from circular lesions probably caused by basidiospores of *Thanatephorus* cucumeris.

^f Isolates obtained from hymenial layers of *T. cucumeris* on petioles of table beets.

Table 1. (continued from preceding page)

Plant tissue	Nuclei	AG	Disease severity rating		Mean growth rate	
Isolate	per cell ^a	group	Foliage ^c	Root ^d	(cm/48hr)	
591	13.2	2-2	6.6	67.6	4.0	
593	7.8	2-2	6.4	84.7	3.5	
583	5.8	2-2	6.4	81.1	5.1	
603	7.2	2-2	6.2	93.7	4.8	
590	9.8	2-2	5.6	81.1	4.6	
550	7.5	5	5.0	91.9	4.6	
569	5.0	5	5.0	77.5	5.8	
548	8.4	2-2	5.0	91.0	6.0	
579	4.6	2-2	4.4	78.4	3.1	
549	10.0	2-2	4.2	99.1	7.2	
552	2.0	В	4.2	12.6	3.1	
602	12.2	2-2	3.8	84.7	4.4	
571	2.0	В	3.6	11.7	4.6	
551	5.7	5	3.6	90.8	4.5	
572	2.0	В	3.4	19.8	4.2	
567	6.0	5	2.8	64.9	5.5	
607	6.9	4	2.6	78.4	4.7	
592	2.0	В	2.6	9.7	3.9	
573 574	2.0	В	2.6	10.8	4.2	
574 570	2.0	В	2.2	14.2	1.3	
570	2.0	В	2.2	18.0	3.4	
568	2.0	В	1.8	8.6	2.5	
580	2.0	В	1.6	9.9	4.2	
Leaves ^e	7.2	2.2	0.0	00.1	4.6	
676 666	7.3	2-2	9.0	88.1	4.6	
667	8.9	2-2 2-2	9.0	90.1	4.7	
672	7.8 6.1	2-2 2-2	9.0	98.2	4.6	
671	6.1	2-2 2-2	8.8	83.8	4.3	
673	7.6	2-2 2-2	8.8 8.6	88.3	3.8	
669	7.0	2-2 2-2	8.4	71.1 91.7	5.0	
675	8.1	2-2	8.4 8.4	88.3	4.3 5.4	
670	8.0	2-2	7.4	88.5	3.4	
668	8.9	2-2	6.8	73.7	3.7	
674	7.4	2-2	5.6	89.1	4.2	
Petioles ^f	7	2-2	5.0	07.1	4.2	
665	8.5	2-2	8.8	100.0	4.2	
659	7.5	2-2	8.8	88.3	4.6	
660	7.7	2-2	8.8	89.2	4.7	
663	9.6	2-2	8.4	90.1	5.2	
656	9.1	2-2	8.4	98.2	4.2	
661	9.1	2-2	8.2	98.1	4.6	
662	10.8	2-2	7.6	95.5	4.0	
658	7.8	2-2	7.2	89.2	4.3	
664	7.4	2-2	6.8	73.0	4.0	
652	8.5	2-2	6.8	83.8	4.7	
657	8.6	2-2	6.6	64.8	4.6	
653	6.9	2-2	6.4	20.1	3.2	
655	9.4	2-2	4.6	47.5	4.7	
654	9.4	2-2	3.4	84.7	3.2	
651	8.4	2-2	2.2	66.6	3.3	
LSD (0.05)			1.0	13.1	0.5	

to foliar tissues of beets.

There were highly significant differences in the virulence of the isolates to root tissues of table beets as determined by the total incidence of pre- and postemergence damping-off (Table 1). All these isolates of Rhizoctonia were pathogenic. However, total disease incidence ranged from 8.6 to 100% (Table 1). The AG-2-2 isolates of R. solani from beet petioles, roots, and hymenial layers of T. cucumeris showed a high root disease incidence. In contrast, binucleate isolates were weakly virulent to beet roots. There was no correlation between the growth rate of the isolates of Rhizoctonia on PDA and their virulence to roots of table beets. Similar results were obtained when 10 Rhizoctonia isolates selected at random were inoculated to foliar and root tissues of table beets in a repetition of these tests (R=0.98 and 0.93 for the pathogenicity and virulence to foliar and root tissues, respectively). These high coefficients of correlation indicate that a repetition of the tests with all the isolates of Rhizoctonia would show a similar behavior.

DISCUSSION

The majority (92%) of the isolates of *Rhizoctonia* obtained from naturally infected table beets in New York State were *R. solani* and its teleomorph, *T. cucumeris. R. solani* was first reported causing severe root rot of table beets in

New York State in 1953 by Natti (in Abawi et al [1]). Foliar blight of sugar beets caused by R. solani was first described by Kotila in 1947 (11). He reported that the latter disease was caused by a different strain of the fungus that had a distinct pattern of attack from those strains of R. solani causing crown, root, and dry rot of sugar beets. The occurrence of T. cucumeris on sugar beet under field conditions has also been observed by Herr (7). However, the role of basidiospores in disease development was not well understood (14,29). It was reported that foliar infections on sugar beets in Japan were caused by basidiospores of R. solani AG-2-2 (28). Naito and Sugimoto, cited by Ogoshi (14), found that foliar blight of sugar beet caused by R. solani AG-2-2 IV isolates was initiated by basidiospores, and they gave a detailed account of the infection process. Herr (7) reported the occurrence of hymenia of AG-2-2 on sugar beet in 1982. The occurrence of T. cucumeris on table beets was reported for the first time in New York by Olaya and Abawi in 1991 (17).

Isolates of R. solani and the teleomorph stage originating from table beet root, petiole, crown, and leaf tissues belonged principally to AG-2-2 (80%). Identified AG-4 and AG-2-1 isolates were collected mainly from beet petioles at low frequency (6 and 2%, respectively). AG-5 isolates were obtained only from beet roots at a low frequency (4%). Four of the 11 anastomosis groups reported in the literature were identified among these isolates of R. solani obtained from table beets grown in central New York State. Windels and Nabben (30) found six anastomosis groups (AG-1, 2-1, 2-2, 3, 4, and 5) among R. solani isolates obtained from sugar beets in Minnesota. The same anastomosis groups were also reported from sugar beets in Japan (Naito et al, cited by Windels and Nabben [30]). Herr and Roberts (8) reported AG-2 and AG-4 of R. solani in soils and weed samples from sugar beet fields. AG-2, AG-4, and AG-5 were reported to cause damping-off on sugar beets in Ireland (18). Ruppel (22) reported that AG-2 isolates were associated with root rot and AG-4 isolates were associated with crown and foliar diseases of sugar beets. Ui (28) reported that AG-1 and AG-4 isolates of R. solani were causing damping-off on sugar beet seedlings and AG-2-2 isolates were causing root rot. He also found that basidiospores of R. solani AG-2-2 (T. cucumeris) caused foliar blight infections.

The high recovery frequency of AG-2-2 isolates of *R. solani* from table beet roots, petioles, and leaves in central New York may be due to the limited crop rotation being practiced in this old production area. Komoto et al, reported by Ogoshi (14), demonstrated that the

rate of isolation of AG-2-2 isolates increased and the rate of isolation of AG-5 and AG-1 decreased with continuous cropping of sugar beets. In addition, Herr and Roberts (8) suggested that anastomosis groups of *R. solani* are also influenced by soil texture and thus may predominate in a specific geographic area. Furthermore, Ogoshi (14) stated that the cultivated crop exerts a strong influence on the prevalence of a given anastomosis group, as the host range of anastomosis groups is generally limited to a few crop families.

Isolates of R. solani AG-2 have been divided principally into two intraspecific groups, AG-2-1 and AG-2-2, according to the frequency of anastomosis. Two pathogenic types have been described within AG-2-2: AG-2-2 IIIB, pathogenic to mat rush (Juncus effusus L. var. decipiens), and AG-2-2 IV, pathogenic to B. vulgaris (14). Liu and Sinclair (12) placed isolates of R. solani AG-2 in five genetically distinct subgroups—2A, 2B, 2C, 2D, and 2E-based on isozyme polymorphism and DNA restriction analyses. Intraspecific group 2A corresponded to AG-2-1, 2B to AG-2-2 IIIB, 2C to AG-2-2 IV, 2D to AG-2-2, and 2E to a newly identified group known previously as AG-2-2 and related to AG-2-1 (12). However, Stevens Johnk et al (26) subsequently demonstrated by fatty acid analyses and anastomosis test that group 2E of Liu and Sinclair (12), which causes tobacco leaf target spot (23,24), is in fact AG-3.

The virulence of R. solani on table beets varied according to the anastomosis group and the organ of table beet inoculated. AG-2-2 isolates were the most virulent, whereas AG-4 and AG-5 were intermediate. However, other anastomosis group isolates of R. solani have the potential to cause significant infection and damage to table beets. For example, AG-5 isolates principally affect roots and are considered to be less aggressive than other anastomosis group isolates of R. solani (30). These isolates become more aggressive when plants are under stress by other diseases or unfavorable environmental conditions (30). The binucleates isolates were low in virulence to both the foliar and root tissues of beets. Binucleate isolates have been characterized as having low growth rate and low virulence to sugar beets and other crops (8,14,15). Excluding the binucleate isolates, there was no apparent correlation between the growth rate of the isolates and their virulence to either root or foliar tissues of table beets. A number of authors (5,6,15) have reported a strong correlation between these two variables, but Cardenas (3) did not detect such a relationship.

There are reported differences between foliar and subterranean isolates of R. solani, with foliar isolates having a faster growth rate, producing larger numbers of small sclerotia, and being more sensitive to carbon dioxide than the subterranean isolates (5,6). These characteristics allow the foliar and subterranean isolates of R. solani to be more adapted and aggressive to the plant part from which they were originally isolated. In this study, however, no differences were observed between the isolates of R. solani collected from foliar and root tissues of table beets. Foliar isolates were very similar to root isolates in their virulence to foliar tissues as well as to root tissues of table beets. The similarity in cultural morphology, pathogenicity, and other characteristics of the isolates of R. solani collected from foliar and root tissues of table beets may be the outcome of standard cultivation practices. During cultivation, considerable amounts of infested soil are being thrown onto petioles and crowns of table beets (16). Therefore, root and foliar tissues of table beets are exposed to the same inoculum source and strains of R. solani. We concluded that a widespread strain of R. solani AG-2-2 and its teleomorph are causing both foliar blight (pocket rot) and root rot of table beets in central New

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