Relationship Between Vegetative Compatibility and Pathogenicity of Isolates of *Fusarium oxysporum* f. sp. *tuberosi* from Potato

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ABSTRACT

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Thirty-five South African isolates of *Fusarium oxysporum* f. sp. *tuberosi*, which cause dry rot, stem-end rot, and wilt of potatoes (*Solanum tuberosum*), were characterized by pathogenicity and vegetative compatibility. The isolates were assigned to six vegetative compatibility groups (VCGs) according to pairings of complementary nitrate-nonutilizing (*nit*) mutants induced on chlorate-containing media. A complex pattern of association between disease symptoms caused in potatoes, VCG, and geographic distribution was found. For instance, 14 of the 16 isolates assigned to VCGs A and B were originally isolated from stem-end rot. Only one stem-end rot isolate was assigned to a different VCG. *F. o. tuberosi* isolates that were originally isolated from dry rot tubers were assigned to VCGs E and F. Isolates causing dry rot that were collected in the Transvaal Highveld, South Africa, belonged to VCG E, whereas isolates from the eastern Orange Free State, South Africa, were assigned to VCG F. Eight of the 10 isolates that were isolated from wilted potato plants were assigned to VCGs C and D. These isolates originated from several geographical locations. The remaining two isolates obtained from wilted potato plants were assigned to VCG A. Pathogenicity and vegetative compatibility reactions in VCGs A and C suggest that a relationship between isolates that cause stem-end rot and those that cause wilt of potatoes might exist.

Fusarium oxysporum f. sp. *tuberosi* W. C. Snyder & H. N. Hans. causes dry rot, stem-end rot, and wilt of potatoes (*Solanum tuberosum* L.) (16). For the breeding of *Fusarium*-resistant potato cultivars, a reliable identification method that distinguishes between the different *F. oxysporum* isolates that cause the three diseases is needed.

Snyder and Hansen (22) grouped strains of *F. oxysporum* into formae speciales on the basis of their host specificity. With few exceptions, strains of the fungus, whether from the same or different formae speciales, are morphologically indistinguishable. Formae speciales have been subdivided into physiological races that attack only certain cultivars of a host species (16). Puhalla (18) showed that the vegetative compatibility group (VCG) may be a handy tool for differentiating forma speciales of *F. oxysporum*. Fungal strains that are able to anastomose (form heterokaryons) with each other are considered vegetatively compatible and are assigned to a single VCG. Puhalla (18) first used auxotrophic, nitrate-nonutilizing (*nit*) mutants to establish a vegetative compatibility system in *F. oxysporum*. *Nit* mutants can be readily recovered in high frequencies by selecting for chlorate resistance (2,4,6,9,10,12). Puhalla (18) placed 21 strains of *F. oxysporum* into 16 VCGs. A correlation between VCG and formae speciales was observed.

Correll et al (3) refined the heterokaryon technique by demonstrating that *nit* mutants of *F. oxysporum* could be subdivided into at least three phenotypic classes. These classes could be differentiated by the ability of *nit* mutants to use various nitrogen sources. *F. oxysporum* has no known teleomorph, and genetic analysis is therefore not possible. Puhalla and Spieth (20) and Klittich and Leslie (13) discovered similar phenotypes among *nit* mutants of *F. moniliforme* J. Sheld., a closely related species with a teleomorph. Two of the phenotypic classes of *F. moniliforme* were single locus mutations of the gene for nitrate reductase and for the pathway-specific regulatory gene for nitrite reductase. These phenotypes were labeled with the genotype designations *nit1* and *nit3*, respectively (13,14,26). The third phenotype in *F. moniliforme* mapped to one of five loci required for the synthesis of the molybdenum cofactor, which is necessary for nitrate reduction and purine dehydrogenase (13,15). This phenotype was described in *Aspergillus nidulans* (Eidam) G. Wint. (7) and was called NitM (13). Correll et al (3) presumed similar mutations were likely to exist among the classes of *nit* mutants in *F. oxysporum*.

In this investigation, pathogenicity and vegetative compatibility tests from pairings of complementary *nit* mutants representing three different phenotypic classes were evaluated for their usefulness in differentiating among isolates of *F. o. tuberosi* that cause dry rot, stem-end rot, and wilt of potato.

MATERIALS AND METHODS

Isolates. Of the 35 *F. o. tuberosi* isolates that were selected randomly, 10 were isolated from potato tubers with dry rot, 15 were from stem-end rot lesions, isolated by the method of Theron and Holtz (24), and 10 were from wilted potato plants. Potato tubers with dry and stem-end rot lesions were sampled at sorting tables in the eastern Orange Free State (24 farms) and the Transvaal Highveld (27 farms), South Africa, during the summer of 1985–1986. Tubers were kept in paper bags at 5 C and 50–70% relative humidity (RH) until isolations were made (24). Wilted potato plants were collected from farms in the potato-producing areas where wilt symptoms occurred during the summers of 1985–1986 and 1986–1987. The plants were washed, surface-disinfected in 3% sodium hypochlorite for 5 min, and allowed to dry. Stems were cut in half, and five pieces were dissected randomly from the discolored vascular tissue of each stem. Each piece was placed on potato-dextrose agar (PDA) and incubated at 25 C for 4–5 days under fluorescent and black lights for a 12-h photoperiod. Single conidial isolates of *F. o. tuberosi* were obtained (24), transferred to carnation leaf agar (CLA) (11) and PDA, and incubated as described above for 10–14 days. We identified isolates by using the system of Nelson, Toussoun, and Marasas (17). All the isolates used were incubated on CLA slants and were also lyophilized.

Pathogenicity tests for Fusarium stem-end rot. A sterile sand-Pronutro (Nutri-product, Cerebos Foods, South Africa) growth medium of 300 cm^3 of washed sand, 100 cm^3 of Pronutro, and

100 cm^3 of distilled water (G. Holtz, *personal communication*) was prepared in 1,000-cm^3 Erlenmeyer flasks. Mycelial plugs (2 mm^3) were cut aseptically from the edge of a colony of each of the 35 *F. o. tuberosi* isolates, grown on CLA, and transferred to the sand-Pronutro growth media. The flasks, each inoculated with a different *F. o. tuberosi* isolate, were incubated for 4 wk at 25 C under lights as described before. The flasks were shaken daily to ensure even dispersal of the mycelium. Plastic pots (15 cm diameter) were filled with infested soil (950 cm^3 of soil/200 cm^3 of inoculum), fertilized at an amount equivalent to 1,100 kg/ha (3:2:1; N-P-K [25]), and then planted with sprouted potato tubers of the cultivar Vanderplank. The experiment was done in a greenhouse (25 ± 2 C, 12-h photoperiod) as a randomized block design with 15 replicates. The plants were irrigated daily and were grown to maturity. The progeny tubers were left until at least 3 wk after the foliage had died back and then lifted, and then the tubers were assessed for stem-end rot. Results from this pathogenicity test and the following two were statistically analyzed by analysis of variance; Tukey's multiple range analysis ($P < 0.01$) was used.

Pathogenicity tests for Fusarium wilt. We prepared conidial suspensions of the 35 *F. o. tuberosi* isolates by transferring lyophilized conidia to CLA. After incubation at 25 C for 2 wk under lights as previously described, conidia were washed from cultures with sterile distilled water and counted in a hemacytometer; the suspension was diluted to 10^5 conidia per milliliter. Sprouts were cut from sound, unblemished potato tubers, cultivar Vanderplank, with a 10-mm-diameter corkborer, planted in seed trays filled with sterile vermiculite, and irrigated daily. After emergence, plantlets were transplanted to 25- × 25- × 15-mm plastic containers (five planted per container) filled with a 1:1 mixture of peatmoss and vermiculite and kept at 25 C for a 12-h photoperiod. Field capacity was maintained, and a full-strength Hoagland's solution was applied weekly. After the plants had reached a height of approximately 20 cm, the plastic containers were put into Wisconsin soil temperature tanks that were illuminated with 40-W fluorescent tubes for a 12-h photoperiod. The water temperature of the tanks was kept at 38 C, and the soil temperature in the containers stabilized at 35 C. The water and soil temperatures were monitored with a datalogger (Grant 1200, Grant Instruments [Cambridge] Limited, Bulbeck Mill, Barnington, Cambridge, England) and had a maximum fluctuation of 0.5 C. Plants were kept dry for 24 h before they were inoculated. Plants were uprooted from the containers, and their roots were immersed in each conidial suspension for 5 min. Inoculated plants were returned to the containers. Twenty plants were inoculated with each conidial suspension. After inoculation, the soil in the containers was brought back to field capacity until wilt symptoms developed. Wilt symptoms developed between 7 and 14 days. Isolations and identifications were done as described before. The experiment was done twice for each of the two growing seasons of potatoes in South Africa.

Pathogenicity tests for Fusarium dry rot. Conidial suspensions of the 35 *F. o. tuberosi* isolates were prepared as described for Fusarium wilt. The suspensions were diluted to 10^4 conidia per milliliter. Unblemished potato tubers of cultivars Vanderplank, BP1, and Late Harvest were disinfected for 15 min in 3% sodium hypochlorite and allowed to dry. We inoculated 20 tubers of each cultivar approximately halfway between rose and heel ends by injecting 0.2 cm^3 of the spore suspension 8 mm into the tissue with a Socorex 2-187 self-refilling type syringe (23). The tubers were then incubated in paper bags and kept at 25 ± 2 C and 50–70% RH to promote dry rot development (25). After a 3-wk incubation, tubers were cut in half at the inoculation site, and the extent of dry rot development was determined according to a disease index scale used by Theron and Holtz (23). The experiment was done as a randomized block design with three replications; 20 tubers were used each time. The experiment was done twice for each of the growing seasons of potatoes in South Africa.

Vegetative compatibility of generation of *nit* mutants. Chlorate-resistant mutants were generated from the 35 *F. o. tuberosi* isolates on potato-sucrose chlorate medium (KPS) and minimal chlorate medium (KMM) (18) as well as on four alternative media: potato-dextrose chlorate medium (PDC) (3), malt chlorate agar (MA), cornmeal chlorate agar (CMA), and Czapek-Dox chlorate agar (CDA) all amended with potassium chlorate (15 g/L). The chlorate-resistant mutants obtained were grown on minimal medium (MM) (18) containing only nitrate as the nitrogen source, which tested for a deficiency in the nitrate reduction pathway. Mutants that grew on MM as thin expansive colonies with no aerial mycelium were considered *nit* mutants. These mutants showed wild-type growth on complete medium (CM) (3).

Vegetative compatibility of *nit* mutant phenotypes. The physiological phenotypic classes of all *nit* mutants were determined by colony morphology on media containing one of five different nitrogen sources (nitrate, nitrite, hypoxanthine, ammonium, and uric acid [3]). The *nit* mutants were assigned to three phenotypic classes. These classes presumably represent a mutation at either a nitrate reductase structural locus (*nit1*), a nitrate assimilation pathway-specific regulatory locus (*nit3*), or at least five loci that affect the assembly of a molybdenum-containing cofactor that is necessary for nitrate reductase (EC 1.6.6.6.) activity (NitM). To distinguish nitrite reductase mutants from pathway-specific regulatory mutants, we evaluated *nit* mutants that grew as thin colonies on nitrite medium for nitrate reductase activity by testing for nitrite excretion as described by Cove (7).

Vegetative compatibility groups. All *nit* mutants generated from a single isolate were paired in genetic complementation tests. All the *nit* mutants recovered from the same parent were paired with at least one *nit1*, one *nit3*, and one NitM mutant from that parent (3). Genetic complementation was indicated by the presence of dense aerial growth where the mycelia of two *nit* mutant colonies came in contact and formed a heterokaryon. A *nit1*, *nit3*, and NitM mutant from each isolate was paired in all possible combinations for the determination of vegetative compatibility groups.

RESULTS

Pathogenicity tests. The 10 isolates that were originally from potato stems with wilt symptoms (isolates 16–20 and 31–35) induced wilt symptoms in Vanderplank potato plants (Tables 1,2). Isolates 31 and 32 were weaker wilt pathogens than the other wilt isolates. These two isolates had a lower disease severity on the potato plants, and fewer potato plants developed disease symptoms (Table 2). Isolate 33 also caused stem-end rot symptoms in inoculated tubers, and isolate 31 was the only isolate of the 10 that caused all three disease symptoms (Table 2). Eight of the 10 isolates initially isolated from wilted potato plants caused the trial plants to wilt before they initiated progeny tubers. Because of this, the ability of these isolates to cause stem-end rot could not be assessed (Table 2). All 15 isolates initially from potato tubers with stem-end rot induced stem-end rot after inoculation (Tables 1,2). Isolate 4 from potato tubers with stem-end rot lesions also induced wilt symptoms. Only eight of the 10 isolates (isolates 21–25, 27, 28, and 30) from tubers with dry rot symptoms induced dry rot symptoms in inoculated tubers of Vanderplank and Late Harvest. On BP1, however, the other two isolates, 26 and 29, were weakly virulent pathogens. Isolate 23 from a potato tuber with dry rot symptoms induced weak wilt symptoms in Vanderplank. Isolates 26, 29, and 23 had a lower disease severity, and fewer tubers developed disease symptoms (Table 2).

Recovery of *nit* mutants. Isolates of *F. o. tuberosi* readily formed chlorate-resistant sectors on the six chlorate-containing media. All the isolates were more inhibited on KMM than on KPS. The average number of sectors per petri dish per isolate on KMM, MA, and CMA ranged from 0.5 to 3.9. Less than 30% of the isolates developed more than two sectors, and only 20% developed two or more phenotypically different *nit* mutants. On KPS and PDC, there were between 1.7 and 5.5 sectors per isolate. Of all the isolates, 85% developed at least two phenotypically different *nit* mutants on KPS and PDC. Of these isolates, 65% developed two or more phenotypically different *nit* mutants. Not all the isolates formed *nit* mutants on KPS, MA, CMA,

and CDA. On KMM and PDC, all the isolates generated at least one *nit* mutant. Thus, KMM and PDC were used in all the other tests. We evaluated all *nit* mutants to determine the physiological phenotypic classes.

***Nit* mutant phenotypes.** All the *nit* mutants obtained from the *F. o. tuberosi* isolates could be divided into three phenotypic classes. The majority of *nit* mutants were *nit1* mutants (Table 3). The frequency of *nit1* mutants was higher on PDC than on KMM, whereas the frequencies of *nit3* and NitM mutants were higher on KMM than on PDC (Table 3). A few sectors that were resistant to chlorate were recovered, but they had a wild-type colony morphology on MM. The frequency of these wild-type mutants was higher on PDC than on KMM (Table 3).

***Nit* mutant complementation tests.** Complementation between different *nit* mutants was indicated by the development of dense aerial growth where the mycelia of the colonies grew together and anastomosed. When NitM mutants were paired (or were involved in the pairing), complementation occurred more rapidly and resulted in heterokaryons that were more robust than those of other *nit* mutant pairs. When *nit1* and *nit3* mutants were paired, weak vegetative compatibility reactions were obtained. When *nit* and *nit3* mutants were paired, heterokaryons were not obtained within the first 3 wk, and even after 3 wk the compatibility reactions were weak. In some isolates, no compatibility reactions were observed even after 4 wk.

Vegetative compatibility groups. The 35 isolates were assigned to six VCGs (Table 1). Initially, a few *nit* mutants in one VCG were able to complement *nit* mutants in another VCG. For example, a *nit* mutant (NitA) of isolate 4 (VCG C) complemented a *nit* mutant (NitD) of isolate 8 (VCG A) but no other mutant in that VCG. The NitA mutant of isolate 4 did not complement NitB and NitD of isolate 32 in its own VCG, but it did complement NitC of that isolate. After the physiological phenotypes of the different *nit* mutants were determined and a NitM mutant was recovered and used as the tester mutant, the *nit* mutants in one VCG no longer complemented *nit* mutants in another VCG. Each VCG contained isolates that caused primarily one of the three diseases. For example, VCGs A and B contained mainly isolates that caused stem-end rot symptoms, whereas VCGs C and D contained mainly isolates that caused wilt symptoms. VCGs E and F contained only isolates that caused dry rot of potato. VCG E contained isolates from potato tubers grown on the Transvaal Highveld, whereas VCG F contained isolates from potato tubers grown in the eastern Orange Free State.

DISCUSSION

The phenotypic classes recovered during this study corresponded with those Correll et al (3) recovered from *F. oxysporum*. Puhalla (18) designated *nit* mutants arbitrarily as NitA and NitB. Initially, this was done in the present study as well. It was found that a *nit* mutant (NitA) of isolate 4 (VCG C) complemented a *nit* mutant (NitD) of isolate 8 (VCG A) but no other mutant in that VCG. The NitA mutant of isolate 4 did not complement NitB and NitD of isolate 32 in its own VCG, but it did complement NitC of that isolate. Correll et al (5) obtained similar results with *nit* mutants of *F. oxysporum* (Schlechtend.:Fr.) f. sp. *apii* (R. R. Nelson & Sherb.) W. C. Snyder & H. N. Hans. race 2

TABLE 1. Geographical location, pathogenicity tests, and vegetative compatibility groups (VCGs) of isolates of *Fusarium oxysporum* f. sp. *tuberosi* from South Africa

Isolate number	Geographical origin[x]	Original disease symptom[y]	Pathogenicity tests with cv. Vanderplank[z]			VCG
			Stem-end rot	Dry rot	Wilt	
1	TVLH	SER	+	−	−	A
5	TVLH	SER	+	−	−	A
8	EOFS	SER	+	−	−	A
11	TVLH	SER	+	−	−	A
12	TVLH	SER	+	−	−	A
15	EOFS	SER	+	−	−	A
31	RDP	W	+	+(w)	+(w)	A
33	PONTDRIF	W	+	−	+	A
2	TVLH	SER	+	−	−	B
3	EOFS	SER	+	−	−	B
6	TVLH	SER	+	−	−	B
7	EOFS	SER	+	−	−	B
9	EOFS	SER	+	−	−	B
10	EOFS	SER	+	−	−	B
13	TVLH	SER	+	−	−	B
14	EOFS	SER	+	−	−	B
4	EOFS	SER	+	−	+	C
32	RDP	W	*	−	+(w)	C
34	PONTDRIF	W	*	−	+	C
35	RDP	W	*	−	+	C
16	TVLH	W	*	−	+	D
17	TVLH	W	*	−	+	D
18	GAMTOOS	W	*	−	+	D
19	EOFS	W	*	−	+	D
20	RDP	W	*	−	+	D
21	TVLH	DR	−	+	−	E
22	TVLH	DR	−	+	−	E
25	TVLH	DR	−	+	−	E
26	TVLH	DR	−	−	−	E
23	EOFS	DR	−	+	(w)	F
24	EOFS	DR	−	+	−	F
27	EOFS	DR	−	+	−	F
28	EOFS	DR	−	+	−	F
29	EOFS	DR	−	−	−	F
30	EOFS	DR	−	+	−	F

[x] TVLH, Transvaal Highveld; EOFS, eastern Orange Free State; RDP, Roodeplaat (Vegetable and Ornamental Plant Research Institute).
[y] SER, stem-end rot; DR, dry rot; W, wilt.
[z] (w), Weak; +, symptom-positive; −, symptom-negative; *, no stem-end rot symptoms could be obtained, because plants wilted before progeny tubers were initiated.

isolates. They found that 64% of *F. o. apii* race 2 isolates did not pair with either of the two original complementary *nit* mutants of *F. o. apii* race 2 (NitA and NitB). However, all of these isolates did pair with an additional *F. o. apii* race 2 NitC mutant. After the physiological phenotypes of the *nit* mutants in this study had been determined, we included a NitM mutant as the tester mutant in all the complementation tests. Although the process was time-consuming, we were able to obtain a NitM mutant for every isolate used in our study by using the methods of Correll et al (3) and Klittich and Leslie (13). When a NitM mutant was recovered, another *nit* mutant tester was not needed. This shortened the whole process. Correll et al (3) also reported that the NitM mutant was a most reliable *nit* mutant tester strain in vegetative compatibility tests, and therefore it was necessary to

TABLE 2. Differences in the ability of isolates of *Fusarium oxysporum* f. sp. *tuberosi* from South Africa to cause dry rot, stem-end rot, and wilt in the cultivar Vanderplank

Isolate number	Average dry rot index value[w]	Homologous groups[x]	Average percentage of wilt[y]	Homologous groups	Average percentage of stem-end rot[z]	Homologous groups
2	0.0	a	0.0	a	54.4	h
3	0.0	a	0.0	a	51.4	efg
4	0.0	a	51.4	d	53.6	h
5	0.0	a	0.0	a	50.20	de
6	0.0	a	0.0	a	52.8	gh
8	0.0	a	0.0	a	52.6	fgh
9	0.0	a	0.0	a	49.2	d
11	0.0	a	0.0	a	62.0	j
15	0.0	a	0.0	a	54.6	h
25	0.0	a	0.0	a	0.0	a
29	0.0	a	0.0	a	0.0	a
33	0.0	a	68.0	e	39.4	b
34	0.0	a	72.0	ef	*	*
1	0.05	a	0.0	a	53.4	h
10	0.06	a	0.0	a	42.2	c
14	0.06	a	0.0	a	52.8	gh
20	0.06	a	81.0	g	*	*
17	0.07	ab	93.0	h	*	*
7	0.08	abc	0.0	a	50.80	def
16	0.14	abcd	100.0	i	*	*
32	0.14	abcd	44.0	c	*	*
13	0.15	abcd	0.0	a	67.4	k
35	0.16	abcd	100.0	i	*	*
18	0.22	abcde	91.0	h	*	*
12	0.25	abcdef	0.0	a	57.6	i
30	0.45	abcdefg	0.0	a	0.0	a
19	0.73	bdefg	75.0	f	*	*
31	0.88	eghi	39.0	c	39.4	b
21	1.36	hij	0.0	a	0.0	a
22	1.43	ij	0.0	a	0.0	a
K	1.49	j	0.0	a	0.0	a
26	1.52	j	0.0	a	0.0	a
28	1.53	j	0.0	a	0.0	a
24	1.53	ij	0.0	a	0.0	a
23	1.67	j	16.0	b	0.0	a
27	1.87	j	0.0	a	0.0	a

[w] Average dry rot index value obtained from three replications with 20 tubers each 3 wk after inoculation. 0 = No dry rot; 5 = 100% dry rot.
[x] Homologous groups obtained by Tukey's multiple range analysis ($P < 0.01$). There are no significant statistical differences between the isolates with the same letters.
[y] Average percentage of wilt obtained from five replications with 20 plants each 7–14 days after inoculation.
[z] Average percentage of stem-end rot obtained from five replications with 20 tubers each 3 mo after inoculation.

TABLE 3. Frequency of phenotypic classes of nitrate nonutilizing (*nit*) mutants recovered from 35 isolates of *Fusarium oxysporum* f. sp. *tuberosi* from South Africa on two chlorate-containing media

			Chlorate medium			
			KMM[w]		PDC[x]	
Phenotypic classes[u]	Number of *nit* mutants II	Percentage of *nit* mutants[v]	Number of *nit* mutants (III)	Percentage of *nit* mutants[y]	Number of *nit* mutants (IV)	Percentage of *nit* mutants[z]
nit1	568	56.35	93	16.37	475	83.63
nit3	284	28.17	239	84.15	45	15.85
NitM	116	11.51	99	85.34	17	14.66
Wild type	40	3.97	4	10.00	36	90.00
Total (I)	1,008					

[u] According to Correll et al (3).
[v] I/II × 100.
[w] Minimal chlorate medium.
[x] Potato-dextrose chlorate medium.
[y] III/II × 100.
[z] IV/II × 100.

identify a NitM tester strain for each different VCG (3). The best medium for obtaining NitM mutants was KMM, but if an isolate did not produce a NitM mutant on KMM, PDC or one of the alternative chlorate-containing media could be used. No vegetative self-incompatibility isolates were obtained during this study.

Results obtained with isolate 4 (stem-end rot isolate [in a wilt VCG] causing wilt and stem-end rot symptoms after inoculation) and isolates 31 and 33 (wilt isolates [in a stem-end rot VCG] causing wilt and stem-end rot symptoms after inoculation) indicate that a relationship between Fusarium wilt and stem-end rot might exist. Fusarium wilt isolates might induce Fusarium stem-end rot symptoms in potato tubers and vice versa. Bosland and Williams (2) reported that two pathogenic races of *F. oxysporum* (Schlechtend.:Fr.) f. sp. *conglutinans* (Wollenweb.) W. C. Snyder & H. N. Hans. belonged to the same VCG. It is significant that isolates causing a specific disease symptom belong to more than one VCG. More VCGs could be found if more isolates were examined. Although there is no simple relationship between isolates and VCGs, the presence of pathogenic variants within a single VCG is important, because studies of sexually reproducing fungi such as *Gibberella fujikuroi* (20) and *A. nidulans* (8) indicate that a high degree of genetic homology is required for isolates to be vegetatively compatible. This homology suggests that isolates that differ in pathogenicity within a VCG may differ at a relatively small number of loci (12). Pathogenic diversity within VCGs A, B, C, and D suggests that a parasexual cycle may occur among isolates in every group. Parasexuality might also lead to the development of new races (12). More information on the genetic relationships among VCGs and isolates that cause Fusarium wilt and stem-end rot will be required before vegetative compatibility can be used as a tool that distinguishes between isolates that cause these two diseases. VCGs E and F, however, contained only dry rot isolates; this suggests that dry rot isolates may be a more distinct group than the wilt and stem-end rot groups. It was also of interest that the isolates in VCGs E and F represented two geographically distinct groups; they were recovered from areas where cultural practices differ. A similar situation was also reported by Jacobson and Gordon (12) for *F. o. conglutinans* race 2 isolates from the San Joaquin Valley. These results suggest that vegetative compatibility tests may be used to distinguish isolates that cause dry rot from isolates that cause wilt or stem-end rot.

Two of the dry rot isolates did not induce dry rot symptoms, although they were still vegetatively compatible with the other eight dry rot isolates, which retained their virulence. Therefore, virulence might be lost in these two isolates because of mutation in the progenitors, whereas the vegetative compatibility loci were retained. These two isolates showed weak virulence reactions on BP1, which suggests that these two isolates could be weaker pathogens than the others. Moreover, because Late Harvest and Vanderplank are more resistant than BP1, this reaction might be a reflection of the cultivar resistance in combination with the weaker pathogenic potential of these two isolates. No single isolate may safely be regarded as representative of the pathogenic potential of all *F. o. tuberosi* isolates causing one of the three diseases of potatoes. This consideration bears directly on the selection of isolates for use in screening potato cultivars for resistance to Fusarium dry rot, stem-end rot, and wilt. The Fusarium wilt isolates still belong to the same VCG after reisolation. These results stress the possibility of using VCGs within *F. o. tuberosi* as naturally occurring markers in greenhouse and field inoculation experiments. These observations are based on very few isolates and need to be substantiated by more extensive collections and further characterization of VCGs of *F. o. tuberosi* isolates that cause potato dry rot, stem-end rot, and wilt.

Pathogenicity tests, although time-consuming and influenced by temperature, humidity, and pH, have been and will be useful for the characterization of these three diseases of potatoes. Finding other techniques that are less time-consuming and variable than pathogenicity tests would be useful in differentiating *F. o. tuberosi* isolates, which apparently are a genetically diverse group unified by the common trait of pathogenicity to potatoes. In other studies, vegetative compatibility tests have been useful in identifying subspecific groups within a morphological species (1,8,19,21). Once the relationship between wilt and stem-end rot is established, vegetative compatibility could be useful for the plant pathologist and plant breeder who want to distinguish between *F. o. tuberosi* isolates that cause these three diseases of potato.

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