Susceptibility of "Resistant" Tomato Cultivars to Fusarium Wilt

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ABSTRACT

Missouri Accession 160 and other tomato lines possessing the I gene for resistance to Fusarium wilt incited by race 1 seemed susceptible to race 1 following inoculation via the Wellman root-dip method. University of Florida tomato breeding line 126915-1-8-1, 'Walter,' and 'Florida MH-1' were resistant to race 1, indicating that these lines possessed a gene (or genes) for resistance that Missouri Accession 160 did not. Approximately 17% of the I-gene cultivars were less tolerant of race 2 than several race 1-tolerant cultivars. However, the "Vertifolia" effect was shown not to be inevitable, because 21% of the I-gene varieties were more tolerant of race 2 than the race 1-tolerant varieties.

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The I gene for resistance to Fusarium wilt of tomato *(Lycopersicon esculentum* Mill.), incited by *Fusarium oxysporum* Schlecht. f. sp. *lycopersici* (Sacc.) Snyd. and Hans. race 1, confers near "immunity" according to Bohn and Tucker (2). They reported that less than 1.0% of the Missouri Accession 160 *(L. pimpinellifolium* Mill.) plants exhibited symptoms when planted into soil highly infested with race 1. Porte (7) reported P. I. 79532 (i.e., Missouri Accession 160) was 100% resistant in his 1935 and 1936 tests. Subsequently Porte and Walker (8) released Pan American, a cultivar developed from a cross between Marglobe (tolerant to race1) and P. I. 79532. Pan American, in the release circular, was described as being 95 to 100% resistant to race 1. More recently, Cirulli and Alexander (3) reported the incidence of diseased plants of P. I. 79532 to be less than 5.0% following inoculation with race 1.

Crill et al. (4) have observed that many present-day cultivars possessing the I gene are not as resistant to race 1 as has been reported for Missouri Accession 160. This suggests that plant breeders may have lost genes for tolerance that Missouri Accession 160 possessed in addition to the I gene, and that in their concern to retain the I gene they may have fallen into the "vertifolia" pitfall as predicted by Van der Plank (12). However, the possibility exists equally that Missouri Accession 160 does not have tolerance genes for the plant breeder to lose and that under Florida conditions, and the procedures developed at the Agricultural Research and Education Center in Bradenton (AREC-Bradenton), Missouri Accession 160 is not as resistant as previous workers have reported.

Alexander and Hoover (1) reported the resistance of P. 126915-1-8-1) homozygous for resistance to races 1 and 2. races 1 and 2. This was confirmed by Stall and Walter (9) who developed through selection an inbred line (U. F. 126915-1-8-1(homozygous for resistance to races 1 and 2. According to Cirulli and Alexander (3) this resistance was regulated by two genes, one conferring resistance to race 1 and one conferring resistance to race 2. They suggested that the symbol I represent the resistance gene to race 1 derived from Missouri Accession 160 and that the symbol I-2 represent the resistance gene to race 2 obtained from U. F. 126915-1-8-1. The gene for resistance to race 1 in U. F. 126915-1-8-1 was left undesignated because they were unable to determine if it was the same as the I gene.

The genes for resistance to races 1 and 2 possessed by the cultivars 'Walter' (10) and 'Florida MH-1' (5) were derived from U. F. 126915-1-8-1. These cultivars repeatedly have been observed to be resistant to race 1 when inoculated via the Wellman root-dip method (13), whereas most cultivars possessing the I gene obtained from Missouri Accession 160 appear susceptible when so inoculated. At least three possible explanations of this apparent discrepancy are obvious; (i) the plant breeders lost tolerance genes in their concern for maintaining the I gene, (ii) the gene for resistance in U. F. 126915-1-8-1 is different from the I gene, and (iii) the single dominant genes for resistance to race 1 in Missouri Accession 160 and U. F. 126915-1-8-1 are the same, but the latter contains tolerance genes that the former does not.

Numerous experiments comparing the wilt reactions of several tomato cultivars and lines have been conducted at the AREC-Bradenton with essentially the same results. Herein are reported the results of two such experiments which were designed to determine whether the "vertifolia" effect actually has occurred, and whether Missouri Accession 160 actually does contain genes which condition tolerance to race 1 that are absent in present-day cultivars. Missouri Accession 160 also was compared directly with U. F. 126915-1-8-1 to determine whether these two plant introduction derivatives have similar Fusarium wilt reactions, or whether the resistance of U. F. 126915-1-8-1 is indeed different from that of Missouri Accession 160.

MATERIALS AND METHODS.—Ten-day-old seedlings of 33 tomato cultivars in test 1, and 13 cultivars in test 2, were root-dip inoculated (13) with *F. oxysporum* f. sp. *lycopersici* race 1 and race 2, dibbled into steam-pasteurized amended soil (1 part Leon fine sand: 1 part peat, v/v in wooden 51 ×38-×7.6-cm (20-× 15-× 3-inch flats, and placed in a plant production house in test 1, and in a greenhouse in test 2. Ten plants of each cultivar were transplanted without inoculation in both tests to serve as controls. A split-plot design was used with five replications in the first test and four in the second. Whole plots consisted of pathogen races and subplots of tomato cultivars. Each treatment in both tests consisted of 20 plants of each of the cultivars inoculated with race 1 and 20 plants with race 2.

Inoculum was produced by growing cultures of race 1 and 2 separately on PDA in petri plates for 10 days at 28 C with continuous light with an intensity of 1,614 lx (150 ft-

c). The Oristano (3) race 1 culture, which had been tested for pathogenicity and maintained in soil tubes (11) under refrigeration, originated from successive single microspore isolations of a culture furnished by L. J. Alexander of the Ohio Agricultural Research and Development Center, Wooster. The race 2 culture was derived from a Florida isolate which had been tested for pathogenicity and maintained in soil tubes. The contents of the plates were placed in a microblender with a small amount of sterile deionized water and briefly comminuted to produce a dense inoculum suspension of spores, mycelium, and agar. Average spore concentrations, as determined with a Levy corpuscle counting chamber in test 1, were 9.0×10^6 and 8.25×10^6 spores/ml for race 1 and 2, respectively, and in test two, 9.25×10^6 and 14.5×10^6 for race 1 and 2, respectively. Hyphal fragments were not counted, but the spore numbers indicate that dense inocula were used.

If root-dip-inoculated susceptible seedlings are incubated under favorable environmental conditions, true Fusarium wilt symptoms, which are readily distinguishable from the toxin syndrome first studied by

TABLE 1. Percentage wilt and death of various tomato cultivars following root-dip inoculation with *Fusarium oxysporum* f. sp. *lycopersici* races 1 and 2

Experiment no. and cultivars	Race 1 wilt reaction	Race 1-inoculated		Race 2-inoculated	
		diseased (%) plants	dead (%) plants	diseased (%) plants	dead (%) plants
Experiment 1					
Bonny Best	Susc.	97	85	98	77
Santa Rita	Susc.	93	57	92	34
Highlander	Susc.	90	72	96	74
Earliana	Susc.	85	41	88	32
Pritchard	Tol.	85	43	75	19
Marglobe	Tol.	74	47	86	39
Grothen's Globe	Tol.	72	25	85	29
Campbell 19	Res.	54	13	87	70
Globemaster	Res.	54	8	64	9
Healani	Res.	52	14	97	76
Indian River	Res.	51	8	85	34
Manapal	Res.	48	13	96	57
Immokalee	Res.	45	11	81	38
Floralou	Res.	44	5	77	25
Atkinson	Res.	44	11	91	60
Manasota	Res.	43	9	91	60
Manalucie	Res.	42	3	82	34
Tropic	Res.	41	6	93	51
Marion	Res.	34	5	73	34
Tropi-Red	Res.	31	5	94	55
Tropi-Gro	Res.	31	7	79	25
Campbell-28	Res.	29	2	64	23
Floradel	Res.	29	4	74	25
Campbell 17	Res.	28	9	94	61
Bonus VFN	Res.	24	2	90	35
Homestead 24	Res.	22	2	83	21
Homestead 61	Res.	21	4	83	22
Jefferson	Res.	19	4	66	14
VF 145	Res.	16	0	82	11
Supermarket	Res.	9	2	68	5
Homestead 500	Res.	7	3	56	17
Florida MH-1	Res.	2	2	6	0
Walter	Res.	2	0	0	0
Experiment 2					
Highlander	Susc.	100	56	98	43
Bonny Best	Susc.	99	19	94	6
Earliana	Susc.	98	51	91	26
Pritchard	Tol.	100	59	100	36
Marglobe	Tol.	75	6	60	1
Missouri Acc. 160	Res.	40	36	43	19
Floradel	Res.	30	4	85	14
Jefferson	Res.	16	0	49	1
Tropic	Res.	15	0	78	3
Homestead 24	Res.	14	0	56	3
U.F. 126915-1-8-1	Res.	4	0	0	0
Florida MH-1	Res.	0	0	4	0
Walter	Res.	0	0	0	0

White (14) and Haymaker (6), consistently develop within 5-10 days and often test plants can be evaluated accurately after 10-12 days. In the experiments reported herein, all plants were examined externally and internally for disease symptoms 20 and 15 days after inoculation in tests 1 and 2, respectively. Data are reported as the percentage of the inoculated plants with wilt symptoms and the percentage of the inoculated plants that were dead at the time of examination.

Bonny Best, Highlander, Earliana, and Santa Rita were considered to be race 1-susceptible cultivars, and were assumed to possess no or few genes for tolerance to race 1 or race 2. The race 1 tolerant cultivars used in the tests were Marglobe, Pritchard, and Grothen's Globe. Although the percentages of diseased and dead plants for Pritchard were greater than for some of the susceptible cultivars, it was placed with the tolerant group because it long has been recognized as a race 1-tolerant cultivar (2).

RESULTS.—In experiment 1, none of the cultivars possessing the I gene were entirely free of Fusarium wilt incited by race 1. In fact, approximately 46% of the plants of these cultivars had a wilt incidence 40 to 54% and 83% of them had a wilt incidence greater than 20% (Table 1). Such cultivars would appear to be susceptible or tolerant to race 1, not "immune." These same cultivars, however, were less susceptible to race 1 than the known tolerant cultivars (Pritchard, Marglobe, Grothen's Globe; 72-85% wilt, 25-47% death) or the susceptible cultivars (Bonny Best, Earliana, Highlander, Santa Rita; 85-97% wilt, 41-85% death). Homestead 500, Supermarket, Walter, and Florida MH-1 were nearly free of infection by race 1 (2-9% wilt, 0-3% dead).

Walter and Florida MH-1 were the only cultivars resistant to race 2 (Table 1), although nearly all of the I gene-containing cultivars were less infected by race 2 than Bonny Best or Highlander. Nearly 17% of the I cultivars were more susceptible to race 2 than the race 1-tolerant cultivars, 62% were equally susceptible, and 21% were less susceptible (Table 1).

All noninoculated plants in this and the second experiment remained disease-free.

In experiment 2, forty percent of the Missouri Accession 160 plants developed wilt symptoms and 36% were dead within 15 days after inoculation with race 1 (Table 1). The incidence of diseased plants and of dead plants for the I gene-containing cultivars (Floradel, Jefferson, Tropic, Homestead 24) were considerably less than those of Missouri Accession 160, from which the I gene was derived in the process of cultivar development.

U. F. 126915-1-8-1, Walter, and Florida MH-1 were resistant to both race 1 and race 2. No other cultivar or line reacted in this manner to race 2, although Missouri Accession 160, all the I gene-containing cultivars, and the race 1-tolerant Marglobe were less susceptible to race 2 than the race 1-susceptible Bonny Best, Highlander, or Earliana. The presupposed race 1-tolerant cultivar Pritchard was as susceptible to race 2 as the race 1-susceptible cultivars.

DISCUSSION.—Missouri Accession 160 and all cultivars possessing the I gene for resistance appeared susceptible to race 1 in these experiments. Some perhaps were tolerant but certainly not "highly resistant" or "immune."

U. F. 126915-1-8-1, which was selected for resistance to races 1 and 2, Walter, and Florida MH-1 (the resistance of the latter two cultivars to races 1 and 2 was derived from U. F. 126915-1-8-1) were nearly free from infection by race 1 and race 2. Cirulli and Alexander (3) reported that the resistance of U. F. 126915-1-8-1 to race 1 was governed by a single dominant gene. They did not assign a symbol to this gene because they were unable to determine if it was the same or different from the I gene. However, since U. F. 126915-1-8-1 reacted differently to race 1 than did Missouri Accesssion 160, the former apparently possesses a gene (or genes) for resistance to race 1 that the latter does not. U. F. 126915-1-8-1 may possess the I gene plus multiple genes for tolerance that are lacking in Missouri Accession 160. Possibly the I-2 gene also confers some resistance to race 1 and the combination of I and I-2 results in transgressive resistance to race 1, or perhaps the I gene is not involved at all and the resistance is governed by an altogether different gene.

Although Missouri Accession 160 did not appear resistant to race 2, it was as tolerant to race 2 as it was to race 1. U. F. 126915-1-8-1, Walter, and Florida MH-1, all of which possess the I-2 gene, were nearly free of disease incited by race 2. Since approximately 17% of the I gene cultivars had more disease caused by race 2 than the race 1-tolerant cultivars, care should be exercised by plant breeders as Van der Plank suggests (12) lest their new cultivars prove to be more susceptible to a "new" race than a cultivar bred for tolerance to the established race(s). This "vertifolia" effect is not inevitable, however, since 21% of the tested I gene-containing cultivars were more tolerant to race 2 than the race 1-tolerant cultivars.

Because of the many reports that Missouri Accession 160 was nearly "immune" to race 1, and because a high wilt incidence in many I gene-containing cultivars had been observed, it was thought that tomato breeders might have lost genes for tolerance to race 1 in their preoccupation with the I gene. However, apparently such is not the case since 40% of the Missouri Accession 160 plants wilted and 36% died within 15 days after inoculation with race 1, whereas all four of the I gene-containing commercial cultivars in the test had wilt incidences less than 40% and death percentages of less than 4%.

With few exceptions, I gene-containing tomato cultivars exhibited considerable wilt development when inoculated with race 1 via the Wellman root-dip method. Consequently, an unidentified race 1 isolate mistakenly could be designated as race 2 unless known race 1 and 2 cultures are included in tests to determine pathogenesis. Furthermore, research delving into the nature of pathogenesis and host resistance, where the *Lycopersicon:Fusarium* host:pathogen interaction is utilized, perhaps should be interpreted on the basis of genetic tolerance and not resistance when I gene-containing tomato cultivars are used.

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