

Races of *Fusarium oxysporum* f. sp. *lisi*, Causal Agents of Wilt of Pea

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The authors thank A. T. Bolton, D. A. Doling, W. A. Haglund, N. Hubbeling, A. Kerr, J. M. Kraft, A. S. Lawyer, D. D. Roberts, T. P. Reiling, and G. S. Taylor for fungus isolates.

We appreciate the donation of seed by The Asgrow Seed Co., A. T. Bolton, M. J. Crampton, Ferry-Morse Seed Co., E. T. Gritton, N. Hubbeling, A. Kerr, J. M. Kraft, A. S. Lawyer, and Charles Sharpe & Co. Seed were also secured from Burpee and Olds Seed Companies.

Accepted for publication 14 January 1974.

ABSTRACT

Nineteen isolates of the pea-wilt *Fusarium* from four countries and five states of the United States were used to inoculate some or all of 27 cultivars of pea. Plants were grown in sterilized sand supplemented with a nutrient solution. Fungus inoculum was poured around the cut roots of the plants. Wounding was not necessary to obtain wilting, however, wounding increased the rate of symptom development and percentage of wilted plants. Ten races, rather than the four designated by the donors as races 1, 2, 4, and 5, were differentiated with seven cultivars. Race separation and a comparison with the results of others was difficult because some seed with the same cultivar name differed in genes for resistance. In cross-inoculations, two races of f. sp. *lisi*, formerly thought to be races 1 and 2 but

classified herein as races 6 and 5, respectively, were nonpathogenic on plants in 49 different genera, species, or cultivars that have been useful in identifying forms and races of *F. oxysporum*. Fifty-one f. spp. and races of *F. oxysporum* pathogenic on their respective hosts were nonpathogenic on the susceptible pea cultivars Thomas Laxton or Little Marvel. When new races arise by mutations or recombinations in pathogenic f. spp. of *F. oxysporum*, one would expect that they could be found most easily in locations where the crop had been grown intensively for many years. This may explain why the four isolates received indirectly as races 1 and 2 from one location are different from their previous designations.

Phytopathology 64:849-857.

Important vascular *Fusarium* diseases of numerous crop plants, among them the garden, canning, and field pea (*Pisum sativum* L.), have been reported from many countries. Satisfactory control measures for these diseases depend chiefly on the development of resistant cultivars (cvs). This has been a slow and expensive process for some crops, notably cotton, flax, tomato, and banana. The development of wilt-resistant peas must continue since, with the discovery of high resistance and its incorporation in some cvs, new races of the pathogen

have appeared. This likewise has occurred with other wilt fusaria.

In 1928, Linford described wilt of pea in Wisconsin and gave *Fusarium orthoceras* App. & Wr. var. *lisi* as the causal agent (28). Under a revised classification, it is *Fusarium oxysporum* Schlecht. f. sp. *lisi* (Linford) race 1 Snyder & Hans. (38). Another race which causes 'near wilt' was described by Snyder (37) in 1933 and by Snyder and Walker (39) in 1935 and is designated race 2 in the revised

classification. This race caused wilt of numerous cvs that were resistant to race 1.

In 1948, inoculations of pea were included in our investigations of the pathogenic capabilities of the formae speciales and races of *Fusarium oxysporum* in the vascular wilt group. During this investigation, the celery-wilt pathogen [*F. oxysporum* Schlecht. f. sp. *apii* (Nels. & Sherbakoff) Snyder & Hans.] was found to cause wilt of pea (4, 5).

In 1951, Schreuder (33) reported race 3 in the Netherlands, which caused wilt of some cvs that were resistant in the United States. Since the names of the cvs tested were not given, the validity of race 3 cannot be determined from the data.

Buxton (10) found a race that was similar to Schreuder's race 3, since it caused wilt of Delwiche Commando and New Era, both resistant to race 2. He proposed that the English strain provisionally be called race 3A until a comparison could be made, but a comparison was not made, and, in 1957, he called this isolate race 3 (11).

In 1966, Bolton et al. (9) described race 4, which caused wilt of cvs WR Perfection, Wisconsin C183, and New Era but not of New Wales.

In 1970, Haglund and Kraft (20) described race 5 based on experiments with races 1, 2, and 5, and the published information for races 3 (33) and 4 (9). Delwiche Commando, Wisconsin Perfection, Dark Skin Perfection, WR Perfection, New Era, and New Wales were the wilt-resistant cvs cited to separate the races. Race 5 caused wilt of the wilt-resistant cvs tested, viz., the latter four.

Among the wilt fusaria, flax wilt [*F. oxysporum* f. sp. *lini* (Bolley) Snyder & Hans.] appears to be an extreme in race complexities with an indefinite number of races (25). However, one might expect fewer pathogenic races among the wilt fusaria in general, due to the soil environment in which they usually persist, and the absence of a known sexual stage. It seemed likely that there were more than four or five races of the pea-wilt *Fusarium*, since this is a seed-borne fungus (22, 23, 36, 40, 43) attacking a world-wide crop having many resistant cvs grown under varied environmental conditions. Furthermore, a survey of the literature on varietal susceptibility and resistance of pea in each of which at least 12 cvs were tested (10, 12, 13, 14, 15, 16, 17, 21, 24, 28, 29, 30, 31, 41, 44, 45, 46) and a comparison of our results with those of other workers revealed anomalies in the classification of cvs, which suggested that more races might be involved. A search for additional races seemed worthy of investigation.

Cultures from a wide geographical range and numerous cvs as possible differentials were selected since these seemed to offer the best possibility to detect races. The results obtained and a discussion of various aspects of the problem of pathogenic race differentiation in *F. oxysporum* f. sp. *pisi* are presented.

MATERIALS AND METHODS.—Nineteen isolates of *F. oxysporum* f. sp. *pisi* were selected from a collection obtained from Australia, Canada, England, Netherlands, and five states of the United States.

Twenty-seven cvs were selected as possible differentials, and enough seed from each lot (with a few

exceptions) were obtained to use throughout the investigation.

Generally, plants were grown in sand in 8-liter glazed pots that had been sterilized at 1.05 kg-force/cm² (15 psi) for 8 h or more. A 2-cm diam lateral hole at the bottom of the pots provided drainage. A considerable range in size of sand particles was permissible, but extreme variations in particle size were avoided. For the past few years, the sand usually had been screened, washed, and dried at 113 C.

Usually 20 seed, treated with a hypochlorite solution (1,667 ppm available chlorine), were planted per pot, spaced evenly in a circle 2.5 cm from the periphery and 2.5 cm deep. The roots of all plants were cut by pressing an inverted Buchner funnel into the sand and then pouring the liquid inoculum around them. Roots were cut when the plants were small (4- to 5-leaf stage for peas) for the first inoculation and again 5 to 7 days later for the second inoculation. Peas were grown in the greenhouse during the fall, winter, and spring. The nutrient solution for growing plants (1), and for the fungus inoculum (3) are given elsewhere. When large plants required more liquid during the day, only water was used. The possible ill effects of unequal ion absorption were prevented by flushing each pot with at least 1 liter of water every 7 to 10 days. Precautions were taken to prevent cross-contaminations.

Monoconidial isolates, derived chiefly from microspores, were the source of the inoculum, and the corresponding mass cultures were tested for comparison. The isolates were maintained on potato-dextrose agar (PDA) slants, the tubes sealed with Parafilm® (Marathon Oil Company), and stored at about 5 C. In preparing the inoculum, 10 ml of sterile water were added to a fresh slant culture, and spores and mycelium were loosened with a sterile needle. One ml of this suspension was added to 500 ml of the liquid medium in a 2-liter flask. The flasks were kept at approximately 28 C for 72 h and were shaken several times daily. The inoculum was composed of fragments of hyphae, microspores, and bud cells and was used unwashed and undiluted. This procedure provided an inoculum concn in the optimum range since an objective has been to determine the full capabilities of the wilt fusaria. Dilutions of 1:1 or 1:2 of the inoculum of several f. spp. did not change significantly the percentages of wilt of their respective susceptible hosts. The undiluted inoculum gave the same relative susceptibility ratings of cvs of cotton or cowpeas in the greenhouse as in separate heavily infested field plots, and there was no evidence that staling products, with toxic effects on the plants, accumulated in the medium.

Experiments with peas in field plots have not been possible, but observations in a few commercial fields showed that symptom expression in the greenhouse was similar to that in the field. In general, the variations in symptom expression seemed to depend more on the cvs than on the race of the fungus. Diseased plants showed either yellowing, drying, or flagging of leaves as a one-sided effect from bottom to top, or sometimes a complete collapse, and were recorded as wilted when the symptoms had advanced to the top leaf. After 5 to 7 wk, a few plants in which symptoms had not extended to the top were also recorded as wilted.

TABLE 1. Reactions of cultivars of pea to isolates of 10 races of *Fusarium oxysporum* f. sp. *pisi*

Cultivar	Isolate →	Host response (external symptoms) ^a to Race																			
		1					2					4		5		6	7	8	9	10	11
		A	B	C	D	E	F	G	H	I	K	L	M	N	O	P	Q	R	S	T	
WR Alaska		R ₂	R ₃	R	R ₂	R ₄	S ₂	S ₂	S	S ₂	S	S	S ₅	S ₂	S ₃	R	S	S	S ₂	S ₃	
WR Thomas Laxton		R ₃	R ₄	R ₃	R ₂	R ₃	S ₂	S	S	S ₂	S ₂	S	S ₄	S ₂	S ₃	R ₂	S	S	S ₂	S ₂	
New Era		R ₂	R ₃	R	R ₃	R ₂	R ₂	R	R	R ₂	R ₆	R	S ₃	S ₃	S ₆	R	R ₂	R	R	R ₃	
New Wales		R	R	R ₈	R	R	R ₃	R ₃	R ₃	R ₃	S ₄	S	S	S ₃	R ₂	R	S ₃	S ₂	S	R	
Double One		S	S	S ₂	S ₃	S	R	R	R	I ₃	R	R	R ₃	R	R ₂	R ₃	R	R	R	R ₃	
Roi des Fins Verts		S ₂	S	S ₉	S	S ₂	R	R ₂	R ₂	R	R ₂	R	S ₂	S ₄	S ₂	S ₂	R	R ₂	R ₂	R ₂	
Dwarf Gray Sugar		R	R ₂	R ₁₈	R ₂	R ₃	S ₃	S	S	S ₂	S ₂	S	S ₅	S ₂	R ₆	R ₂	S	S	S ₂	S ₃	
Morse 60		R	R	R ₈	R	R ₃	S ₂	S	S	S ₃	S ₃	I	S ₃	S	R ₃	R ₂	S	S	S	I ₂	
Freezonian		R	R	R ₆			S ₂	S	S	S ₂	S	S	S	I ₂	R		S	S	S		
WR Perfection		R	R	R					R		I	I		R			S	S	I	R ₃	
Dark Skin Perfection					R	R	R			I ₂	R ₂		S ₂	S	R	R ₂	R ₂	I	R ₂		
Carter's Daisy		R	R	R							R	R		S		R	R	S ₃	S ₃		
Flavanda		R	R ₂	R ₂							I	R	S ₂	S ₂	R		I ₃	S ₂	I ₂		
WR Greenfeast		R	R	R			S ₂	S ₂		S	I ₃	R	I ₂		R		R	R	S ₄	R	
Green Era		R	R	R							R	R			R		R	R	R	R	
Koroza		R	R	R							R	R			R	R	R	R	R		
WR Lincoln		R	R	R			S ₂	I	S	S ₂	I ₂	R	R ₃	I ₃	R ₂		S ₂	S ₂	I ₂	R ₂	
New Season		R	R	R							R	R			R		R	R	R		
Nott's Excelsior		R	R	R							I ₂	I ₂			R		I	S	R		
Rovar		R	R	R ₂							R	R			R		S ₂	S ₂	R		
Wisconsin C183		R	R	R							S	S			R		S	S	I		
WR Massey		R	R	R							S	S			I		S	S	S		
Massey		S ₂	S ₂	S		S					S	S			S		S	S	S		
Wando						S	S			S	S		S ₂	S ₂	S		S	S	S		
Blue Bantam		S	S	S							S	S			S ₂		S	S	S		
Kelvedon Wonder		S	S ₂	S ₂		S ₂				S					S						
Little Marvel		S ₄	S ₇	S ₁₀	S ₃	S ₉	S ₄	S ₂	S ₃	S ₄	S ₁₁	S ₂	S	S ₁₄	S ₁₈	S ₂	S ₆	S ₆	S ₃	S ₃	

^aSymptoms: R = resistant, 0-39% wilt; I = intermediate, 40-59% wilt; S = susceptible, 60-100% wilt. Subscripts = number of tests; no subscript = 1 test.

Only the percentages of plants with external wilt symptoms are given, although at the end of an experiment plants without external symptoms were cut to determine the extent of vascular discoloration. We believe that external symptoms were the best criterion for evaluating the races and their degrees of virulence and that vascular discoloration was of minor importance.

RESULTS.—The 19 isolates tested and data for their separation into races based on the differential reactions of the cvs are given in Table 1. Seven were designated by the donors as race 1 (A, B, D, E, O, P, T), seven as race 2 (C, F, H, I, Q, R, S), one as race 4 (K), one as race 5 (M), one as possibly race 3 (L), and two collected by us (G, N) were assumed to be race 2. Nine isolates available at the beginning of the experiments were used to inoculate 24-27 cvs; those obtained later were used on 10-15 cvs. Genes for resistance of some cvs in different seed lots with the same name were clearly not the same. Until pure lines (perhaps from an official seed bank) can be used, the race classification must necessarily be considered as tentative.

Race 1 (A, B, C, D, E).—Isolates A and E were from England, B and C from the Netherlands (C as race 2), and D from the United States. Isolates O, P, and T were received indirectly as race 1 from Wisconsin but were different from those of race 1 (A-E, inclusive) and all other isolates on several cvs. They are, therefore, new races 6, 7, and 11, respectively.

Race 2 (F, G, H, I).—Isolates F, H, and I were supplied by donors as race 2 from Washington, Utah, and Illinois, respectively; G was supposedly seed-borne as it was isolated by us from a noninoculated plant of cultivar WR Thomas Luxton.

We classified isolates A-E as race 1 and F-I as race 2 because some of the resistant cvs developed early as WR Alaska, WR Perfection, and Freezonian were resistant to race 1 and susceptible to race 2; whereas, New Era and New Wales developed later were resistant to both races (18, 19). Double One and Roi des Fins Verts were susceptible to race 1 and resistant to race 2, thus further supporting the concept of these races.

Race(s) 3 and 3A.—Isolates of these races were not available. Their validity is considered in the discussion.

Race 4 (K, L).—Race 4 was established by Bolton et al. (9) on the basis of the resistance of New Wales and the susceptibility of New Era, WR Perfection, and Wisconsin C183. This race number is retained, but the reactions of the cvs from our seed stocks were not the same to isolate K

as those reported by Bolton et al. (9), since New Wales was susceptible and New Era was resistant. We investigated these differences by inoculating simultaneously plants of New Wales from our seed and from seed supplied by Bolton with isolate K (race 4) from Bolton (Table 2). Those from Bolton's seed were resistant (9) and those from our seed were susceptible (6). Isolate Q, that was similar in pathogenicity to isolate K on most cvs, and isolate O, that was different, were also included in these inoculations (Table 2). The similar pathogenic reactions of K and Q, and the opposite reactions of O to plants of each seed lot, again showed that seed with the same name had different genes for resistance.

The difficulty of race identification by differentials with the same name but different genes for resistance is shown in Table 3 by the reactions of different lots of New Era to four isolates of race 4 and isolates of races 5, 6, and 8. In the table, the seed lots are given as from Gritton (A), the same as we have used for several years; from Bolton (B); and two from Kraft, one as Kraft (C) and the other as P. I. 244, 195 (D). The isolates of race 4 were from: (i) Bolton, (ii) Haglund (originally from Bolton), (iii) Roberts (originally from Haglund but reisolated from a wilted New Era plant), and (iv) Roberts (via England). Isolates of races 5 and 6 that have caused severe wilt of plants from lot A were included. Every isolate could not be tested on seed lot B because only a few seeds were available, but race 6 caused wilt of plants from all lots of seed and race 5 from lots A, C, and D. An isolate of race 8 was tried only on lots A and B, and the plants therefrom were resistant, suggesting that these lots probably had similar genes for resistance. However, with isolates of race 4 that could be used, the lots fell into two classes: A and B, resistant; C and D, susceptible or intermediate. Plants from the two latter lots were alike in general appearance but were clearly different from those of lot A. Plants from the A and B lots were resistant to race 4 in our experiments (Table 3), but Bolton et al. reported their lot (B) susceptible (9). This difference was not resolved due to the lack of seed.

Isolate L (from the Netherlands) was similar to isolate K and was placed in race 4.

Race 5 (M, N).—Isolate M was from Haglund and N was collected in South Carolina in 1955. Collections of *f. sp. pisi* were made at irregular intervals in a few fields of peas from 1948 onward. All isolates caused wilt of WR Alaska and were assumed to be race 2. Later, isolate N

TABLE 2. Contrasts in the percentages of wilted plants from two lots of New Wales seed

Fusarium isolate	Seed Source					
	A ^a			B ^b		
	External symptoms (%)	Plants (No.)	Tests (No.)	External symptoms (%)	Plants (No.)	Test (No.)
K, race 4 ^c	80	54	3	0	12	1
Q, race 8	90	31	2	0	12	1
O, race 6	6	31	2	86	14	1
Uninoculated	0	35	2			

^aFrom Gritton.

^bFrom Bolton.

^cFrom Bolton.

TABLE 3. Percentages of wilted plants of four lots of cultivar New Era inoculated with isolates of four races of *Fusarium oxysporum* f. sp. *pisi*

	A ^a			B ^b			C ^c			D ^d		
	External symptoms (%)	Plants (No.)	Tests (No.)	External symptoms (%)	Plants (No.)	Tests (No.)	External symptoms (%)	Plants (No.)	Tests (No.)	External symptoms (%)	Plants (No.)	Tests (No.)
K, race 4 ^c	6	63	4	0	14	1	74	27	2	73	26	2
Race 4 ⁱ	3	33	2				33	24	2	44	18	2
Race 4 ^g	3	27	2				52	21	2	48	21	2
Race 4 ^h	0	15	1									
M, race 5	100	17	1				91	22	2	100	7	1
N, race 5	97	33	3				100	11	1	100	8	1
O, race 6	75	28	2	82	11	1	100	8	1	83	6	1
Q, race 8	3	30	2	0	11	1						

^aGritton.^cFrom Bolton.^bBolton.ⁱFrom Bolton via Haglund.^eKraft.^gFrom Bolton via Roberts via Haglund.^dPI 244195.^hFrom Bolton via Roberts via England.TABLE 4. Ten races of *Fusarium oxysporum* f. sp. *pisi* shown by different host responses of seven cultivars.

Cultivar	Race										
	1	2	4	5	6	7	8	9	10	11	
WR Alaska	R ^a	S	S	S	S	R	S	S	S	S	
Carter's Daisy	R		R		S		R	S	S		
Double One	S	R	R	R	R	R	R	R	R	R	
WR Greenfeast	R	S	IR ^b	I	R		R	S	R		
WR Lincoln	R	IS ^c	IR ^d	I	R		S	S	I	R	
New Era	R	R	R	S	S	R	R	R	R	R	
New Wales	R	R	S	S	R	R	S	S	S	R	
Little Marvel	S	S	S	S	S	S	S	S	S	S	

^aSymptoms: R = resistant, 0-39% wilt; I = intermediate, 40-59% wilt; S = susceptible, 60-100% wilt.^bI in four tests, R in one test.^cI in one test, S in five tests.^dI in two tests, R in one test.

was discovered to cause wilt of New Era and New Wales, and, upon further comparisons, was found to be race 5. The N isolate collected in 1955 was used here, since it is the only continuous single-spore line now available.

Race 6.—Isolate O, received indirectly as race 1 from Wisconsin, was used to inoculate 27 cvs. The different wilt-reactions of five cvs to this isolate and those in race 1 show O to be another race, designated race 6.

Race 7.—Isolate P, received indirectly as race 1 from Wisconsin, was somewhat less virulent on Little Marvel than all other isolates if measured by the average number of days for severe wilt, but it was not appreciably different on Roi des Fins Verts. In three separate experiments, the resistance of Double One to isolate P was distinct from its susceptibility to other isolates of race 1; therefore, another race is indicated, which is designated race 7.

Race 8.—Isolate Q, received indirectly as race 2 from Wisconsin, was assumed to be typical of race 2 until the susceptibility of New Wales was discovered, resembling in this respect four other isolates (K, L, R, S). It is designated race 8.

Race 9.—Isolate R was received from Australia as race 2, but the reactions of two cvs to the race 2 isolates and R were different. It is designated race 9.

Race 10.—The reactions of New Wales and WR Greenfeast were different to isolate S, received from England as race 2A, than to the race 2 isolates. Due to these and other differences, it is designated race 10.

Race 11.—Isolate T, received indirectly as race 1 from Wisconsin, caused the same wilt reactions on nine cvs as the four race 2 isolates (F-I, inclusive) and, therefore, resembles the race 2 group to which New Wales is resistant. However, WR Lincoln reacted differently to isolate T than to the race 2 isolates, indicating that T is another race, which is designated race 11.

Minimum number of cultivars for separating 10 races.—Excluding race(s) 3 and 3A for which isolates were unavailable, ten races can be differentiated by inoculating seven "wilt-resistant" cvs; WR Alaska, Carter's Daisy or Dwarf Telephone, Double One, WR Greenfeast, WR Lincoln, New Era, and New Wales (Table 4). Cvs WR Alaska, Carter's Daisy, New Era, and New Wales were the most satisfactory for the differentiation, since there were no intermediate classes (40-59% wilt). WR Lincoln was in four such classes after inoculation with 16 of the isolates (Table 1).

Inoculation of *Pisum sativum* L. susceptible cvs Thomas Laxton or Little Marvel with other wilt fusaria.—The pathogenicity of *f. sp. apii* has been mentioned. Fifty-one other *f. spp.* and races were nonpathogenic on these cvs. Thirty-eight of them are given in reference number 5, page 890; additional ones were *aechmeae*, *callistephi* race 2, *cassiae*, *chrysanthemi*, *fragariae*, *gladioli*, *lagenariae*, *lilii*, *narcissi*, *rauvolfi*, *ricini*, *tulipae*, and a *f. sp.* to be described causing wilt of Bambarra groundnut (*Voandzeia subterranea* L.).

Inoculation of other plants with races of *f. sp. pisi*.—For several years, cultures of *f. sp. pisi* that were considered to be races 1 and 2 were used in cross-inoculations. The original race 1 isolate lost pathogenicity; the "race 1" isolate used since 1956 is now classified as race 6 (O, Table 1). The isolate originally labeled race 2 is now classified as race 5 (N, Table 1).

These isolates were nonpathogenic on 49 genera, species, and cvs of plants (5, page 890) that have been useful in the separation of forms and races of *F. oxysporum*, and they were also nonpathogenic on Bambarra groundnut (*Voandzeia subterranea* L. 'Ntaba Ntidu'); castorbean (*Ricinus communis* L. 'Red Spire'); celery (*Apium graveolens* L. var. *dulce* DC. 'Golden Detroit'); cotton (*Gossypium arboreum* L. 'K-1') and (*G. barbadense* L. 'Pima 14-7-11'); eggplant (*Solanum melongena* L. 'Black Beauty'); gladiolus (*Gladiolus* spp. 'Picardy'); Mexican sunflower (*Tithonia rotundifolia* [Mill.] Blake 'Torch'); sweet william (*Dianthus barbatus* L. 'Giant Pure White'); and wallflower (*Cheiranthus cheiri* L. 'annual, single, mixed').

DISCUSSION.—Schreuder (33) did not list the cvs necessary to establish race 3, which was associated with early yellowing of peas. She stated in the English summary that, since the fungus caused wilt of some cvs that were resistant in the United States, it was race 3 and should not be confused with near wilt (race 2). Haglund and Kraft (20) apparently assumed that she used Rondo, Wisconsin Perfection, and Delwiche Commando as differentials. In 1954, Seinhorst (35) and Labryere and Seinhorst (27) reported that the disease probably was caused mostly by the nematode, *Hoplolaimus uniformis*, since the symptoms of early yellowing could not be reproduced by inoculation with *f. sp. pisi* race 3. Labryere et al. (26) concluded that early yellowing of peas was caused by the interaction of *Hoplolaimus uniformis* and one or more other factors, one of which could be *F. oxysporum f. sp. pisi* race 3. The stress on cortical decay, the method of testing for wilt, the low temp of 15-18 C during the experiment, and the uncertainty of the virulence of the *Fusarium*, or its identity with race 3 of Schreuder, all cause doubt that a tracheomycosis was involved.

Since Schreuder gave no differential cvs for race 3, but Buxton gave several for his provisional race 3A, later called race 3, it appears that race 3 should be credited to Buxton.

The difficulty of race identification by comparing one's data with those of others has been shown for hosts with the same name but different gene combinations, which is in contrast to the opinion (20) that "The use of published data with respect to the reaction of race 3 and 4 are more valid than the comparative studies made with attenuated strains or cultural mutants of the isolates described as *Fusarium oxysporum f. pisi* races 3 and 4". In our extensive tests, the race 4 isolate supplied to us by its discoverer was virulent and not an attenuated strain. Numerous inquiries indicate that no isolates of race 3 are available, and the published data are so meager that the validity of this race as given by Schreuder is uncertain. We have been aware of the problem of attenuated strains for many years (2) and are continually checking isolates for virulence. Only virulent isolates were used in the experiments reported here.

The articles by Bhide et al. (7), Sukapure et al. (41), and Safraneck (32) suggest other races. A new race is mentioned in Belgium (30), but the data are insufficient to characterize it among the races given here.

In 1967, Sulaiman and Utikar (42) reported races 3, 4, and 5 as new races of *f. pisi*, based on a comparative

cultural, morphological, and physiological study of six cultures. No mention was made of race(s) 3 and 3A of Schreuder (33) and Buxton (10) or race 4 of Bolton et al. (9), which were described before 1967. A review of numerous articles in the literature and our experiences over many years with the variability of *F. oxysporum* led us to believe that it was impossible to differentiate races on morphological characteristics and presently available cultural methods. The physiological character of pathogenicity also is variable, but it is one that we believe the pathologist has to use to define his cultures with a reasonable degree of certainty. Since differential cvs were not given by the authors (42), these races 3, 4, and 5 are invalid in our conception of a proper characterization of races of *F. oxysporum* f. sp. *pisi*.

During the preparation of the present paper, Roberts and Kraft (31) reported that isolates of races 1, 2, 4, and 5 could be recognized on plates of modified agar media.

Origin of new races.—*Fusarium oxysporum* has been said to be much like the rusts in pathogenic specificity, and, if so, one might expect resistant cvs to succumb to new races within a relatively short time, but this has not always been the case. Some formae speciales have a rather broad base of genes for pathogenicity, but this has not been demonstrated for f. sp. *pisi*; yet the number of races on pea reported here indicates that mutations or recombinations are occurring, and that other races undoubtedly will be found.

Race 1 of *F. oxysporum* f. sp. *pisi* was supposedly the original race, which was recognized about 1924 and described in 1928 (28). According to Walker (47), when processing varieties of canning peas were rapidly converted in the 1930's and 1940's to monogenic resistance to this race, the common wilt disease in this country was practically eliminated. However, race 2 appeared and subsequently was found to be widespread. It has been assumed that the isolates of this race have the same genetic composition for pathogenicity. Was the selection pressure and the gene mutation the same in all the innumerable places where the race has been found, or has it been disseminated widely probably through seed transmission? If selection pressure is a major factor in the change of genes for pathogenicity in the pea-wilt fungus, locations where peas have been grown intensively for many years would appear to be the logical places to find them.

In 1963, Walker (47) noted that mutation or recombination of genes had not been a serious problem with the three wilt diseases caused by f. spp. *conglutinans*, *lycopersici*, and *pisi*. However, that new pathogens are developing is illustrated by the susceptibility of resistant tomato cvs to isolates of f. sp. *lycopersici* (race 2) and the susceptibility of the resistant pea cvs New Era and New Wales to isolates of f. sp. *pisi* (race 5) in locations as distant as Washington and South Carolina.

The race situation in f. sp. *pisi* will be further clarified only when more isolates from widely separated regions of the United States and elsewhere are tested on a sufficient number of differentials.

Preservation of cultures and differential cultivars.—Investigators of *Fusarium*-wilt diseases might profit from the experiences of those working with other diseases, i.e. wheat stem rust, and develop some

international cooperation for identifying and preserving the pathogens as well as a source of cvs for use as differentials. Many workers concerned with the genetics of host and pathogen have emphasized the desirability of using isolines or near isolines of the host, pathogen, or both for comparative studies of host-pathogen interaction in disease-susceptible and disease-resistant cvs. Where knowledge of the genotypes of the host is available, the selection of widely varying types would be advantageous in identifying a large number of races of the fungus. With an organism such as *F. oxysporum*, the use of a large number of preserved cultures that retain selective pathogenicity and a high degree of virulence also would be desirable.

Previous to this investigation, we deposited 45 ff. sp. and races of *F. oxysporum* with The American Type Culture Collection. The lyophilized cultures have not been tested, but 18 isolates after being frozen under nitrogen for various periods of time, were as virulent as when sent to the ATCC. If a code could be established to recognize forms and races of living cultures, some of these might become type specimens.

The ATCC numbers assigned to isolates reported herein are listed below:

Race 1 ATCC 26043	Race 7 ATCC 26045
Race 2 ATCC 26044	Race 8 ATCC 26046
Race 4 ATCC 26087	Race 9 ATCC 26047
Race 5 ATCC 16607	Race 10 ATCC 26048
Race 6 ATCC 16606	Race 11 ATCC 26049

Proposal for races.—It is proposed that the races of *Fusarium oxysporum* Schlecht. emend. Snyder & Hansen f. sp. *pisi* (Tables 1 and 4) pathogenic in the vascular tissues and causing wilt of *Pisum sativum* L. be designated as follows:

(R, resistant = 0-39% wilt; I, intermediate = 40-50% wilt; S, susceptible = 60-100% wilt; external symptoms only).

Race 1 Linford (38).

Syn. *F. orthoceras* App. & Wr. var. *pisi* Linford (28).

F. oxysporum Schl. var. *orthoceras* (App. & Wr.) Bilal (8).

A race causing wilt symptoms described by Linford (28) and others (10, 14, 15, 22, 34, 39).

Differentials: WR Alaska, R; Double One, S.

Race 2 Snyder (38).

Syn. *F. oxysporum* Schl. f. 8 Snyder (39).

A race causing wilt symptoms described by Snyder and Walker (39) and others (34).

Differentials: WR Alaska, S; WR Lincoln, S; and New Wales, R.

Race 3 (Schreuder) Buxton emend. Armstrong and Armstrong.

Syn. *F. oxysporum* Schl. em. Sn. et H. *F. pisi* race 3 Schreuder (33).

A race causing wilt of some American and Dutch wilt-resistant pea cvs, but names of cvs not given by Schreuder. Causing wilt of cvs resistant to race 1, cvs Alaska, Alderman, Delwiche Commando, Dwarf Grey Sugar, New Era, and Wisconsin Perfection according to Buxton (10).

Race 4 (Bolton, Nuttall, and Lyall [9]) emend. Armstrong and Armstrong.

Differentials: Carter's Daisy, R; WR Lincoln, I; New Era, R; New Wales, S.

Race 5 Haglund and Kraft (20).

Differentials: Double One, R; New Era, S; New Wales, S.

Race 6 Armstrong and Armstrong.

Differentials: New Era, S; New Wales, R.

Race 7 Armstrong and Armstrong.

Differentials: WR Alaska, R; Double One, R.

Race 8 Armstrong and Armstrong.

Differentials: Carter's Daisy, R; WR Lincoln, S; New Era, R; New Wales, S; Rovar, S.

Race 9 Armstrong and Armstrong.

Differentials: Carter's Daisy, S; WR Greenfeast, S; New Era, R; New Wales, S.

Race 10 Armstrong and Armstrong.

Differentials: Carter's Daisy, S; WR Greenfeast, R; New Era, R; New Wales, S.

Race 11 Armstrong and Armstrong.

Differentials: WR Alaska, S; WR Lincoln, R; New Era, R; New Wales, R.

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