Inheritance of Resistance to Races 0 and 2 of *Fusarium oxysporum* f. sp. *melonis* in a Gynoecious Muskmelon

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ABSTRACT

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The mode of inheritance of resistance to Fusarium wilt (*Fusarium oxysporum* f. sp. *melonis*) races 0 and 2 in WI 998FR, a gynoecious muskmelon (*Cucumis melo*), was determined by analyzing segregation of F_1, F_2, and BC_1 populations of crosses with susceptible cultivar PMR 45. Resistance to races 0 and 2 in WI 998FR was conferred by a single dominant gene. In allelism tests, resistance to races 0 and 2 in WI 998FR was determined to be controlled by the gene (*Fom*-1), which also confers resistance in the cultivar Doublon. The gene controlling resistance to races 0 and 2 in WI 998FR was different from the gene (*Fom*-3) controlling resistance to races 0 and 2 in the cultivar Perlita FR.

Fusarium wilt, caused by *Fusarium oxysporum* Schlect. f. sp. *melonis* (Leach & Currence) Snyd. & Hans. (*F. o.* f. sp. *melonis*) has become an increasingly important disease of muskmelon (*Cucumis melo* L.) in the United States and Canada. Practical control of this soilborne disease is limited to breeding cultivars resistant to the pathogen. The gynoecious muskmelon-breeding population Wisconsin (WI) 998 (6) was found to be segregating for resistance to *F. o.* f. sp. *melonis* races 0 and 2 (*unpublished*). The resistant WI 998 selection is designated WI 998FR (*Fusarium*-resistant).

The purpose of this paper is to report the reaction of WI 998FR to *F. o.* f. sp. *melonis* races 0, 1, 2, and 1,2, the mode of inheritance of resistance to races 0 and 2 in WI 998FR, and the genetic relationship of WI 998FR to other sources of resistance to Fusarium wilt.

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MATERIALS AND METHODS

Standard hybridization techniques for muskmelon (11) were used to make crosses between the homozygous *F. o.* f. sp. *melonis* races 0 and 2-resistant selection WI 998FR and the *F. o.* f. sp. *melonis* races 0, 1, 2, and 1,2-susceptible cultivar Powdery-Mildew-Resistant 45 (PMR 45) to determine the mode of inheritance of resistance to Fusarium wilt in WI 998FR. A second series of crosses was made between the resistant selection WI 998FR and the homozygous *F. o.* f. sp. *melonis* races 0 and 2-resistant cultivars Doublon and Perlita FR to identify the alleles for resistance.

The cultivars PMR 45, Doublon, and Perlita FR are andromonoecious, and the hermaphrodite flower was crossed by bud pollination. The gynoecious sex expression of WI 998FR was modified with $AgNO_3$ at 200 ppm (4) to induce pollen-bearing hermaphroditic flowers, which were used as the pollen parents for reciprocal crosses to produce F_1 progenies. The F_1 progenies from the cross (andromonoecious × gynoecious) and the reciprocal cross were monoecious. Pistillate flowers were pollinated with staminate flowers from the same plant (F_1 self-pollinated) to produce F_2 progenies.

Inoculum for races 0, 1, and 1,2 was obtained from G. Risser (Station d'Ameloration des Plantes Maraicheres, Montfavet, France). Inoculum for race 2 was derived from field isolates of *F. o.* f. sp. *melonis* obtained from wilted muskmelon plants from the San Joaquin Valley of California. Races are classified according to the new race nomenclature proposed by Risser et al (9). The inoculum consisted of a mixture of macroconidia and microconidia prepared from acidified potato-dextrose agar (APDA) cultures grown for 7–10 days at 20–24 C with 24 hr of light. The inoculum concentration used in the inheritance studies was about 0.5×10^6 conidia per milliliter. Resistance of WI 998FR to races 0, 1, 2, and 1,2 was tested at inoculum concentrations of 0.025×10^6, 0.05×10^6, 0.1×10^6, 0.5×10^6, and 1.0×10^6 conidia per milliliter.

Seeds of plants to be assayed for disease reaction were treated with 5% calcium hypochlorite solution for 5 min and placed in autoclaved vermiculite (seedling pots). After about 10 days, plants in the cotyledon to first-true-leaf stage were removed from the seedling pots and the roots were washed in tap water, pruned to about 2.5 cm, and dipped for 1 min in the inoculum suspension. The inoculated seedlings were transplanted into cell-type (cell volume 55 ml) plastic growing trays (one plant per cell) filled with a sterilized potting mix of peat and vermiculite (1:1) and placed in a greenhouse at 20–27 C. Control plant roots were pruned to about 2.5 cm and dipped in tap water only.

Plants were examined periodically, and the numbers of yellowed, necrotic, wilted, or dead seedlings were recorded. Final assessments of the wilt reaction were made 28 days after inoculation.

Plants free of wilt symptoms were considered resistant. At the end of a test, selected resistant plants were transplanted into 10-L pots and grown to maturity for either self- or cross-pollinations.

Differential cultivars Charentais T, Doublon, and CM 17-187 as well as the parents PMR 45, Perlita FR, and WI 998FR were included in each test to ensure that there was no change in pathogen virulence or race.

RESULTS

Reaction of WI 998FR to races 0, 1, 2, and 1,2. Twenty-six seedlings of WI 998FR and 10 seedlings of each of the three differential cultivars were inoculated with the races of *F. o.* f. sp. *melonis* over the range of inoculum concentrations. The breeding population WI 998FR was resistant to races 0 and 2 at the highest inoculum concentration (1.0×10^6 conidia per milliliter) and susceptible to races 1 and 1,2 at the lowest concentration (0.025×10^6 conidia per milliliter).

Inheritance of resistance to races 0 and 2. Crosses between WI 998FR and PMR 45 produced F_1 progenies resistant to both races 0 and 2. The segregation observed in the F_2 generation (Table 1) suggested simple inheritance (3:1) of the disease reaction, with resistance controlled by a single dominant gene.

To verify the pattern of resistance to races 0 and 2, the F_1 was backcrossed to susceptible PMR 45. The BC_1 progenies of this cross gave a good fit to a 1:1 ratio of resistant to susceptible plants. The F_1 backcrossed to resistant WI 998FR resulted in all resistant progenies (Table 1). The segregation of the F_1, F_2, and BC_1 progenies clearly support the single-gene hypothesis.

Allelism tests. Crosses between WI 998FR, which has a dominant gene for resistance to races 0 and 2, and Perlita FR, which has the dominant gene *Fom*-3 (12) that confers resistance to races 0 and 2, segregated to a ratio of about 15:1 (resistant:susceptible) when inoculated with race 0 or 2 (Table 2). Two different, independently inherited genes for resistance are indicated in WI 998FR and Perlita FR.

WI 998FR was also crossed with Doublon, reported to have the dominant gene *Fom*-1 (8,12) that confers resistance to both races 0 and 2. The evidence from the allelism test of F_2 (WI 998FR × Doublon) indicates the same gene (*Fom*-1) confers resistance to races 0 and 2 in WI 998FR (Table 2).

DISCUSSION

The WI 998 population was derived from material described by Rowe (10) and traces back to a hermaphrodite × monoecious cross made at Michigan State University in 1960 (6). Seed of the two parents—the hermaphrodite line secured from Poole and Grimball (7) and a line identified as "Morden Monoecious" provided by the Research Station at Morden, Manitoba, Canada—is no longer available. Therefore, the source of Fusarium wilt resistance in WI 998 cannot be traced.

Heterosis has been demonstrated in F_1 hybrid muskmelon (1–3), but massive production of hybrid seed has been hindered by hand-labor requirements and technical problems. The use of gynoecious sex expression (5,6,10) in muskmelon hybrid seed production eliminates hand pollination, chemical treatments, and manual roguing, which are necessary in F_1 hybrid seed production fields when andromonoecious, monoecious, or genetic male-sterile lines are used as parents. Gynoecious sex expression offers the potential of providing F_1 seed of lower cost. The gene *Fom*-1 in gynoecious WI 998FR could be used directly to secure Fusarium wilt-resistant F_1 hybrids, thus avoiding a longer breeding program that would be required to develop early-maturing Fusarium wilt-resistant, true-breeding cultivars.

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Table 1. Segregation in progenies from crosses between resistant (R) breeding line WI 998FR and susceptible (S) cultivar PMR 45 after inoculation with race 0 or race 2 of *Fusarium oxysporum* f. sp. *melonis*

Parents and crosses	Expected ratio	Observed (no.) R	Observed (no.) S	χ^2	*P*
Inoculated with race 0					
WI 998FR (W)	All R	18	0	...	...
PMR 45	All S	0	20	...	...
F_1 W × PMR 45	All R	42	0	...	...
PMR 45 × W	All R	50	0	...	...
F_2 W × PMR 45	3:1	74	20	0.79	0.5–0.3
PMR 45 × W	3:1	70	28	0.67	0.5–0.3
BC_1 (W × PMR 45) × PMR 45	1:1	79	66	1.16	0.3–0.2
(PMR 45 × W) × PMR 45	1:1	64	71	0.36	0.7–0.5
BC_1 (W × PMR 45) × W	All R	48	0	...	...
(PMR 45 × W) × W	All R	51	0	...	...
Inoculated with race 2					
WI 998FR (W)	All R	17	0	...	...
PMR 45	All S	0	18	...	...
F_1 W × PMR 45	All R	50	0	...	...
PMR 45 × W	All R	47	0	...	...
F_2 W × PMR 45	3:1	77	19	1.38	0.3–0.2
PMR 45 × W	3:1	78	21	0.76	0.5–0.3
BC_1 (W × PMR 45) × PMR 45	1:1	41	50	0.89	0.5–0.3
(PMR 45 × W) × PMR 45	1:1	47	43	0.18	0.7–0.5
BC_1 (W × PMR 45) × W	All R	50	0	...	...
(PMR 45 × W) × W	All R	46	0	...	...

Table 2. Segregation for susceptibility (S) or resistance (R) in progenies from crosses between resistant WI 998FR and resistant Doublon or resistant Perlita FR after inoculation with race 0 or race 2 of *Fusarium oxysporum* f. sp. *melonis*

Parents and crosses	Observed (no.) R	Observed (no.) S	χ^2 (15:1)	*P*
Inoculated with race 0				
PMR 45	0	26	...	...
WI 998FR (W)	10	0	...	...
Doublon (D)	17	0	...	...
Perlita FR (PFR)	17	0	...	...
F_2 (D × W)	97	0	...	...
(W × D)	93	0	...	...
F_2 (PFR × W)	72	4	0.13	0.9–0.7
(W × PFR)	140	12	0.70	0.5–0.3
Inoculated with race 2				
PMR 45	0	25	...	...
WI 998FR (W)	17	0	...	...
Doublon (D)	17	0	...	...
Perlita FR (PFR)	17	0	...	...
F_2 (D × W)	88	0	...	...
(W × D)	97	0	...	...
F_2 (PFR × W)	167	8	0.84	0.5–0.3
(W × PFR)	163	15	1.43	0.3–0.2

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