Identification of the Races of Fusarium oxysporum f. sp. melonis Causing Wilt of Muskmelon in California

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ABSTRACT

Fusarium oxysporum f. sp. *melonis* was identified as the cause of wilt in two fields in Riverside County, California. The isolates of the fungus were tested on muskmelon cultivars differentially resistant to Races 1 to 4 of *Fusarium oxysporum* f. sp. *melonis*. Results showed that these isolates were of Races 2 and 3 that were previously unreported in the U.S.

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Since 1930 Fusarium wilt of muskmelon (*Cucumis melo*) has been a serious disease in the U.S. (2) where it has been reported as the cause of severe crop losses in several areas. The occurrence of the disease in California has been recorded (6), but whether this was due to *Fusarium oxysporum* f. sp. *melonis* (Leach and Currence, Synder and Hansen) is unconfirmed.

Although the existence of physiological races of *Fusarium oxysporum* f. sp. *melonis* has been confirmed (5), only Race 1 was known to occur in the United States.

In 1972, Fusarium wilt was identified as the cause of the loss of 100% of the crop in a field in Arlington, Riverside County, California. *Fusarium oxysporum* f. sp. *melonis* was isolated from the diseased plants. In 1973, another report of the disease from Riverside County was received and confirmed. The field was 15 miles from that field where the original isolate was obtained. Preliminary tests showed these isolates to be highly pathogenic to all the cultivars of muskmelon tested. Of particular interest was the fact that several muskmelon cultivars resistant to Race 1 were susceptible. These isolates were tested against melon cultivars used to differentiate among Races 1, 2, 3, and 4 (5). We report here the results of these tests which indicate that one isolate is Race 2 and the other is probably Race 3.

The original isolates of *Fusarium oxysporum* f. sp. *melonis* were obtained from sections of diseased plant tissue. The sections were surface disinfected with 1.0% sodium hypochlorite (10% chlorox) for 10 minutes and transferred to PCNB peptone-agar (4). Resultant colonies were transferred to potato-dextrose agar, identified as *Fusarium oxysporum* by microscopic examination, and transferred to glucose-peptone agar (GPA).

Isolates were also recovered from soil collected from fields in which the disease had been identified. A modified Anderson Air Sampler (1) was used to distribute 0.1 g of soil samples previously screened to pass a 38-μm screen over 300 areas of petri dishes containing water agar. Fungal colonies were identified as *Fusarium oxysporum* and tested for pathogenicity.

Cantaloupe seeds were germinated in steamed UC-planting mix (3). At the four- to six-leaf stage, the seedlings were inoculated by dipping the roots in a suspension containing approximately 10^5 conidia/ml. Four inoculated seedlings were transplanted to a 15.2-cm diameter pot for 12 plants per experiment. The cultivars tested were Charentais T, Doublon, and CM 17.187. The differential resistance is shown in Table 1.

Inoculation of the varieties Charentais, Doublon, and CM 17.187 with a known *F. oxysporum melonis* Race 1 isolate gave the predicted reaction (Tables 1 and 2). When these same cultivars were inoculated with conidia of the California isolates, the isolate designated X-22 gave the reactions expected for Race 3, and the isolate designated X-38 gave the reactions characteristic of Race 2 (Table 2).

TABLE 1. Differential resistance to *Fusarium oxysporum melonis* as described by Risser et al. (4)

	Fusarium oxysporum melonis isolate:		
Melon cultivar	Race 1	Race 2	Race 3
Charentais	S[a]	S	S
Doublon	R	S	S
CM 17.187	R	R	S

[a]R = resistant, S = susceptible.

TABLE 2. Differential resistance of melon cultivars to *Fusarium oxysporum melonis* isolates from California

	Fusarium oxysporum melonis isolate:		
Melon cultivar	I-498 from Israel[b] (Race 1)	X-38 from Moreno, CA (Race 2)	X-22 from Arlington, CA (Race 3)
Charentais	S[a]	S	S
Doublon	R	S	S
CM 17.187	R	R	S

[a]R = resistant, S = susceptible.
[b]Supplied by Dr. David Netzer.

The cultivar responses were clearly different from those of the known Race 1 isolate and from each other. These results are in general agreement with those of Risser et al. (5). However, those authors describe the response of Doublon to Race 2 as yellowing and tissue decay. In our tests, several isolates of the fungus did produce such symptoms, whereas others, obtained from the same field produced wilting and death of Doublon plants.

These data indicate that the California isolates are not members of Race 1, the only race reported previously in the United States. The fact that two different races were isolated from such a limited geographical area is puzzling, especially since there has been no noticeable spread of the disease into other melon-growing areas of the state. We are continuing our surveys of the major melon-growing areas to assess the disease incidence outside Riverside County.

Studies are now under way to identify resistance to Race 2 and Race 3 of *Fusarium oxysporum melonis* in commercial American muskmelon cultivars.

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