Seedling Disease of Muskmelon and Mixed Melons in California Caused by *Fusarium equiseti*

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ABSTRACT

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The cause of a seedling disease of *Cucumis melo* in the southern San Joaquin Valley, Kern County, California, was identified as *Fusarium equiseti*. Symptoms were reproducible on seedlings grown in soil at cool temperatures, 13–24 C (air temperature 27–33 C), when soil surrounding the hypocotyl was allowed to dry. In soils fumigated with chloropicrin and reinfested with *F. equiseti* (10^4 macroconidia per gram of air-dried soil), 47% of *C. melo* seedlings damped-off and the survivors were 32% smaller in fresh weight compared with seedlings in fumigated noninfested soils 22 days after sowing. Osmotically priming seed to increase the velocity of emergence did not influence disease severity. The pathogen causes characteristic dry cortical rot on hypocotyls of many species in the Cucurbitaceae; all *C. melo* tested were severely affected, whereas all *Cucurbita* species were low to intermediate in resistance. The fungus was readily isolated from plants that did not show lesions. In field soils containing diseased seedlings, viable pathogen propagules ranged from 300 to 500/g of air-dried soil. The disease was not observed in more northern or central melon production areas of California.

Additional key words: *Cucumis sativa*

Commercial cultivation of muskmelon and mixed melons (*Cucumis melo* L., Reticulatus and Inodorus groups) in California extends from the northern (39° latitude) to the southern border (32° latitude) in semiarid valleys. Damping-off of melon seedlings incited by *Pythium* spp. and *Rhizoctonia solani* Kühn is ubiquitous in the region, but recently, a more severe seedling disease caused by *Cephalosporium cucurbitaerum* Gubler & Grogan was identified and described (5). Routine isolations from diseased seedlings recovered this pathogen in the Sacramento Valley and in the northern and central San Joaquin Valley. In the southern San Joaquin, however, a disease of similar symptomatology was observed, but isolations were negative for *C. cucurbitaerum*. This paper describes the etiology of the disease in the southern San Joaquin Valley. The influences of soil temperature, soil moisture, and velocity of seedling emergence on host susceptibility are studied, and crop rotation is discussed.

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MATERIALS AND METHODS

Diseased seedlings of *C. melo* (Persian, Crenshaw, Golden Beauty casaba, and honeydew) and propagule-infested soil samples were collected from fields where poor stands occurred when planting dates were not followed by warmer soil temperatures within a 2-wk period. Seedlings were washed for 1 hr under running water, surface-sterilized in a 1% solution of sodium hypochlorite for 10 min, and blotted dry on paper towels. Isolations were made from seedling hypocotyl segments cut from just above the margin of the lesion. Hypocotyl segments were embedded in 1% water agar in petri dishes and incubated at room temperature (24 C). Isolates of *Fusarium* spp. were identified after transfer of spores from a sporodochium to autoclaved blades of grass on the surface of 2% water agar in petri dishes. Cultures were incubated under lights (cool-white fluorescent lamps; 17 hr light/7 hr dark) for 7 days at 25–27 C.

Pathogenicity trials and host range studies. Pathogenicity trials and host range studies were conducted in fumigated field soil (Gila fine sandy loam) into which the fungus was reintroduced. Soils were fumigated in pots containing 1 L of air-dried soil (sieved through a 2-mm mesh) by adding 200 ml of a 0.5% aqueous solution of trichloronitromethane. The pots were doubly sealed in polypropylene bags for 2 days at 27 C. After fumigation, soil was air-dried and infested with *Fusarium equiseti* (Corda) Sacc. (sensu stricto [2]) by moistening to field capacity with a 200-ml aqueous suspension of macroconidia, 10^4 ml^{-1}. Soils of the controls were moistened with 200 ml of water. Macroconidia used in pathogenicity trials were derived from a single-spore culture. Conidia were harvested from cultures grown on grass blades on water agar.

Seed was surface-sterilized 10 min in 1% sodium hypochlorite and rinsed in sterile water. Ten seeds were then embedded in potato-dextrose agar (PDA) in tubes to check for contamination, and 10 seeds per pot (9 × 9 × 20 cm) were planted 2.5 cm deep in soil. Soil was then covered sparsely with perlite. In host range tests, five seeds of *C. melo*, Reticulatus group 'PMR-45' were planted in each pot as susceptible controls along with five seeds of the host being tested for susceptibility. Four watertight pots per host were partially immersed in a temperature-controlled, circulating water bath so that the water surface on the outside of the pot was level with the soil surface. Water temperature was set at 16 ± 1 C during general pathogenicity and host range tests and at 16, 24, and 32 C in separate tests of host susceptibility. The water bath was in a glasshouse with controlled air temperatures of 27–33 C. Emerging seedlings were not watered, and surface soil was allowed to dry to mimic field conditions. Seedlings were harvested 22 days after sowing, counted, and fresh weight and height measured. Fresh weight and height were measured from the root-hypocotyl junction. Fresh measurements were taken so that reisolation of the pathogen could be made from the hypocotyls. The severity of disease symptoms was assessed and rated from 0 to 3 as follows: 0 = no symptoms, 1 = minute superficial dry lesions on the root surface at the junction of the root and hypocotyl, 2 = superficial dry lesion 2–5

mm long on hypocotyl, and 3 = deep dry lesion 5–15 mm long in cortical tissue of hypocotyl. Experiments were repeated three times.

Estimation of inoculum density of *Fusarium* spp. in soil. Eight soil samples (Gila fine sandy loam) were taken from each of three fields showing poor stands, and samples from each field were mixed. The soil samples were air-dried and diluted 1:1,000 in 0.1% water agar. One milliliter of soil suspension was plated on the selective, acidified peptone-PCNB medium (8,9). After 5 days of growth at 27 C, *Fusarium* cultures were transferred to acidified PDA and pigmentation of the medium beneath the colonies was noted after an additional 5 days of incubation at 27 C. *Fusarium* colonies showing peach, buff, yellow, beige, or brown pigmentation were transferred to autoclaved grass blades on 2% water agar and incubated 7 days at 27 C under cool-white fluorescent lamps (17 hr light/7 hr dark). Sporodochia on autoclaved grass blades were examined microscopically for identification to species according to Booth's classification (1).

Host-pathogen temperature interrelationships. The susceptibility of a seedling to damping-off at specific temperatures often agrees closely with the ratio of the rate of seedling emergence to the rate of fungal growth (7). The severity of the melon seedling disease in Kern County appeared to be associated with cool soil temperatures; thus, it was of interest to determine if the calculated host-pathogen temperature interrelationship agreed with experimental results.

Cups of fumigated soil (Gila fine sandy loam), moistened to field capacity, were placed in incubators (9, 12, 15, 18, 21, 24, 27, 30, 33, and 36 C) to determine the rate of seedling emergence of cantaloupe (*C. melo*, Reticulatus group 'PMR-45'). After the soil equilibrated to temperature, 10 seeds were planted in each of five cups and the cups were placed into a sealed plastic bag to maintain uniform soil moisture. Emerged seedlings were counted daily, and the rate of emergence at each temperature was calculated (7) by the following formula: rate of emergence = [total emergence 15 days after planting × 100]/[sum of (each daily emergence increase × days since planting)].

Flasks of potato-dextrose broth (20 ml/250-ml flask) were inoculated with *F. equiseti* in triplicate and incubated at each temperature alongside the seedlings. The mean growth rate (mg day^{-1}) of *F. equiseti* was determined after 15 days of growth at each temperature by measuring dry weight of mycelium after drying at 90 C for 24 hr. A ratio of seedling emergence to fungal growth rate was calculated, and the three sets of data were graphed.

Emergence tests with osmotically primed seed. To test the hypothesis that an increased rate of seedling emergence would decrease the susceptibility of the host to the pathogen, seed was osmotically primed, sown in cool soils (16 C), challenged with *F. equiseti*, and compared with untreated seed as follows. Osmotically primed seed (cantaloupe PMR-45; soaked 6 days in aerated 3% KNO_3 at 25 C, rinsed, and dried) and untreated seed from the same seed lot was obtained from Kent Bradford, Department of Vegetable Crops, University of California, Davis. Ten seeds were planted in each of 28 deep pots (9 × 9 × 20 cm). All pots contained fumigated soil (Gila fine sandy loam), and 14 pots were reinfested with *F. equiseti* at 10^4 macroconidia per gram of air-dried soil (as described previously). The pots were incubated at 16 C in a circulating water bath in a greenhouse, air temperature 27–33 C. The emerged seedlings were counted twice a day during the emergence period, and postemergence damping-off was recorded. Seedling fresh weight and height were measured after 22 days, about 17 days after emergence. The experiment was repeated three times.

RESULTS

Pathogenicity. *F. equiseti* was isolated from 100% of 226 field-grown seedlings (*C. melo* 'Persian,' 'Crenshaw,' and 'Golden Beauty' casaba) showing cankers on the hypocotyls. In addition to *F. equiseti, Chaetomium* spp. were isolated from 11% of the seedlings; *F. solani* was not isolated. In fumigated soils reinfested with *F. equiseti* macroconidia, seedlings showed cankers on the hypocotyls only when soil temperatures ranged from 13 to 25 C and soils surrounding the hypocotyls were allowed to dry during emergence. *F. equiseti* was isolated from seedlings grown in cool soils moistened to field capacity, but cankers and postemergence damping-off were not observed if the soil surface remained moist. Preemergence damping-off was not evident in fumigated soils reinfested with *F. equiseti*. However, survival of seedlings was significantly different from controls by the sixth day after emergence (11 days after sowing), about 30% less survival than controls (Table 1). Thereafter, survival progressively diminished with the onset of postemergence damping-off (Table 1). *F. equiseti* was isolated from 100% of the damped-off seedlings. Those seedlings that survived were stunted and their fresh weight and height were significantly less than controls, about 68 and 74%, respectively (Table 2). Although cankers were present, about 57% of the stunted seedlings survived beyond 17 days after emergence (22 days from sowing). *F. equiseti* was isolated from 100% of the stunted and cankered seedlings that survived. An isolate of *F. equiseti* from cotton seed and one from wheat culms also caused cankers and stunting of *C. melo* seedlings in cool soil. A low percentage (less than 1%) of surface-sterilized seed of *C. melo* yielded fusaria

Table 1. Daily statistical comparisons of the survival of *Cucumis melo* seedlings in soil infested with *Fusarium equiseti* versus fumigated soil (control)[a]

	Level of significant difference (%) between soil treatments					
	Days after emergence					
Test	5	6	7	9	11	13
1	NS[b]	7.9[c]	1.4	0.3	0.1	0.1
2	NS	4.9	2.3	0.6	0.2	0.1
3	NS	9.6	1.3	0.5	0.1	0.1

[a] Comparisons are expressed as the percent probability that the observed decrease in survival in infested soil could be expected to occur by chance (i.e., percent significance level).
[b] NS = mean survival in infested soil is not significantly less than fumigated soil treatment (control).
[c] Percent level of significant difference calculated by one-way analysis of variance. When a significant difference in survival occurred, the survival was less in infested soil in all instances.

Table 2. Survival, fresh weight, and height of *Cucumis melo* seedlings 15 days after emergence in fumigated soil reinfested with *Fusarium equiseti* compared with control seedlings in fumigated soil

Replicated tests	Soil treatment	Mean fresh weight (g)	Mean height (mm)	Mean survival (%)
1	Control	0.68	9.25	98.0
	F. equiseti	0.46	7.85	43.0
	Difference (%)	67.50**[a]	84.70**	43.9**
2	Control	0.61	9.74	97.0
	F. equiseti	0.37	6.37	62.5
	Difference (%)	60.70**	65.40**	64.5**
3	Control	0.57	11.14	100.0
	F. equiseti	0.44	7.98	62.5
	Difference (%)	76.20	71.60**	62.5**

[a] Mean of measurements of plants in infested soil divided by the mean of measurements in fumigated soil × 100; ** = a significantly lower value ($P = 0.05$) in infested soil.

Table 3. Disease reactions of plants after growing 22 days in cool soil (16 C) infested with *Fusarium equiseti*

Host		Severity of symptoms[a]	Isolation of *F. equiseti* from tissue[b]
Common name	**Latin name**		
Cantaloupe	*Cucumis melo* L., Reticulatus group	3	+
Persian	*C. melo*, Reticulatus group	3	+
Honeydew	*C. melo*, Inodorus group	3	+
Crenshaw	*C. melo*, Inodorus group	3	+
Juan Canari	*C. melo*, Inodorus group	3	+
Casaba	*C. melo* 'Golden Beauty,' Inodorus group	2	+
Casaba	*C. melo* 'Santa Claus,' Inodorus group	3	+
Snake melon	*C. melo*, Flexuosus group	3	+
Oriental pickling melon	*C. melo*, Conomon group	3	+
Gherkin	*C. anguria*	1	+
Cucumber	*C. sativa*	3	+
Pumpkin	*Cucurbita pepo* L. var. *pepo* (L.) Alef.	1	+
Ornamental gourd	*C. pepo* var. *ovifera* (L.) Alef.	0–1	+
Bush pumpkin	*C. pepo* var. *melopepo* (L.) Alef.	2	+
Winter squash	*C. moschata* (Duch.) Poir	1	+
Marrow	*C. maxima* Duch.	1	+
Cushaw	*C. mixta* Pang.	1	+
...	*C. andeana* Naud.	1	+
Watermelon	*Citrullus lanatus* (Thunb.) Matsum. & Nakai	0	+
Calabash	*Lagenaria siceraria* (Mol.) Standl. 'Kettle'	0	+
Luffa	*Luffa aegyptiaca* Mill.	0	+
Bitter melon	*Momordica charantia* L. var. *chinensis*	1	+
Chinese preserving melon	*Benincasa hispida* (Thunb.) Cogn. var. *chieh-que*	2	+
Cumin	*Cuminum cyminum* L.	0	+
Maize	*Zea mays* L. var. *rugosa* Bonaf.	3	+
Cotton	*Gossypium hirsutum* L.	0	+
Wheat	*Triticum aestivum* L.	0	+
Onion	*Allium cepa* L.	0	0

[a] Increasing numbers correspond to increase in severity of canker on the hypocotyl: 0 = no symptoms, 1 = minute superficial dry lesions on the root surface at the junction of the root and hypocotyl, 2 = superficial dry lesion 2–5 mm long on hypocotyl, 3 = deep dry lesion 5–15 mm long in cortical tissue of hypocotyl.

[b] Hypocotyls surface-sterilized for 15 min in 1% sodium hypochlorite and embedded in 1% water agar.

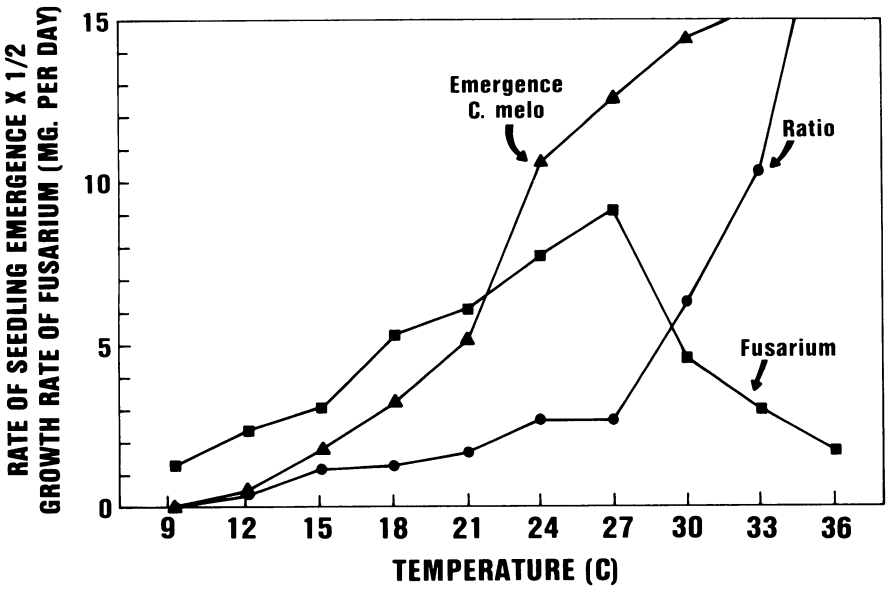

Fig. 1. Temperature interrelationship between *Cucumis melo* and *Fusarium equiseti*: host emergence rate, growth rate (mg day^{-1}) of the pathogen, and the ratio of the emergence rate to the growth rate. Rate of seedling emergence =[total emergence 15 days after planting × 100]/[sum of (each daily emergence increase × days since planting)]. Ratio = [rate of seedling emergence of *C.* melo]/[growth rate of *F. equiseti* in mg day^{-1}].

(*Fusarium oxysporum*) when plated on PDA; however, none of the seedborne fusaria were *F. equiseti*.

Host range. Symptoms on *Cucumis anguria, Cucurbita* spp., and *Momordica charantia* were scarcely evident, with minute (3–5 mm long) tan superficial lesions encircling the root at the root-hypocotyl junction. A black lesion on the lower stem occurred on infected maize. Golden Beauty casaba, summer crookneck squash, and Chinese preserving melon had intermediate symptoms, whereas watermelon, gourds, luffa, cotton, wheat, cumin, and onion appeared immune to the cankering disease (Table 3). *F. equiseti* was readily isolated from all Cucurbitaceae seedlings grown in infested soil as well as cumin, maize, cotton, and wheat (Table 3) regardless of the lengthy period of surface disinfestation (15 min) before isolation. *C. melo* and *C. sativa* were severely diseased with deep cankers on the hypocotyls.

Inoculum density. Air-dried soil samples from fields containing diseased seedlings had about 3,300 propagules per gram of *F. oxysporum* and *F. solani*, 1,300 propagules per gram of *F. roseum*, and 300–500 propagules per gram of *F. equiseti*.

Temperature interrelationships. The pathogenicity of *F. equiseti* on *C. melo* was influenced by soil temperature. Seedlings showed disease symptoms (cankers or stunting) when soil temperatures in the root zone were 16 or 24 C but not at 32 C. Furthermore, seedlings with cankers apparently recovered from disease when soils subsequently reached 30 C. During recovery, lateral roots developed from the hypocotyl tissue above a canker. Seedlings with large cankers that girdled the hypocotyl succumbed regardless of the change in soil temperature. The severity of infection at different temperatures was not correlated closely with the growth of the pathogen expressed as increase in dry

weight of mycelium (milligrams per day) (Fig. 1). Also, the severity of infection was not correlated with the rate of emergence of the host at different temperatures. However, the slope of the ratio between the host emergence rate and the pathogen's growth rate at the same temperature conformed closely with host susceptibility (severity of postemergence damping-off, cankering, and stunting). The flat slope of the ratio line (Fig. 1) corresponded with host susceptibility and the transition to a steep slope was correlated with host resistance and potential for recovery.

Emergence tests with osmotically primed seed. About 50% of the osmotically primed seed germinated within 3 days and 91% within 5 days on damp blotting paper at 19 C compared with 0 and 7% germination of control seed. The final percentage of germination of primed seed was 91 compared with controls of 43% after 16 days (K. Bradford, *personal communication*). However, in soil tests, we saw little difference in velocity of emergence between primed and non-primed seed. Untreated seed of *C. melo* germinated more quickly and uniformly when sown in soil than on damp blotting paper. Differences in the rate of emergence between seed treatments usually were not statistically significant beyond the first day of emergence (5 days after sowing) (Table 4). In infested soil, postemergence damping-off of seedlings from primed seed was not significantly different from damping-off of seedlings from untreated seed. Seedlings from osmotically primed seed were not significantly different from controls in fresh weight, height, final emergence, or survival 22 days after sowing in infested or fumigated soil.

DISCUSSION

Although *F. equiseti* has been isolated from numerous agronomic hosts including cucurbits (4,6), its pathogenicity has been opened to question. In California, *F. equiseti* was isolated from internal tissues of cotton seed (11) and was recognized as a minor pathogen causing root and culm rot of wheat (9). A cotton and a wheat isolate were tested and found to be pathogenic on *C. melo* in the present study. Wheat and cotton are the primary crops rotated with mixed melons and muskmelon in the southern San Joaquin Valley, and the ability of *F. equiseti* to colonize cotton gin trash and wheat stubble incorporated into soil as well as to parasitize melon seedlings could result in increases of inoculum density. Also, land leasing practices in the area may influence disease severity. Rental charges are based on the potential earnings from a high-value-per-acre crop, thus discouraging rotation with many lower value crops.

In fields cropped to melons for successive years, disease caused by *F. equiseti* was more prevalent. This fact suggests that rotation with wheat and cotton crops may not be the major influence on disease incidence on subsequent melon crops. Cotton seed could be the initial source of *F. equiseti* contamination. However, the soils probably have resident populations of *F. equiseti* because of its ubiquitous distribution in arid and semiarid temperate and subtropical soils (1).

The temperature interrelationships are clearly expressed in the slope of the ratio of the host emergence rate to the pathogen growth rate (Fig. 1). Cankering, stunting, and postemergence damping-off were most severe at temperatures corresponding to the flat slope (lowest ratios) and no disease occurred above 30 C (the steepest slope) in three trials. The conclusion supported by the results is that planting at soil temperatures more favorable for emergence (27 C or higher) would encourage escape from *F. equiseti* infection. This obvious choice for the grower may be precluded by economic considerations. The region where *F. equiseti* causes damage is a melon-growing area primarily because early crops mature at a time when limited market supply favors high prices (just after the harvest of the Imperial and Palo Verde valleys is consumed and before the more northern crops are harvested). A delay in crop maturity of only 2 wk may decrease the value of the harvest. Therefore, growers plant as early as possible, based on soil temperature measurements of about 13 C, with the expectation that soils will warm within a week.

Under field conditions, melon seeds usually are planted into soil moistened by furrow irrigation; the field is not subsequently irrigated until after emergence to avoid crusting of the soil surface. In attempts to reproduce the disease, we simulated the dry soil surface condition that often occurs in the field. Seeds were sown in deep pots containing prewetted soils and were not remoistened for 2 wk. Soils remained moist in the root zone, and the uninoculated control seedlings remained healthy.

The disposition of hosts to fusaria by xeric soil conditions or drought stress is known for several host-pathogen combinations (3,12). A mild water stress of maize, even though no visible symptoms of water deficit were apparent, disposed plants to infection and systemic colonization by *F. moniliforme* (12). Subsequently, under high evaporative demand conditions, the infected maize was chronically stressed regardless of adequate soil moisture. A mild water deficit during or after emergence could dispose *C. melo* seedlings to disease by *F. equiseti*. Additional research is needed to clarify the role of dry soil surrounding the hypocotyl on the susceptibility of *C. melo* to *F. equiseti*.

The degree of susceptibility of other hosts in the Cucurbitaceae to cankering and stunting by *F. equiseti* may involve general resistance or, alternatively, a tolerance to undefined disposing influences of dry surface soil and the plant's physiology in cool soils. Difficulty in reproducing disease symptoms reported for infected cumin (10) and wheat (4) may have been due to unfavorable disease conditions for the host-pathogen combination.

Table 4. Daily statistical comparisons of accumulated emergence of osmotically primed seed of *Cucumis melo* 'PMR-45' and controls from the same seed lot in fumigated soil and in soil infested with *Fusarium equiseti*[a]

		Level of significant difference (%) between seed treatments				
		Days after first emergence				
Test	Soil treatment	1	2	3	4	5
1	Fumigated	5[b]	18	NS[c]	NS	NS
	Fumigated + *F. equiseti*	0.5	11	15	23	NS
2	Fumigated	8	1	11	17	28
	Fumigated + *F. equiseti*	7	11	NS	NS	NS
3	Fumigated	2	4	12	17	13
	Fumigated + *F. equiseti*	6	NS	NS	NS	NS

[a] Comparisons are expressed as the percent probability that the observed greater emergence by primed seed could be expected to occur by chance (i.e., percent significance level).
[b] Level of significant difference between seed treatments as determined by one-way analysis of variance. When a significant difference existed, the primed seed emergence was greater in all instances.
[c] NS = mean emergence of primed seed is not significantly greater than control seed.

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