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Comparison of *Fusarium solani* and *F. oxysporum* as Causal Agents of Fruit Rot and Root Rot of Muskmelon

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Abstract. **Rotting muskmelon fruits commonly are associated with commercial fields that are affected by the root rot/vine decline disease syndrome found in southern Texas. Four isolates of *Fusarium solani* previously shown to be either weakly pathogenic or nonpathogenic to muskmelon seedlings caused extensive rot on mechanically wounded muskmelon fruits. Two of these isolates caused more extensive fruit rot than either *F. solani* (Mart.) Sacc. f. sp. *cucurbitae* W.C. Snyder & H.N. Hans. or *F. oxysporum* Schlechtend.:Fr. *melonis* (Leach & Currence) W.C. Snyder & H.N. Hans., causal agents of fusarium crown and foot rot of cucurbits and fusarium wilt of muskmelon, respectively. In other tests, root-dip inoculation of seedlings showed that all muskmelon cultigens included in this study and the breeding line MR-1 were susceptible to a California and an Arkansas strain of *F. s.* f. sp. *cucurbitae* race 1.**

In 1986, muskmelon producers in the Lower Rio Grande Valley (LRGV) of southern Texas suffered high economic losses due to root rot/vine decline disease, locally referred to as root rot (Champaco, 1990; Champaco et al., 1988). The disease has persisted through the 1993 season (information added during manuscript revision). Symptoms include leaf yellowing and dieback of the crown leaves, cortical rot of the taproot and lateral roots, and discoloration of the vascular system in the roots and crown, The disease has symptoms similar to fusarium wilt (caused by *Fusarium oxysporum* f. sp. *melonis)* and fusarium crown and foot rot (caused by *F. solani* f. sp. *cucurbitae).* However, based on several experiments performed under greenhouse conditions, numerous *F. solani* and *F. oxysporum* isolates recovered from taproots and lateral roots of infected field-grown muskmelon plants were nonpathogenic on various muskmelon cultivars, suggesting that these fungi were probably not the causal agents of root rot (Champaco, 1990). Recently, Mertely et al. (1991 and 1993) showed that *Monosporascus cannonballus* Pollack & Uecker was the primary casual agent of this disease.

Isolates of *F. solani* were recovered three to ten times more frequently from roots of plants showing symptoms of root rot (Champaco, 1990; Mertely et al., 1991) than were *F. oxysporum* isolates. Additionally, rotted fruits were often associated with the presence of root rot in commercial muskmelon fields, and these *Fusarium* spp. may serve as potential fruit rot pathogens. A previous report by Godfrey (cited in Toussoun and Snyder, 1961) cited *F. solani* as a dominant factor in rotting mature melon fruits under wet weather conditions in Texas. *Fusarium o.* f. sp. *melonis* (race 0) has been reported in Texas (Martyn et al., 1987); however, there has been no prior report of *F. s.* f. sp. *cucurbitae* in the state. Additionally, numerous isolates of *F. solani* taken from roots of muskmelon plants showing root-rot symptoms were not pathogenic in greenhouse inoculation studies, which suggests that *F. s.* f. sp. *cucurbitae* most likely is not a causal agent of root rot in Texas (Champaco, 1990).

Fusarium crown and foot rot disease was first reported on squash *(Cucurbitapepo* L.) in 1930 in South Africa (Doidge and Kresfelder, 1932). The disease has since been reported in the Americas, Europe, and Australia (Sherf and MacNab, 1986). It was reported on cucumber *(Cucumis sativus* L.) in glasshouses in the Netherlands in 1960 (Paternotte, 1987) and reappeared there in 1980, causing foot rot in courgette *(Cucurbita pepo* L.). *Fusariums.* f. sp. *cucurbitae* is not as widespread in the United States as is *F. o.* f. sp. *melonis.* The first report of *F. s.* f. sp. *cucurbitae* in the United States was in California in 1938 (Snyder, 1938). In 1970, Sumner (1976) isolated the pathogen from summer squash *(Cucurbita pepo* var. *melopepo)* in Georgia. In 1978, Boyette et al. (1984) isolated a strain of *F.s.* f. sp. *cucurbitae* from Texas gourd *[Cucurbita texana* (A.) Gray] in Arkansas. Much of the interest in *F. s.* f. sp. *cucurbitae* in Arkansas is in its potential as a biocontrol agent for Texas gourd, a persistent weed problem in cotton *(Gossypium hirsutum* L.) and soybean [Gly*cine max* (L.) Merr.] (Boyette et al., 1984; Weidemann and Templeton, 1988).

There are two described races of *F.s.* f. sp. *cucurbitae* (Toussoun and Snyder, 1961); however, recent work (Van Etten and Kistler, 1988) has separated these into two differing mating populations (MP), which most probably are different species. Race 1 is a root-, stem-, and fruit-rot pathogen; occurs worldwide; and is in MP-I. Race 2, which is pathogenic primarily to mature cucurbit fruits, has only been described from the United States (California and Ohio) and is included in MP-V. Pathogenic-isolates of *F. s.* f. sp. *cucurbitae* are generally heterothallic. The teleomorph is *Nectria haematococca* Berk. & Broome. Fusarium crown and foot rot has been reported only on pumpkin, squash, and marrow under field conditions; however, other cucurbitaceous hosts are susceptible under greenhouse inoculation conditions (R. D. M., unpublished; Tousson and Snyder, 1961). There is no identified resistance to *F.s.* f. sp. *cucurbitae* among the cultivated muskmelons.

The development of muskmelon breeding lines PI-12411lF by Cohen and Eyal (1987) in Israel and MR-1 by Thomas (1986) in the United States has stimulated considerable interest in muskmelon breeding. Both breeding lines were derived from PI- 124111 and have resistance to powdery mildew *[Sphaerotheca faliginea* (Schlechtend.:Fr) Pollacci], downy mildew *[Pseudoperonospora cubensis* (Berk. & M.A. Curtis) Rostovzev], and to races 0, 1, and 2 of *F. o.* f. sp. *melonis* (Zink and Thomas, 1990). The disease reaction of these lines to *F. s.* f. sp. *cucurbitae* has not been reported.

The objectives of the present study were to 1) compare the relative abilities of several isolates of *F. oxysporum* and *F. solani* to cause fruit rot under laboratory conditions and 2) evaluate the disease reaction of commercial muskmelon cultigens, germplasm, and MR-1 to two geographic isolates of *F. s.* f. sp. *cucurbitae* race 1 (California and Arkansas).

The fusarium strains used were obtained from the stock collection maintained at the Fusarium Laboratory at Texas A&M Univ. The isolate of *F. o.* f. sp. *melonis* race 0 originated from Maryland (E. Dutky, Univ. of Maryland, College Park). Two isolates of *F.s.* f. sp. *cucurbitae* race 1 were used. One was from California (J. Watterson, Petoseed Co., Woodland, Calif.) and the other from Arkansas (G. Weidemann, Univ. of Arkansas, Fayetteville). Four *Fusarium solani* isolates obtained from roots of infected muskmelon plants in commercial fields in Texas were used: lA-la, 34B-1, 63C-2, and 0629. All *F. solani* isolates, except the two crown and foot-

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rot isolates, included in this study were nonpathogenic or only weakly pathogenic on muskmelon seedlings in a previous greenhouse inoculation study (Champaco, 1990). Each isolate was maintained as single-spored cultures in sterile soil (McKeen and Wensley, 1961). Seeds of the cultivars and hybrids were obtained from commercial seed companies. The fusarium wilt host differentials, Doublon and 1088 CM 17-187, were obtained from F. Zink (Univ. of California, Davis). MR-1 was obtained from C. Thomas (U.S. Dept. of Agriculture/Agricultural Research Service, Charleston, S.C.).

Inoculum of each *Fusarium* isolate was increased by placing several granules of soil culture in 50 ml of fusarium mineral salts liquid medium (Esposito and Fletcher, 1961) contained in a 250-ml Erlenmeyer flask. The inoculum was incubated on a rotary shaker at 100 rpm held at 25 ± 2C under continuous fluorescent light (860 $\mu mol \cdot m^{-2} \cdot s^{-1}$) for 3 to 4 days. After incubation, the conidia were filtered through eight layers of sterile cheesecloth, and the conidial suspension was adjusted to 1×10^6 microconidia/ml using a hemacytometer.

The isolates used in the fruit-rot study were lA-la, 34B-1, 63C-2, 0629, *F. s.* f. sp. *cucurbitae* race 1 (California), and *F. o.* f. sp. *melonis* race O. Ripe muskmelon fruit (cultivar unknown) purchased from a local grocery store were washed and flamed with 70% ethanol. Five wounds, 7 mm in diameter were made on the surface of each fruit with a ethanol-flamed cork borer. Wounds were located at the stem end and blossom end of the fruit, and at three other locations around the circumference of the fruit. At each wound location, the plug of fruit was removed and 25 µl of fungal inoculum (1×10^6 suspension) was placed in the exposed flesh. The plugs were replaced to cover the holes after inoculation. Each fruit was inoculated with one isolate and there were three one-fruit replications for each isolate. Each fruit was covered with a clear plastic bag for 24 h and set on petri dishes (80 x 100 mm) maintained in a darkened growth room at ambient temperature (20C). At the end of 24 h, the plastic bags were removed, humidifiers and growth lights (12-h light/day cycle) were turned on, and the temperature maintained at ≈ 22C. Fruits were incubated under these conditions for 10 days. Length of time required for initial rot development, and the mean depth and mean maximum diameter (severity) of the rot caused by each isolate were recorded. The data were subjected to analysis of variance (ANOVA) and the means separated according to Duncan's multiple range test (et = 0.05).

Hybrid muskmelons are becoming increasingly more important in commercial production in Texas; however, no data on the disease response to *F. s.* f. sp. *solani* are available on these hybrids. In addition, MR-1 has received considerable attention because of its good resistance to several diseases. Therefore, muskmelon cultigens were tested against two strains of *F.s.* f. sp. *cucurbitae.* Seeds of the commercial cultigens were surface-disinfected in 10% bleach (0.525% NaOCl) for 60 see, rinsed in sterile distilled water, and planted in seedling flats containing a 4 vermiculite :4 perlite :1 peatmoss (by volume) potting mixture. At emergence of the first true leaves (≈10 days), seedlings were uprooted and the roots gently washed under running water to remove the surrounding potting medium. Seedling roots were dipped in the respective inoculum or water (the control treatment) for 20 to 30 sec and transplanted into 1.25-liter (15-cm-diameter) pots containing an 8 pasteurized sand :2 vermiculite :2 perlite : 1 peatmoss mixture. Depending on the number of available seedlings, there were two, three, or four seedlings per pot, and three or four replications of each treatment. Inoculated seedlings were maintained under greenhouse conditions for 4 weeks. Observations were made at 1-to 2-day intervals and the percentage of dead plants recorded. Due to time and space limitations, all cultivars were not included in any one test. However, two internal standard cultivars were included in each test; there was no significant difference in disease seventy between tests, which indicates there was no cultivar × experiment interaction. Consequently, the data presented in Table 1 are the combined data of two experiments performed over 1 year. Doublon, 1088 CM17-187, 'Perlita', and 'Topmark' were the fusarium wilt host differentials used to identify the various races of *F. o.* f. sp. *melonis.* Data were transformed by arcsin transformation and weighted before ANOVA. The means were separated using Fisher's LSD test ($\alpha = 0.05$).

All isolates caused rot in the muskmelon fruit (Fig. 1). Mycelial growth was evident 4 days after inoculation at all wound locations and initial symptoms of rot also were present at that time. *Fusarium solani* isolate 34B-1 caused the most extensive rot, with 5.5 cm mean maximum diameter and 3.4 cm mean maximum depth of lesions (Fig. 2 A and B). *Fusarium o.* f. sp. *melonis* caused the least amount of rot, with 3.8 cm mean maximum diameter and 2.3 cm mean maximum depth. The extent of fruit rot caused by isolate 34B-1 and *F. o.* f. sp. *melonis* was statistically($\alpha =$ 0.05) different. All remaining isolates caused fruit rot, but they were not statistically different from each other in mean maximum depth, except when compared to the water control (Fig. 2B). Fruit rot severity, based on inoculation site, did not differ, as rot seventy in the stem end, blossom end, or along the circumference of the fruit was similar. Initial observations, however, showed that fungus colonized the stem end and along the fruit circumference before colonizing the blossom end. A musky odor was associated with fruit inoculated with the test isolates, but not with fruit inoculated with *F. o.* f. sp. *melonis* or water.

The absence of any statistical difference in fruit rot severity based on inoculation site is due perhaps to wounding before inoculation, which allowed the isolates an equal opportunity, in spite of location, to colonize the fruit. We presumed that, under field conditions, the pathogen enters the fruit through natural wounds (netting cracks) or from wounds caused by injury due to insects, wind-blown sand, or mechanical disturbances. In general, *F. solani* isolates that are only weakly pathogenic or nonpathogenic to seedlings apparently can be extensive fruit rotters. Isolates 34B-1 and 63C-2 caused more extensive fruit rot than either *F. s.* f. sp. *cucurbitae* or *F. o.* f. sp. *melonis.* Pathogenicity of these two *F. solani* isolates to young plants, however, is questionable. In an earlier greenhouse inoculation study (Champaco, 1990), isolates 34B-1 and 63C-2 caused severe stunting and necrotic petioles, respectively, in young muskmelon seedlings. However, when a similar study was repeated, none of these symptoms occurred and only slight necrosis was observed on the roots.

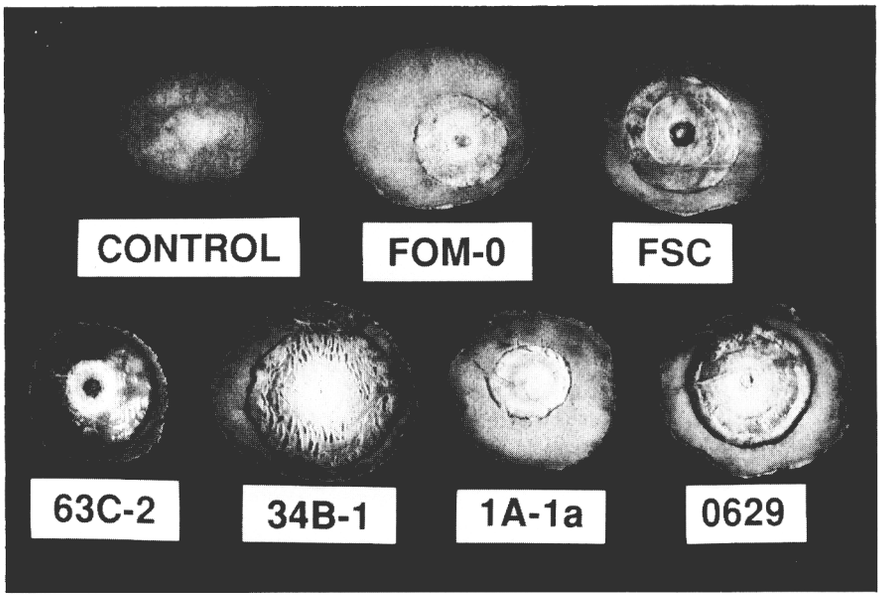

Fig. 1. Comparison of *Fusarium oxysporum* f. sp. *melonis, F. solani* f. sp. *cucurbitae*, and other *Fusarium solani* isolates in causing fruit rot of muskmelon as shown by the inner portion of inoculated fruits. Each fruit slice represents the first outer 5- to 10-mm depth.

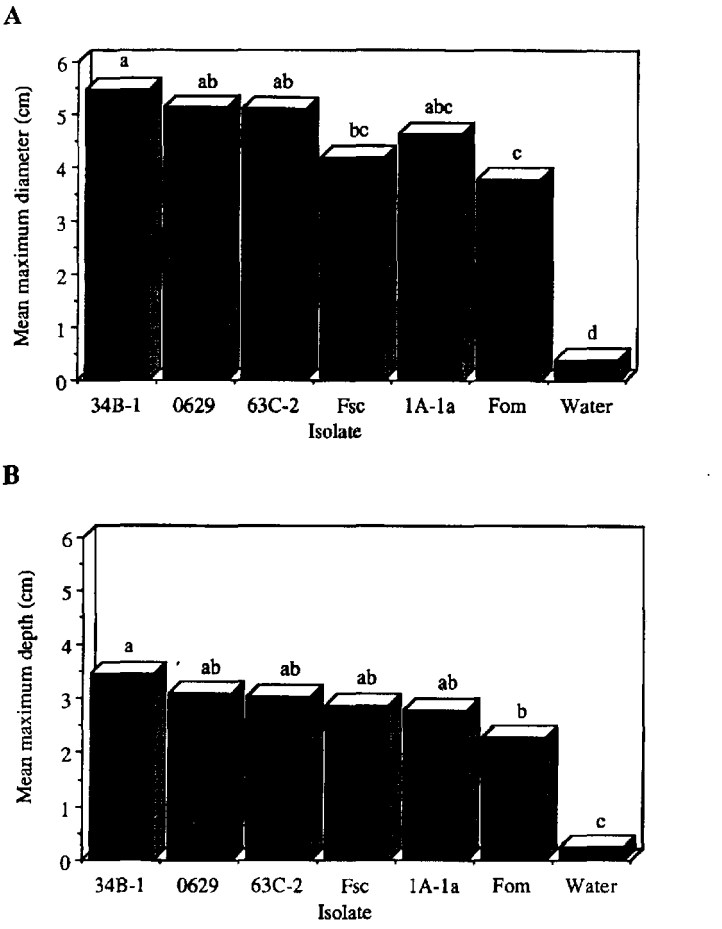

Fig. 2. Severity of muskmelon fruit rot caused by various isolates of *Fusarium* spp. (**A**) Mean maximum diameter of lesion. (**B**) Mean maximum depth of rot. Fsc = *Fusarium solani* f. sp. *cucurbitae* (race 1, California strain); Fom = *Fusarium oxysporum* f. sp. *melonis* race 0 (Maryland). All other isolates of *F. solani* were from roots of plants collected in Texas.

We conclude from the current fruit-rot study that apparently saprophyticisolates of *F. solani* originating from root-rot-infected muskmelon plants can cause extensive rot on muskmelon fruit. However, Toussoun and Snyder (1961) were unable to cause fruit rot symptoms on muskmelon or watermelon *[Citrullus lanatus* (Thunb.) Matsum. & Nakai] with a saprophytic isolate of *F. solani* obtained from a non-cucurbitaceous host. Additionally, they observed no lesions on mature ornamental gourd *(Cucurbita pepo v. ovifera* Alef.) or white bush scallop squash *(C. pepo* L.) fruit when they were set directly on top of saprophyte-infested soil. In contrast, lesions did develop when soil was infested with either race of *F.s.* f. sp. *cucurbitae.*

According to Toussoun and Snyder (1961), Godfrey reported that *F. solani* was a dominant factor in mature melon fruit rotting under wet weather in Texas. He concluded that the *F. solani* isolate used was more similar to race 2 of *F.s.* f. sp. *cucurbitae* in culture characteristics and was not pathogenic to plants. In contrast, the isolate was more effective than race 2 in rotting muskmelon fruits and apparently did not sexually cross with race 2. In the present study, the differences in fruit rot severity between *F. solani* isolates and *F. s.* f. sp. *cucurbitae* race 1, with the exception of 34B-1, were not statistically significant. In general, the test isolates caused more fruit rot than *F.s.* f. sp. *cucurbitae.* The *F. solani* isolates used in this study are aggressive fruit rotters and may be similar to those described by Godfrey (Toussoun and Snyder, 1961).

Fruit rot remains prevalent throughout the LRGV, and based on the present study, *F. solani* could be a dominant factor. Infected fruit are often tilled into the soil and may serve as a reservoir for growth and multiplication of *F. solani.* The high degree of association of *F. solani* with root lesions caused by *Monosporascus cannonballs* (Champaco, 1990; Mertely et al., 1991) may, in turn, provide the necessary inoculum for fruit infection.

Little variation was observed in disease response among the various lines screened (Table 1). All cultigens tested were highly susceptible to both strains of *F. s.* f. sp. *cucurbitae.* None of the plants in the water control treatments died. All four fusaritum wilt differentials, except Doublon, were equally susceptible (all plants died) to the Arkansas and California isolates. Only 50% of the Doublon plants died when inoculated with the California strain. Although statistically significant, we consider this an anomaly because Doublon has shown no resistance to *F.s.* f. sp. *cucurbitae* in other tests (data not shown). Based on the results of these tests, the two *F. s.* f. sp. *cucurbitae* strains had similar effects on their hosts, and no resistance was identified in any of the cultigens tested. These experiments also have shown that MR-1 is highly susceptible to race 1 of *F.s.* f. sp. *cucurbitae.* Although this information is disappointing, this breeding line is still valuable in that it is the only germplasm, other than PI-12411lF (Cohen and Eyal, 1987), with resistance to races 0, 1, and 2 of *F. o.* f. sp. *melonis (Zink* and Thomas, 1990). An additional advantage of MR-1 is its high level of resistance to powdery mildew and downy mildew (Thomas, 1986) and to *Alternaria cucumerina* (Ellis & Everh.) J.A. Elliot (Thomas et al., 1990).

The Arkansas strain of *F. s.* f. sp. *cucurbitae* used in this study was isolated from Texas gourd seeds and seedlings (Boyette et al., 1984). Texas gourd is a problem weed in cotton and soybean in Arkansas, and researchers there have shown *F. s.* f. sp. *cucurbitae* to bean effective biocontrol agent (Boyette et al., 1984; Weidemann and Templeton, 1988). These same studies also showed that the pathogen survived poorly in the soil and did not persist for more than 12 months. In contrast, Nash and Alexander (1965) reported that *F.s.* f. sp. *cucurbitae* survived at least 19 months in artificially infested soil, and Sumner (1976) found that it survived for as long as 20 months in naturally infested fields in Georgia.

Pathogenicity tests revealed a limited host range for *F.s.* f. sp. *cucurbitae* (Boyette et al., 1984). Pathogenicity tests performed at our laboratory (Champaco, 1990; R. D. M., unpublished), however, showed that, in addition to squash, numerous commercial muskmelon cultivars and 'Black Diamond' watermelon

Table 1. Comparison of disease reaction of muskmelon seedlings inoculated at 2 weeks old with twoisolatesof *Fusarium solani* f. sp. *cucurbitae* race 1.

	Dead plants (%)	
	Isolate	
Germplasm	California	Arkansas
Doublon	50 b[z]	100 a
1088CM17-187	83 a	100 a
Perlita	100 a	100 a
Topmark	100 a	100 a
Aragon	100 a	---
Challenger	88 ab	100 a
Easy Rider	100 a	84a
Explorer	100 a	---
Grande Gold	---	100 a
Hiline	100 a	100 a
HMX 5601	100 a	100 a
Hymark	100 a	100 a
Laguna	94a	---
Magnum 45	91 ab	100 a
Mission	100 a	92 ab
MR-1	100 a	---
Producer	100 a	100 a
PSX 1983	100 a	100 a
PSX 2083	100 a	100 a
Sunshine	---	100 a
Topflight	---	96a
XPH 5363	---	94a
XPH 5264	---	100 a

[z]Means followed by the same letters within columns are not statistically different based on Fisher's LSD test (α= 0.05).
[y]Not included in the study.

[determined resistant by Boyette et al. (1984)] were susceptible to *F.s.* f. sp. *cucurbitae*. The impact on the use of *F. s.* f. sp. *cucurbitae* as a biological control agent requires careful consideration. In spite of its appealing characteristics, which make this pathogen an attractive alternative to the use of chemical control, introducing this pathogen, especially to soils where it does not already exist, presents serious problems to cucurbit growers not only in Arkansas, but in neighboring states such as Texas, where muskmelon and watermelon are major production crops. Boyette et al. (1984) acknowledge that the pathogen is spread in the field, but, as yet, the mechanism of spread has not been fully determined. Based on previous work (Champaco, 1990), *F.s.* f. sp. *cucurbitae* has not been detected in Texas. However, the use of *F. s.* f. sp. *cucurbitae* as a biocontrol agent for weeds could spread the pathogen through infected seed or infested soil. The pathogen can be seed-borne to a high degree and lies dormant between the seedcoat and cotyledons (Toussoun and Snyder, 1961). The possibility remains that if *F.s.* f. sp. *cucurbitae is* used as a biocontrol for Texas gourd, it could spread to Texas in the near future.

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