Sudden Wilt of Melons in Southern Israel: Fungal Agents and Relationship with Plant Development

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ABSTRACT

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Fungi belonging to five genera, *Monosporascus* sp., *Pythium aphanidermatum*, *Rhizoctonia solani*, *Olpidium* sp., *Fusarium solani*, and *F. proliferatum*, were the species most frequently isolated from the root systems of wilted melon. Diseased plants were collected from 24 fields in the northern and central Arava region of southern Israel during the fall seasons of 1994 and 1995. In pathogenicity tests conducted under field conditions, in artificially inoculated microplots, the first wilt symptoms were observed at various stages of fruit maturation. High mortality levels (73 to 97%) were recorded for inoculation combinations in which *Monosporascus* sp. was involved. Inoculations with the other fungi listed resulted in lower incidences of wilt. The combination of *F. solani* and *P. aphanidermatum* resulted in higher mortality than that caused by each pathogen alone. *Monosporascus* sp. seems to be the primary pathogen, although other fungi could also induce wilt. The dry weight of plants grown in naturally infested soil ceased to accumulate 33 days after transplanting, in contrast to plants grown in methyl bromide-treated soil. At this stage, the first wilt symptoms were observed. Fruit load affected wilt incidence. At the end of the growing season, 98% mortality was recorded for plants having the normal fruit load (2.5 fruits per plant) compared with 75 and 12% for plants that had their fruits thinned to one or zero per plant, respectively.

Sudden wilt of melon (*Cucumis melo* L.), also known as melon collapse and vine decline, is a worldwide problem, mainly in arid and semiarid regions (13). This disease is a major problem in the arid Arava region of southern Israel. The Arava valley is a part of the Syrian-African rift valley. Due to high prevailing temperatures, this region became the main out-of-season melon growing area for the local market and for export. The disease causes a rapid wilt of plants bearing mature fruits and may result in a total loss of the crop. Soil disinfestation by fumigation with methyl bromide (MB) is the common treatment used in the Arava to control this disease (21). Growing melons as a monoculture

crop, without crop rotation and without MB fumigation between the growing seasons, may result in severe cases of sudden wilt. The causal agent of this phenomenon in Israel has not been identified in all cases. Several soilborne pathogens, such as *Fusarium solani* (Mart.) Sacc. f. sp. *cucurbita* W.C. Snyder & H.W. Hans, *F. equiseti* (Corda) Sacc., *Macrophomina phaseolina* (Tassi) Goid., and *Monosporascus eutypoides* (Petrak) von Arx have been isolated from such wilted plants in various regions in Israel (5,9,17-19).

In Israel, field trials and inoculation experiments conducted by Reuveni et al. (19) showed that *Monosporascus eutypoides* was a primary agent of melon collapse in the Jordan Valley, which is a hot and arid region. Similar phenomena have been reported from the United States, Japan, Tunisia, and Spain (6,11,12,16,22). *Monosporascus cannonballus* Pollack & Uecker was reported as the main pathogen in the southern part of the United States (12-15); while in the northeastern states, vine decline was associated with *Fusarium* wilt and cucumber mosaic virus (CMV) (25). *Acremonium* sp. was isolated from the roots of wilted melon from the Mediterranean

coastal areas of Spain and various regions in California, and was associated with collapse symptoms (1,6). *Monosporascus* sp. was also isolated from a relatively low percentage of wilted melons in the same areas of Spain but failed to induce wilt symptoms by artificial inoculation (6).

Observations in melon fields in Israel show that the severity of the disease may increase under certain environmental conditions. It is common in the Arava region that the melon crop may be totally destroyed in late summer (which is a short growing season when temperatures are high); whereas the following melon crop in the same field in the spring (when temperatures are lower) is unaffected or less severely affected by wilt. Significant differences in sudden wilt incidence between the spring and late summer growing seasons were observed during an evaluation of 15 melon genotypes tested in naturally infested soil and harvested in May, September, and November (2). Generally, the highest disease scores were obtained in the late summer growing season compared with spring and fall. This was interpreted as a genotype by environment interaction, which was found to be highly significant (2), or as a result of enhanced activity of thermotolerant microorganisms. Plant response to this disease seems to be influenced by fruit maturation.

The purposes of this research were: (i) to identify the potential agent(s) of melon sudden wilt in the Arava region of southern Israel; (ii) to estimate the agents' relative importance to disease severity, alone and in combinations; and (iii) to quantify the relationship of dry weight accumulation and fruit load to disease development. Brief reports with partial results have been published (17,18).

MATERIALS AND METHODS

Isolation of fungi from the roots of wilted plants. Wilted melon plants were collected from 24 melon fields (10 plants per field) in the northern and central Arava during the autumn growing seasons (planting in August-September, harvesting in November) of 1994 and 1995. All fields

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from which plants were collected had been fumigated with MB prior to the growing season. All parts of the root tissue were sampled for the determination of their infection with fungi. Roots of wilted plants were excavated with the aid of a shovel at the early stages of plant collapse. Roots of each plant were plated on five petri plates. Nine segments of the taproot were plated on three plates, and two additional plates were plated with six segments of secondary and tertiary roots. Brown to black root lesions were observed on roots later found to be infected with *Rhizoctonia*, and small and light brown lesions were observed on roots infected with *Pythium aphanidermatum*. No symptoms could be detected on roots infected with *Monosporascus*. Root tissues were surface-disinfested for 1 min in 1% NaOCl, rinsed twice in sterile water, and plated on potato dextrose agar (PDA, Difco, Detroit, MI) medium amended with chloramphenicol (Sigma, C-0378) at 250 mg/liter. For further verification of identity of certain fungi, additional media were used in certain cases: water agar (2%), peptone-PCNB *Fusarium* selective medium (3), and asparagine glucose selective medium for *Pythium* (20). Root colonization by *Olpidium* sp. was detected with the aid of a light microscope. Roots and rootlets were thoroughly washed under running tap water, cleared with 10% KOH, acidified in 1% HCl, and stained with boiling 0.05% trypan blue (8).

Preparation of inoculum and pathogenicity tests. Plastic cylinders with open ends, 30 cm diameter and 35 cm high, were filled with field soil and buried flush with the soil level. Plot beds were covered manually with plastic sheets (Polyon, Kibbutz Barkai, Israel), and the edges were buried in trenches to a depth of 20 cm. MB (Dead Sea Bromine Group, Be'er Sheva, Israel) was applied at a rate of 50 g/m² by the hot gas method (7) through the drip irrigation pipes that had been placed on the beds before tarping. After fumigation, the soil content of each cylinder (microplot) was mixed with inoculum of fungi isolated from wilted melon plants prepared as follows: Isolates of *Monosporascus* sp. were grown for 10 days on PDA at 27°C. The content of one petri dish was blended with 100 ml of sterile water and mixed with 10 liters of MB-treated soil. In addition, 5 g of a mixture of soil and inoculated oats, prepared according to Mertely et al. (15), was added to the same soil. *F. solani* and *F. proliferatum* (six and three isolates, respectively) were grown for 1 week on PDA at 27°C. Conidial suspension was prepared by flooding the plates with 10 ml of distilled water and scraping the medium surface with a glass. The inoculum consisted of 100 ml of a mixture of conidial suspensions of the isolates of each species, adjusted to 10⁶ CFU/ml (mainly conidia), and mixed with 10 liters of MB-treated soil.

P. aphanidermatum was grown on PDA

at room temperature for 3 days. Four 1 × 1 cm PDA squares from a culture were cut out and placed in an empty petri dish. Leaves of St. Augustine grass, previously boiled for 30 min in water, were placed on the PDA squares and incubated at room temperature for 2 days, after which 30 ml of sterile water was added to the dish (20). The contents of several such dishes containing zoospores and hyphae with zoospores were combined afterward and incubated for an additional 2 days at room temperature in an Erlenmeyer flask. One week after planting, 100 ml of fungal suspension at a concentration of 5 × 10⁴ CFU (mainly zoospores) was drenched at the base of the transplant stem in each cylinder. This was sufficient to moisten the root system of the transplant.

Melon roots infected with *Olpidium* sp. were thoroughly washed under running tap water and then incubated for 30 min in sterile water (E. Hadar, *personal communication*) into which zoospores were released. The inoculum source was tested for possible presence of other pathogens and was found to be free of *Pythium* and melon necrotic spot virus (MNSV). Each plant was inoculated prior to transplanting by dripping 4 ml of the zoosporic suspension (2 to 4 × 10⁶ spores per ml) onto its roots. Inoculations combining two species of fungi were done by mixing the two inocula with the same 10 liters of soil, using for each species its particular form of inoculation. Infested soil samples from two fields with a history of disease (at Ein Yahav and Ein Tamar) were used as naturally infested controls. MB-fumigated soil was used as an uninoculated control.

Melon plants were transplanted, three per cylinder, each treatment consisting of 10 cylinders. The field around the cylinders was planted with melons at a stand of 12,000 double plants per hectare. Irrigation and fertilization were according to the recommendations for the Arava region.

Two pathogenicity tests were conducted, the first in the late summer of 1995, transplanted on 16 July and harvested in mid-September. Average temperatures at planting were 25 and 40°C night and day, respectively, and 21 and 37°C at harvest. The second test was transplanted on 13 February 1996 and harvested at the beginning of May 1996. Average night and day temperatures at planting were 11 and 21°C, respectively, and at harvest were 25 and 36°C. In both tests, plant roots were sampled 3 weeks after the inoculation and plated on PDA in order to determine the efficacy of the inoculation.

Dry weight accumulation in foliage and fruit. In this trial, melon plants (cv. Arava) were grown in naturally infested soil and in MB-treated soil (control) at the Ein Tamar Experiment Station. Plants were transplanted on 15 September 1995. The average daily temperature during September, October, and November 1995 were 25

to 38°C, 20 to 33°C, and 15 to 28°C, respectively. Plants were uprooted for weighing at the following development stages: male flowering, female flowering, 500 g of fruit, beginning of fruit netting, color breaking, and early maturation, corresponding to 14, 22, 33, 41, 50, and 55 days after transplanting, respectively. Seven plants per treatment were sampled at each stage; the foliage was separated from the fruits, dried at 70°C for 48 h, and weighed. Fruits were weighed twice, 33 and 50 days after transplanting. In addition, roots of wilted plants were plated on PDA for the identification of the pathogen, the experimental plots were evaluated for wilt incidence, and the dates of collapse were recorded for each treatment.

Effect of fruit load on disease severity.

The effect of fruit load on disease severity was tested in a trial conducted in a field with a history of sudden wilt at Ein Tamar Experiment Station. Fruits were removed 30 days after transplanting, when the first wilt symptoms were observed. New fruits were removed once a week until the completion of the experiment. Disease development (number of wilted plants), was evaluated 46, 53, and 61 days after transplanting, and root samples from wilted plants were plated on PDA for identification of the pathogen. Comparison was made among plants with three levels of fruit load: zero fruits, one fruit, and untreated plants with an average of 2.5 fruits per plant. Each treatment was applied to 50 plants, i.e., 10 plants grown on a 5-m row with five replicates.

Statistical analysis. The significance of differences ($P = 0.05$) in the pathogenicity tests and in the fruit load trial were determined by the Einot-Gabriel range test (4). Disease incidence data were arcsine-transformed before analysis. Selected treatments were thereafter compared by contrast t test. Values of foliage dry weight accumulation are accompanied by SE values.

RESULTS

Fungi isolated from wilted melon roots. Fungi belonging to five genera were

Table 1. Fungi isolated from melon plants showing collapse during the fall seasons of 1994 and 1995^z

Fungi	Northern Arava	Central Arava
<i>Monosporascus</i> sp.	35	28
<i>Pythium</i>		
<i>aphanidermatum</i>	94	71
<i>Rhizoctonia solani</i>	41	28
<i>Olpidium</i> sp.	18	86
<i>Fusarium</i> (<i>solani</i> and <i>proliferatum</i>)	100	86

^z Values are proportion (%) of the fields from which indicated fungi were isolated. Wilted plants (10 from each field) were collected from 17 fields in the northern Arava and 7 fields in the central Arava.

those most frequently isolated from the root systems of wilted melon plants collected from 24 fields in the northern and central Arava during the fall seasons of 1994 and 1995 (Table 1). All fields had been fumigated with MB in preparation for the growing season. Therefore, the crop in each field developed normally and wilt incidence was usually lower than 5%. The five most frequent genera of fungi were present in both regions, albeit in different proportions. *Pythium aphanidermatum* and *Fusarium* (mostly *solani* and *proliferatum*) were very common (in 71 to 100% of fields) in both regions. *Olpidium* sp. was substantially more common in the central Arava (86%) than in the northern Arava (18%). *Monosporascus* sp. accounted for only 28 and 35% of the isolations in the northern and central Arava, respectively (Table 1). *Monosporascus* was the main pathogen isolated from two fields at the Experiment Stations of Ein Tamar (northern Arava) and Ein Yahav (central Arava), in which melons had been grown without MB fumigation for several seasons and wilt incidence was 73 to 90% (Tables 2 and 3). Fungi such as *Acremonium* spp., *Alternaria* spp., *Aspergillus* spp., *Chaetomium* spp., *Myrothecium roridum*, *Nigrospora* sp., *Trichoderma* spp., *Fusarium oxysporum*, *F. equiseti*, and *Helmintho-*

sporium spp. were also isolated, but they were substantially less frequent than the species listed in Table 1, and their pathogenicity to melon plants was not tested.

Pathogenicity tests. In the fall of 1995, plants wilted and collapsed in the inoculated microplots starting in mid-September, when fruits were at various stages of maturity. Mortality percentages were recorded 70 days after planting. When plants were inoculated with a single species of pathogen, *Monosporascus* sp. was the most virulent, producing 73% mortality, significantly higher than *P. aphanidermatum*, *F. solani*, *Olpidium* sp., and *F. proliferatum* (Table 2). The ripening of fruits on plants inoculated with *Olpidium* sp. was somewhat delayed compared with those in the other treatments, while wilt was comparable with that in the uninoculated control.

Several combinations of inoculations resulted in high disease incidence (Table 2). The highest mortality levels were recorded for inoculation combinations involving *Monosporascus* sp. (80 to 97%), but they did not significantly differ from inoculation with *Monosporascus* alone (73%). Disease incidence in naturally infested soils from which *Monosporascus* was the pathogen most frequently isolated was also high (73 to 90%). However, the contrast

test comparing *Monosporascus* versus combinations with other fungi showed significantly greater wilt incidence for combinations ($P = 0.013$). This was also the case for the combination of *F. solani* + *P. aphanidermatum* ($P = 0.0001$).

The results obtained in the first trial were essentially confirmed in the second pathogenicity test, conducted in the spring of 1996 (Table 3). *Monosporascus*-inoculated plants and plants grown in naturally infested soil exhibited the highest wilt incidence, ranging from 80 to 90%, similar to the incidence (73 to 90%) in naturally infested soil. *P. aphanidermatum* and the combination of *Pythium* with *F. solani* resulted in 54 and 72% mortality, respectively. *Pythium* was the only fungus that produced different values for sudden wilt in the second test (54%), compared with 23% at the first test. Although *R. solani* was isolated in relatively high percentages from wilted melon plants (Table 1), it failed to induce wilt symptoms in preliminary experiments; thus it was not included in the pathogenicity tests.

Dry weight accumulation in foliage and fruit. The foliage dry weight of plants grown both in MB-fumigated soil and in naturally infested soils accumulated up to 33 days after planting but was 35% greater in plants grown in fumigated soil. This stage was a turning point at which dry weight accumulation ceased for plants in infested soils but continued at a slower rate for plants in fumigated soil (Fig. 1).

The average fruit dry weight of plants grown in MB-treated soil increased from 64 g on the thirty-third day to 184 g 50 days after planting, compared with 51 g and 107 g, respectively, for plants grown in infested soils. The first wilt symptoms were observed in the latter plants 32 to 35 days after planting.

Effect of fruit load on disease severity. The first symptoms of sudden wilt in this experiment were usually associated with fruit maturity. Mortality of 98% of the untreated plants grown in infested soil, with an average of 2.5 fruits per plant, was observed 61 days after planting. Plants grown in infested soil, but with fruit thinned to one or zero per plant, exhibited wilt incidences of 75 and 12%, respectively (Fig. 2). *Monosporascus* sp. was the fungus most frequently isolated from the roots of wilted plants in this experiment.

DISCUSSION

Monosporascus eutypoides has been reported (5,9,19) to be associated with collapse of melons in the Jordan Valley and the Arava of southern Israel. Both regions are arid and characterized by hot climates. *Monosporascus* sp. was the most frequently isolated from nonfumigated naturally infested soil. However, in MB-fumigated soils, it was less frequent than the other fungi, such as *P. aphanidermatum* and *Fusarium* spp. This apparently

Table 2. Response of melon plants to inoculation with fungi isolated from wilted plants or growth in infested soil from two locations^y

Inoculation source	Wilt incidence (%)
<i>Monosporascus</i> sp. + <i>Fusarium proliferatum</i>	97 a ^z
<i>Monosporascus</i> sp. + <i>Fusarium solani</i>	97 a
Naturally infested soil - EinYahav	90 a
<i>Monosporascus</i> sp. + <i>Pythium aphanidermatum</i>	80 a
<i>Monosporascus</i> sp. + <i>Olpidium</i> sp.	80 a
<i>Monosporascus</i> sp.	73 ab
Naturally infested soil - Ein Tamar	73 ab
<i>Fusarium solani</i> + <i>Pythium aphanidermatum</i>	57 abc
<i>Pythium aphanidermatum</i> + <i>Olpidium</i> sp.	30 abcd
<i>Pythium aphanidermatum</i>	23 cd
<i>Fusarium solani</i> + <i>Fusarium proliferatum</i>	17 d
<i>Fusarium solani</i>	17 d
<i>Olpidium</i> sp.	3 d
<i>Fusarium proliferatum</i>	0 d
<i>Fusarium solani</i> + <i>Olpidium</i> sp.	0 d
Methyl bromide-treated soil, noninoculated	0 d

^y The trial was conducted under field conditions in fall 1995. Planting on 16 July 1995. Fruit maturity at mid-September 1995. Disease incidence was recorded 70 days after planting.

^z Values with the same letter are not significantly different ($P = 0.05$).

Table 3. Response of melon plants to inoculation with fungi isolated from wilted plants or growth in infested soil from two locations^y

Inoculation source	Wilt incidence (%)
Naturally infested soil - Ein Tamar	90 a ^z
<i>Monosporascus</i> sp.	88 a
Naturally infested soil - Ein Yahav	80 a
<i>Pythium aphanidermatum</i> + <i>Fusarium solani</i>	72 ab
<i>Pythium aphanidermatum</i>	54 ab
<i>Fusarium solani</i>	16 b
Methyl bromide-treated soil, noninoculated	12 b

^y The trial was conducted under field conditions in spring 1996. Planting on 13 February 1996. Fruit maturity at beginning of May 1996. Disease incidence was recorded 94 days after planting.

^z Values with the same letter are not significantly different ($P = 0.05$).

reflects microbial shift induced by MB fumigation.

Pathogenicity tests were conducted in order to establish the relative importance of the various fungi isolated from wilted plants to the sudden wilt syndrome of melons in the Arava. Using artificial inoculations, *Monosporascus* sp. was found to be the single most virulent species involved in the sudden wilt of melons in the Arava region. *P. aphanidermatum* and *F. solani*, although more frequently isolated, were

shown to be less important as single pathogens under Arava conditions. Their importance seems to be in their ability to co-colonize the roots of melon plants and to induce wilt to a greater extent than their individual effects, thus contributing to the disease complex.

F. proliferatum was not pathogenic by itself or in combination with *F. solani*. *F. proliferatum* is widespread in Israel, where it sometimes causes root rot and stem base rot in various crops (I. S. Ben Ze'ev and E.

Levy, unpublished). In the second test, conducted in the spring of 1996 (Table 3), *P. aphanidermatum* was capable of inducing wilt symptoms in 54% of the inoculated plants, compared with 23% in the fall experiment. These differences between the two experiments might be attributed to the environmental conditions and the length of the growing period.

A variety of organisms has been reported as causal agents of melon collapse in various regions (5,6,9,12,14). It is possible that all of these organisms act similarly by suppressing the root system, finally affecting the balance between sink and source, especially during the period of fruit maturation. Abiotic stresses, such as high temperature, may further aggravate the stress situation.

Foliage dry weight of melon plants ceased to accumulate when the fruits increased in weight. This is in agreement with the reports that reproductive activity, i.e., fruit development, curtails the vegetative growth of plants including cucurbits (10,24). The high demand for water and assimilates by the developing fruit, in addition to the need to continue foliage and root maintenance, can endanger plants with roots damaged by pathogenic organisms. This turning point was observed in the present study around the thirty-third day after planting, when fruit weight was approximately 500 g (Fig. 1), in plants grown in MB-treated soil, but was more pronounced in plants grown in infested soil. At this stage, a reversible loss of turgor was observed first around midday, and wilt symptoms were detected a few days later. The contribution of fruit maturation to enhanced symptom development was further tested by removing the fruits prior to ripening. Fruit removal may reduce the water stress and, consequently, plant wilting (Fig. 2). Similar results, showing delayed symptoms following fruit removal, were reported in trials conducted in Texas (23).

The presented data show that *Monosporascus* can be found in plants grown in MB-treated soils in the first growing season after fumigation. This fungus became the primary pathogen in the two experimental field plots after a few seasons of melon growing without MB application. It seems that *Monosporascus* has an advantage in infesting the soils used for melon monoculture in this particular area.

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Fig. 1. Accumulation of dry weight in the foliage of melon plants grown in methyl bromide-treated soil or in infested soil. Bars indicate SE values. Arrow indicates the time at which the first wilt symptoms were observed.

Fig. 2. Effect of fruit removal on wilt incidence. Bars (for the same date) with a common letter are not significantly different ($P = 0.05$).

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