

Cavity Rot of Winter Melon Caused by *Verticillium dahliae*

W. D. GUBLER, Department of Plant Pathology, University of California, Davis 95616, and E. A. BERNHARDT, Petoseed Research Center, Route 4, Box 1255, Woodland, CA 95695

ABSTRACT

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In each of 3 yr, fruit of winter melon (*Benincasa hispida*) held in nonrefrigerated storage were decayed internally. The fruit appeared sound, but the flesh contained pockets of rot and profuse mycelium. *Verticillium dahliae* was isolated in pure culture both from the rotted fruit tissue and from hyphal tips of the mycelium. The isolates caused a wilt of winter melon seedlings and were characterized by host range tests as the race 1 tomato strain. Symptoms of fruit rot were reproduced by injecting a conidial suspension into the fruit flesh. Although winter melon plants showed no symptoms of Verticillium wilt at harvest, *V. dahliae* was shown by isolation to be present in the roots, stems, and peduncles. This is the first report of *V. dahliae* causing a fruit rot.

The fruit of winter melon (*Benincasa hispida* (Thunb.) Cogn.) are valued for their use in Chinese cooking. The fruit are harvested in autumn and held in storage for several months before being sold. Fruit reaching market in late winter are sold at a premium price.

In January 1983, February 1985, and February 1989, fruit shipped to restaurants arrived with internal decay. In 1989, further examination of fruit at the warehouse of origin in Woodland, California, showed a high incidence of fruit with internal decay. The fruit appeared sound but when cut open showed pockets of dry, brown rot and masses of white mycelium in the placenta and flesh (Fig. 1). This paper reports on the previously undescribed fruit rot caused by *Verticillium dahliae* Kleb.

MATERIALS AND METHODS

Isolations. Fruit were surface-disinfested for 2 min with 0.5% NaOCl. Mycelium from inside the fruit was placed on V8 juice agar (V8A), water agar (WA), and acidified potato-dextrose agar (APDA). Fruit tissue was excised from the margins of brown, rotted areas and placed on WA or V8A. In addition, isolations for bacteria were made by streaking decayed pieces of fruit across the surface of yeast-dextrose agar and nutrient agar. Cultures were incubated at room temperature (22–24 C).

Present address of second author: Plant Science Consulting & Research, 1027 Davis Street, Vacaville, CA 95687.

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To establish the presence of *V. dahliae* in the winter melon plants, isolations were made from asymptomatic plants randomly collected from a field where disease had occurred previously. Roots, stems, and peduncles were surface-disinfested for 1–3 min in 0.5% NaOCl. Tissue sections containing vascular elements were excised and placed on V8A and WA.

Pathogenicity tests. Three-week-old cultures of *V. dahliae* grown on V8A were ground in a blender with enough tap water to produce a fine slurry containing microsclerotia, conidia, and mycelia. The roots of winter melon seedlings (expanded cotyledon stage) were clipped to 1–2 cm and dipped into the agar slurry for 1 min. Inoculated seedlings were transplanted into pasteurized soil in flats and maintained in a greenhouse at 22–24 C. For some pathogenicity tests, a suspension of 10^6 conidia per milliliter of *V. dahliae* was used in place of the agar slurry. The suspension was made by flooding 2-wk-old cultures grown on APDA with tap water and filtering the resulting suspensions through eight layers of cheesecloth to remove mycelial fragments. Plants were inoculated, then treated as described above.

Seedlings of tomato cvs. Bonnie Best and VFN8, pepper cv. Yolo Wonder B, watermelon cv. Sugar Baby, cantaloupe cv. Top Mark, and cucumber cv. SMR 18 were inoculated by dipping their clipped roots into the agar slurry inoculum. All plants were placed in a greenhouse maintained at 22–24 C, watered as needed, and fertilized once a week.

Mature disease-free fruit, obtained from a source with no history of cavity rot, were surface-sterilized by swabbing liberally with 0.5% NaOCl. A 23-gauge needle was used to inject six fruit with 2 ml of sterile distilled water (controls)

and six fruit with 2 ml of a suspension of 10^6 conidia per milliliter of *V. dahliae* to a depth of 1.5 cm. Injection sites were surface-sterilized as previously described, sealed with petroleum jelly, and marked. Inoculated and control fruit were placed in a 2.5 × 1.8 × 1.8 m metal storage shed for 3 mo to duplicate common conditions of storage.

RESULTS AND DISCUSSION

All isolations made from decayed fruit yielded only pure colonies of *V. dahliae*; no bacteria were detected. In addition, large numbers of microsclerotia appeared in the flesh of fruit that was cut and incubated on a laboratory bench. *V. dahliae* was found in the roots, stems, and peduncles of plants collected from the field before and after harvest, although the plants lacked obvious symptoms.

The *V. dahliae* isolates recovered from fruit and plants of winter melon caused wilt in seedlings of winter melon, Top Mark cantaloupe, Sugar Baby watermelon, and Bonnie Best tomato but not in VFN8 tomato, SMR 18 cucumber, or Yolo Wonder B pepper, indicating that the isolates are equivalent in pathogenicity to the race 1 tomato strain (2). This strain of *V. dahliae* is prevalent in soils in the Woodland, California, area, and tomatoes cannot be grown commercially in the area unless they are resistant to this strain.

After 3 mo of storage, typical disease symptoms and signs of the pathogen were observed in the flesh of winter melon. Rot of the internal flesh typically extended laterally 6–8 cm from the point of injection and approximately 1 cm outward toward the rind. Rotted tissue was

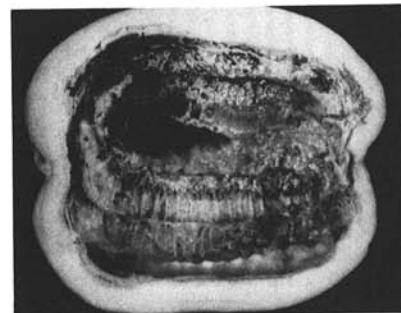


Fig. 1. Cavity rot of winter melon caused by *Verticillium dahliae*, showing white mycelial growth and black mass of microsclerotia.

overgrown with white mycelium, and microsclerotia were present. Externally, fruit appeared sound. The pathogen was recovered in pure culture from all inoculated fruit but not from water-injected control fruit, although flesh in the vicinity of the injection was slightly discolored.

A unique set of conditions appears to lead to cavity rot of winter melon. Symptomless infection allows plants colonized by *V. dahliae* to reach maturity and produce marketable fruit. Also, during the long period of nonrefrigerated storage (October–March) the pathogen appar-

ently is able to grow from the vascular system of the fruit and colonize the adjacent flesh.

Verticillium wilt was first reported on muskmelon in California in 1934 (1) and occurs to some extent in all cucurbit species grown on soil infested with *V. dahliae*. It is possible that other cucurbit species produce a marketable crop even though infected with *V. dahliae*. The reason that *V. dahliae* fruit rot has not been observed in other cucurbits may be because the fruit are refrigerated after harvest or are stored for short periods. Alternatively, the flesh of other cucurbit fruit

may not be susceptible to decay by *V. dahliae*.

We believe this to be the first report of *V. dahliae*, which colonizes the vascular system of many plants, causing a fruit rot. Because the rot is not apparent from the outside of the fruit, we propose that this disease be called Verticillium cavity rot.

LITERATURE CITED

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