Severe Spotting of Fresh Market Tomato Fruit Incited by *Corynespora cassiicola* After Storm-related Injury

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ABSTRACT

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During the 1982–1983 winter growing season, a severe spotting of mature green tomato (*Lycopersicon esculentum*) fruit occurred in South Florida. Symptoms were associated with high winds and heavy rains accompanying a storm. Lesions on wounded fruit surfaces appeared as small necrotic pits or freckles. Although other fungi (*Alternaria alternata, A. solani*, and *Stemphylium* spp.) were recovered occasionally, *Corynespora cassiicola* was isolated most frequently and consistently. Pathogen morphology and conidial measurements for isolates recovered from lesions were consistent with those reported for *C. cassiicola*. Fruit infection on the cultivar Duke occurred through wounds on the epidermis but not through unwounded tissue.

Of the many disease problems facing producers of tomatoes (*Lycopersicon esculentum* Mill.) in Florida, those that result in damage to fruit have the greatest economic impact. Target spot, caused by *Corynespora cassiicola* (Berk. & Curt.) Wei., has been reported to cause premature defoliation of tomato (1,2). Blazquez (2) showed that isolates from blighted foliage could produce damage to greenhouse-grown tomato fruit. Jones and Jones (5) reported fruit losses but did not describe symptoms.

During January 1983, unusual symptoms occurred on field-grown, mature green tomato fruit subsequent to an unseasonably severe storm. High winds associated with the storm suggested the possibility of widespread wounding of fruit from windblown soil particles. Symptoms noted by private pest management consultants and scouts (6) consisted of small necrotic pits or freckles on the fruit surface. Losses were estimated to be up to 25% in some fields. The etiology of the problem was unknown. Fruit with these symptoms were unmarketable because of appearance and subsequent postharvest decay. The objectives of this study were to isolate and identify the causal agent of the fruit spotting and to reproduce the symptoms on healthy fruit.

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MATERIALS AND METHODS

Pathogen isolation and identification. Symptomatic, mature green tomato fruit of the cultivar Duke were obtained from several fields in Dade County, Florida, during January and February 1983. Tissue sections measuring about 2 mm^2 were excised from fruit lesions and surface-disinfested for 20 sec in 0.5% aqueous sodium hypochlorite. After a rinse in sterile deionized water (SDW), sections were blotted dry and plated on Difco potato-dextrose agar (PDA) amended with 1 ml/L of lactic acid. Plates were incubated in darkness for 7 days at 27 C. Sporulation was enhanced by exposure to a 12-hr photoperiod under fluorescent lights (approximately 400 $\mu E \cdot m^{-2} \cdot s^{-1}$) at 23 C for an additional 7 days.

Two isolates from fruit (CC1 and CC9) were chosen for detailed morphological studies. Single conidia were transferred to PDA slants and subsequently maintained at 4 C. The isolates were later transferred to plates of PDA media and grown at 23 C on a laboratory bench for 12 days before conidial measurements were assessed. Approximately 35 conidia in each of three microscope fields at ×400 were measured for each isolate.

Pathogenicity. Three isolates of *C. cassiicola*—CC1, CC9, and CC10—were chosen for pathogenicity studies. Unblemished mature green fruit of the cultivar Duke were harvested from staked plants in a roadside retail field in Homestead, Florida. All test fruit were surface-disinfested for 2 min in 0.5% aqueous sodium hypochlorite and rinsed in SDW.

A sterile microscope slide was used to scrape conidia from 7-day-old PDA cultures. Inoculum of each isolate was standardized to 10^5 conidia per milliliter with SDW and a hemacytometer and applied to fruit with a 250-ml hand-held mist sprayer. Fruit wounded just before inoculation were compared with non-wounded fruit. Very fine wounds were made in some fruit by gently rolling them over No. 135 fine-grade sandpaper (one 360° rotation for each fruit). Other fruit were wounded more severely by puncture with a sharp No. 3 sewing needle that had been pushed through a cork stopper until only 1 mm of the needle tip was exposed. Five fruit were used in each of the three treatments (no wounding, sandpaper wounding, and needle wounding) for each isolate. Five control fruit for each treatment were wounded or left unwounded and sprayed with SDW.

Immediately after inoculation, all fruit were placed on surface-disinfested trays in a dew chamber at 26 C and subjected to high humidity for 18 hr. Fruit were then moved to the laboratory bench (room temperature approximately 25 C) and observed for symptom development over the next 17 days. This experiment was performed twice.

RESULTS

Pathogen isolation and identification. *C. cassiicola* was recovered from 28 of 48 plated fruit lesions. No other organism was isolated consistently. *Alternaria solani* Sorauer, *Stemphylium* spp., and *A. alternata* (Fr.) Keissler were recovered from one, two, and three tissue samples, respectively.

Colonies of the CC1 and CC9 isolates were gray when grown on PDA and produced rapidly spreading mycelium with abundant conidiophores and conidia. Conidia were obclavate-pyriform, thick-walled, pseudoseptate, and hyaline to light brown. Conidial

measurements were within the range of those given for *C. cassiicola* (4,8). The dimensions of 100 conidia of isolate CC1 produced after 12 days on PDA were 22.0–105.0 × 2.8–11.0 μm and averaged 47.5 × 8.0 μm. Likewise, 100 conidia of isolate CC9 produced on PDA were 14.0–124.0 × 3.0–11.0 μm and averaged 42.6 × 6.7 μm. Conidia of both isolates had 2–12 pseudosepta.

Pathogenicity. Symptoms began to appear on all wound-inoculated fruit within 48 hr. Inoculated fruit wounded with sandpaper had symptoms most typical of those seen in the field. Lesions appeared as small, light-brown freckles with darker margins (Fig. 1A). The center of the lesion was slightly sunken and somewhat dry. Lesions on needle-punctured fruit were larger and uniformly dark brown. Over the next 2 wk, fruit lesions enlarged and coalesced, resulting in large areas of sunken, necrotic tissue (Fig 1B). Damage extended about 5 mm into the fruit pulp. Some superficial fungus growth was noted in the centers of larger lesions by the end of the experiment. At 17 days, discrete lesions of sandpaper-wounded inoculated fruit averaged 2.1 mm in diameter; 30 lesions for each isolate were measured to obtain the average.

No symptoms were observed on fruit not wounded before inoculation. Control fruit wounded by sandpaper showed only small, whitish flecks, and fruit wounded by needle showed only shallow depressions. *C. cassiicola* was recovered from 80% of a subsample of test fruit lesions. No differences in virulence were noted among isolates CC1, CC9, and CC10.

DISCUSSION

We demonstrated that *C. cassiicola* was the causal agent of a severe spotting of fresh market mature green tomato fruit in Florida. This pathogen has previously been reported to incite disease on tomato foliage and stems (1,2,5). We were also able to recover *C. cassiicola* from naturally occurring stem lesions (*data not shown*).

Symptoms were produced and the pathogen reisolated from fruit predisposed by wounding. Of the cultivars tested by Blazquez, only the fruit of Walter were successfully infected with *C. cassiicola* without wound predisposition (2). In our study with the cultivar Duke, fruit had to be wounded to become infected. Wounding has been mentioned by others (3,7) to be important in the etiology of the same disease in other crops. Fruit in Florida fields is commonly wounded by wind- and rain-driven soil particles. The epidemic reported in this paper was associated with an unusually severe winter rainstorm. Problems with this disease could occur whenever conditions favor wounding of fruit.

The prevalence of target spot in Florida may be due to the heavy dependence of growers on tank-mix sprays of copper + mancozeb fungicides applied for optimum control of bacterial spot incited by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye (6). These same products give little or no control against target spot. Possible incompatibilities of certain copper + mancozeb combinations have been suggested as one reason for poor control of *C. cassiicola* (5).

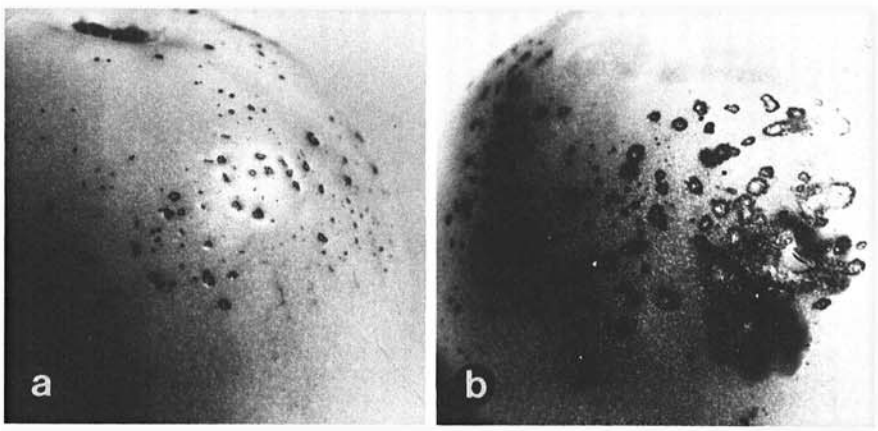

Fig. 1. Mature green tomato fruit with symptoms of target spot incited by *Corynespora cassiicola*: **(A)** 48-hr lesions on fruit predisposed by sandpaper wounding before inoculation and **(B)** larger, 2-wk-old coalescing lesions on wounded and inoculated fruit.

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