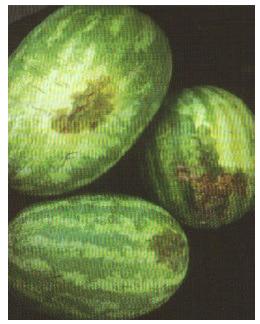
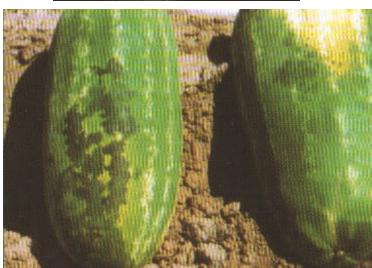
Bacterial Fruit Blotch of Watermelon









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INTRODUCTION

In the spring of 1989, a new bacterial fruit rot of watermelon occurred in commercial watermelon fields in Florida and, as the season progressed, the disease was observed in South Carolina, North Carolina, Maryland, Delaware, and Indiana. In some fields, losses were more than 90% of the total marketable fruit. Since then, the disease also has been found in Alabama, Arkansas, California, Georgia, Iowa, Mississippi, Missouri, Oklahoma, and Texas.

Bacterial fruit blotch has occurred in one or more states in the eastern U.S. every year since 1989. Fortunately, in most of these years, the disease occurred in relatively few fields, but was devastating in some of these. Fruit blotch was wide-spread in Georgia in 1992. The disease was most damaging in 1994, causing losses in thousands of acres distributed over at least 10 states.

Bacterial fruit blotch has occurred in cantaloupes and honey dew melons

CAUSEANDSYMPTOMS

The bacterium that causes fruit blotch of watermelon is **Acidovorax avenae** *subsp.* **citrulli** *(formerly)* **Pseudomonas pseudoalcaligenes** *subsp.* **citrulli).** The fruit blotch bacterium can cause a seedling blight, leaf lesions, and fruit symptoms.

First symptoms in watermelon seedlings appear as a dark, water-soaking on the lower surface of cotyledons and leaves (Fig. 1), followed by necrotic lesions, which may have chlorotic halos (Fig. 2). In young seedlings, lesions can occur in the hypocotyl, resulting in collapse and death of the plant. Foliar symptoms can develop throughout the growing season and, in many cases, they are not numerous nor very distinctive and may be easily confused with other disorders. Leaf lesions are light brown to

reddish-brown in color and often spread along the midrib of the leaf (Fig. 3). Leaf lesions in the field do not result in defoliation, but are important reservoirs of bacteria for fruit infection.

Symptoms on the surface of fruit begin as small, greasy appearing, water-soaked areas a few millimeters in diameter (Fig. 4). These enlarge rapidly to become dark, green, watersoaked lesions several centimeters in diameter with irregular margins. Within a few days, these lesions may rapidly expand to cover the entire upper surface of the fruit, leaving only the ground spot symptomless (Fig.5). In fruit of watermelon cultivars with dark green striped rinds, the water-soaking may be more restricted, resulting in smaller lesions on the lighter green stripe. Under certain environmental conditions and with infection of very young fruit, symptoms may not include the typical water-soaking (Fig 6 & 7). Initially, the lesions do not extend into the flesh of the melon (Fig 8). With age, the center of the lesions may turn brown and crack, and a total fruit rot may develop(Fig 9). A white bacterial ooze, or an effervescent exudate, follows fruit decay.

DISEASE DEVELOPMENT

The fruit blotch bacterium may be introduced into a field by infested seeds, infected transplants, contaminated volunteer water-melons, or infected wild cucurbits. The bacterium can be a contaminant of seeds that are harvested from diseased fruit. Diseased transplants can cause severe problems because the bacterium may spread in the transplant house and result in high numbers of infected plants going into the field. Some of these transplants may harbor the bacterium, but show no symptoms. The bacterium can be recovered from volunteer watermelon seedlings in fields that had fruit blotch the previous season. Wild citron, a widespread weed in the southern U.S., is very susceptible to both foliage and fruit infection. Seed transmission of the bacterium has been demonstrated in citron. Citron and other wild cucurbits are potential over seasoning hosts of the fruit blotch bacterium. The pathogen also may survive from one season to another on infested watermelon rind.

Bacterial fruit blotch appears to be favored by high humidity and high temperature. Symptom development and spread on foliage and fruit is most rapid during periods when the weather is hot, humid, and sunny with afternoon thundershowers. In Florida, this is usually May-June with the spring crop, and in the more northerly states such as Georgia, South Carolina, and Indiana, it is June-July. Optimum conditions for disease development also may occur early with the fall crop in Florida (August-September). Symptoms do not appear to develop as severely during cool, rainy weather. With favorable weather, the bacterium can spread rapidly. Once the bacterium is present in the field, it can be spread by wind-driven rain and mechanical means. A few primary infection sites in a field can result in infection of all plants by the time of harvest. Under some conditions, foliar symptoms may not be very conspicuous and the grower may not realize that there is a problem until fruit symptoms render the crop unmarketable. Therefore, close inspection of plants is essential for early detection of the disease.

Bacteria produced in leaf lesions can spread and infect developing fruit. Two to three week-old fruit are most susceptible to bacterial invasion. The fruit blotch bacterium enters fruit through small openings (stomata) on the fruit surface and a small, water-soaked lesion develops 3-7 days later. Mature fruit are covered with a wax layer that plugs the stomata and prevents entry of the bacteria into the fruit. Once the wax layer forms, mature watermelons can be invaded by the fruit blotch bacterium only after wounding.

CONTROL

The ideal control is to prevent the introduction of the bacterium into the field. Plant seeds that have been tested and found to be free of the fruit blotch bacterium. A negative assay is not proof that the seedlot is free of the bacterium, but does indicate that bacterium was not detected in a representative sample of the seedlot. Use transplants from transplant houses in which there were no seedling symptoms of the fruit blotch disease. Transplant growers should carefully inspect their seedlings, destroy any flats containing seedlings with suspicious symptoms, and separate flats immediately adjacent to those with symptoms from healthy plants. Cultural practices in the transplant house should include minimal manipulation of plants; decontamination of hands, plant containers, and tools after contact with plants; closing sides of the transplant house during storms or windy periods; and destroying discarded plant material. Spread of the bacterium in the greenhouse can be minimized by low humidity, low temperatures, and bottom watering. Irrigations with an overhead boom may result in splash dispersal of the bacteria throughout the greenhouse. Spread of the bacteria may be reduced by practices that minimize or eliminate long periods of leaf wetness. It would also be advisable to segregate different seed lots in the transplant house to reduce the chance of cross contamination. Decontaminate a transplant house that had infected seedlings and wait at least two to three weeks to plant cucurbits in it again. A 0.525% sodium hypochlorite solution (1 part laundry bleach to 9 parts water), or a commercial bactericide may be used for decontamination.

The possibility of the fruit blotch bacterium being introduced into the field from contaminated volunteer watermelons can be reduced by plowing under debris, including watermelon culls, from an infected field. The cur-

rent field should be planted as far as possible from the previous crops. Volunteer watermelon seedlings from previous crops should be destroyed immediately to eliminate potential sources of the fruit blotch bacterium. In northern growing areas in the United States, fruit blotch-affected fields should be planted subsequently to crops that utilize herbicides that will kill volunteer watermelons. Eliminate wild cucurbits and volunteer cucurbits near transplant houses and production fields. Do not work in an infested field if the foliage is wet. Decontaminate irrigation or mechanical equipment before moving it from an infested field to a noninfested field.

Fruit of some cultivars are more susceptible to bacterial fruit blotch than other cultivars. Susceptibility of fruit appears to be related to rind color, with the darker green rind less susceptible than the light green rind. Field observations would also indicate that the fruit of triploids may be less susceptible than diploids. However, the level of resistance currently available in commercial watermelon cultivars will not insure freedom from fruit blotch under conditions which favor disease development.

Figure 1. Watersoaking on the lower surface of watermelon cotyledons.

Applications of copper-containing fungicides have reduced the incidence of fruit blotch symptoms when applications were initiated prior to fruit set. A minimum of two to three copper applications and thorough coverage of the foliage are essential for good disease control. Applications should begin at first flower, or earlier, and continue until all fruit are mature. For control of other foliar diseases, copper-containing fungicides should be used in combination with a broad-spectrum fungicide, if labels permit such usage.

Application of copper often causes some marginal yellowing of watermelon foliage. Under some conditions, slight stunting of the vines may also occur. Field studies have shown that the stunting and marginal yellowing have no detrimental effect on yield. In South Carolina in 1994, weekly applications of copper-containing fungicides at half the recommended rate appeared to give slightly better control of fruit blotch than applications every 14 days at the recommended rate. Marginal chlorosis was noted after the first application of copper but later applications appeared to have no phytotoxic effect. Stunting was not observed with any of the materials or rates.



Figure 2. Lesions in the cotyledons of watermelon seedlings.



Figure 3. Lesions along midrib of leaf.



Figure 4. Initial small, greasy-appearing, water-soaked lesions in watermelon fruit.



Figure 5. Large, water soaked lesion covering much of the upper surface of the fruit.



Figure 6. Atypical lesions with very little water soaking.



Figure 7. Restricted type lesions that often develop on young fruit.

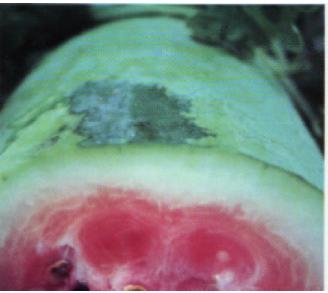


Figure 8. Cross section of fruit blotch lesion demonstrating that lesions are limited to the melon surface initially.



Figure 9. Advanced fruit lesions including cracks in fruit.



Figure 10. Advanced fruit decay accompanied by gas formation.

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