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Bacterial Fruit Blotch of Watermelon

The Hypothetical Exam Question Becomes Reality

Consider the following: "A new disease has appeared on a crop of high unit value in your state. The disease appears to occur sporadically; incidence of affected fields is relatively low. However, near-total losses occur in affected fields. Inconspicuous lesions occur on foliage, but fruit symptoms render the crop unmarketable. Explain how you would determine the cause of the problem. Eventually you learn that the disease is caused by a bacterial pathogen. Describe your investigations into the etiology and epidemiology of the disease. Discuss all possible control options." Sound familiar? We all have responded to that type of question in an examination during our graduate studies, but very few of us have been challenged by such a scenario in real life. The question requires us to apply our compiled experience from the study of plant pathology with our investigative logic to describe an appropriate approach to solve the problem. Bacterial fruit blotch of watermelon offered that challenge to us.

The objective of this paper is to share some of our experiences with this disease. To be sure, the study of bacterial fruit blotch is a fascinating academic exercise. However, our excitement is tempered by the realities of plant disease epidemics in modern times: economic loss due to devastated crops and subsequent high-stakes litigation. The paper includes a description of symptoms based on our observations and the first illustrated disease cycle for bacterial fruit blotch of watermelon. We also trace the chronology of this disease over the past 30 years. Finally, we offer our opinions about the socioeconomic effects of bacterial fruit blotch and the prospects for long-term disease management.

Description of the Disease

Bacterial fruit blotch is a disease of watermelon seedlings and fruit caused by *Acidovorax avenae* subsp. *citrulli* (Schaad et al.) Willems et al. (27). The disease first appeared in commercial watermelon production areas in the United States in 1989 (7,14). Early season outbreaks can result in total loss of fruit at harvest. Fields near severely affected crops can suffer 5 to 50% loss, depending on environmental conditions and the crop growth stage at which fruit blotch becomes established. Although extensive losses have been reported only on watermelon, symptoms on muskmelon have been observed in the field. Pathogenicity on other cucurbits has been demonstrated in research greenhouses and controlled environment facilities.

The characteristic symptom of bacterial fruit blotch is the dark olive green stain, or blotch, on the upper surface of infected fruit (Fig. 1A). Initially, the blotch on watermelon fruit may measure less than 1 cm in diameter. It rapidly expands, so that much of the fruit surface is covered with the lesion in 7 to 10 days (Fig. 1B). As the blotch increases in size, the initial infection site becomes necrotic. The epidermis of the rind ruptures in advanced stages of lesion development (Fig. 1C) and frequently oozes a sticky, clear, amber substance. Fruit lesions rarely extend into the flesh of the melon (Fig. 1D); secondary, rotting organisms are responsible for the ultimate decay and collapse of fruit. Bacterial fruit blotch also attacks muskmelon fruit, often producing water-soaked pits on the fruit surface (Fig. 1E).

Lesions occur on leaves, although foliage surrounding infected fruit may appear healthy to the untrained eye. Leaf lesions are small, dark brown, somewhat angular, and generally inconspicuous (Fig. 1F). When viewed from the bottom of the leaf, the margins of the lesions appear water-soaked, especially in wet or humid weather. Stems, petioles, and roots usually are not infected.

The fruit blotch pathogen also affects watermelon seedlings. Initial symptoms on seedlings appear as water-soaked areas on the underside of infected cotyledons (Fig. 1G). On young seedlings, lesions on the hypocotyl may result in a collapse of the emerging plant. As cotyledons expand, lesions become dark brown and often extend along the length of the midrib (Fig. 1H). Lesions on young true leaves are small, dark brown, and often surrounded by a band of yellow tissue (Fig. 1I). They frequently develop along major leaf veins.

Disease Cycle

Knowledge of the bacterial fruit blotch disease cycle was generated only within the past few years. Figure 2 illustrates the elements of the cycle. We assume that the cycle begins with contaminated seed (step 1), the source of the fruit blotch epidemics in 1989 (18,25). Although direct seeding remains a common practice in southern watermelon production areas, northern fields are almost exclusively transplanted. Transplants also are gaining popularity in the South because of the expense of hybrid diploid and/or triploid seed. Seeds for transplant production are planted in a soilless potting medium in plastic trays (step 2). A warm, humid environment (favorable for disease establishment [10]) is maintained in transplant production facilities used for raising seedlings. Bacteria from infested seed infect the developing seedling as the cotyledons emerge from the seed coat. Almost all facilities employ overhead irrigation, an effective means of splash-dispersing bacteria to neighboring seedlings (step 3) (9). Secondary spread in the transplant house can be responsible for significant proportions of infected seedlings reaching the field (step 4) (10). As plants grow in the field, the pathogen spreads to new leaves and neighboring plants (step 5). Infected plants are not killed and vines do not collapse, but lesions on foliage provide a source of inoculum for infection of immature fruit. Characteristic blotch symptoms appear on fruit shortly before they ripen (step 6).

As diseased fruit decay in the field, seeds associated with affected fruit slip to the soil (step 7). These seeds produce infected volunteer watermelon seedlings the following season and may serve as a

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source of local inoculum. It is believed that infested rind and, in southern states, cucurbit weeds also threaten subsequent watermelon crops (step 8). The magnitude of the threat posed by local inoculum (step 9) is probably much lower than that of infested seed; however, definitive studies quantifying the extent of the threat are lacking.

Disease Chronology

The first account that clearly described the disease that we know as bacterial fruit blotch of watermelon was published in 1969 (2). In their report, Crall and Schenck described a bacterial fruit rot of watermelon that they observed at the Leesburg, Florida, experiment station during the previous 2 years. The paper includes photographs with unmistakable symptoms of bacterial fruit blotch, but the etiology was not determined. In their brief report, the authors implied that this fruit rot was associated with a bacterial leaf spot observed at the same experiment station several years earlier (15), and they referred to a seedborne disease of plant introduction watermelon seedlings described by Webb and Goth (26). In the 1963 report (15), the bacterial leaf spot was observed in commercial fields; but the pathogen was later identified as *Pseudomonas syringae* pv. *lachrymans,* and it did not cause fruit spot symptoms (11). Webb and Goth (26) isolated bacteria that produced white colonies on potato-dextrose agar and beef extract agar, but they did not identify the pathogen. The source of inoculum responsible for the infected watermelons observed at Leesburg in 1967 and 1968 is unknown. Goth and Webb (5) published an abstract in 1975 regarding a bacterial wilt of watermelon seedlings. Several of their observations are inconsistent with currently established characteristics of bacterial fruit blotch, but they concluded that the pathogen was a nonfluorescent pseudomonad that could be seed transmitted. Because they did not complete an etiological study of the disease(s) they observed, an association between their observations and the disease reported by Crall and Schenck is speculative at best. However, in hindsight, it seems likely that the seedling blight described by Webb and Goth (26) and the fruit rot described by Crall and Schenck (2) were caused by the same bacterial pathogen. Crall had planted several plant introductions in his breeding field in 1967 and 1968 that could have harbored the seedborne bacteria. Based on our current knowledge of the epidemiology of watermelon fruit blotch (9,10,12,17,24), it is also possible that weather conditions were unfavorable for the expression of symptoms on fruit in Georgia during the Webb and Goth experiments, whereas conditions favored symptom development on fruit in Leesburg. Further evidence of the connection between the two reports was obtained recently when three isolates of the watermelon seedling blight bacterium from the New Zealand Culture Collection were found to cause bacterial fruit blotch on watermelon (D. L. Hopkins and R. E. Stall, *unpublished*).

Nearly 10 years after the Crall and Schenck (2) paper was published, another description of bacterial fruit blotch appeared in a plant disease handbook published in Queensland, Australia, in 1978 (16). The description was very brief, but a photograph of a watermelon fruit with typical symptoms was included, and the pathogen was described as a *Pseudomonas* species. No mention was made of the extent of disease incidence or losses, or where the disease was observed. According to our research, this was the first time

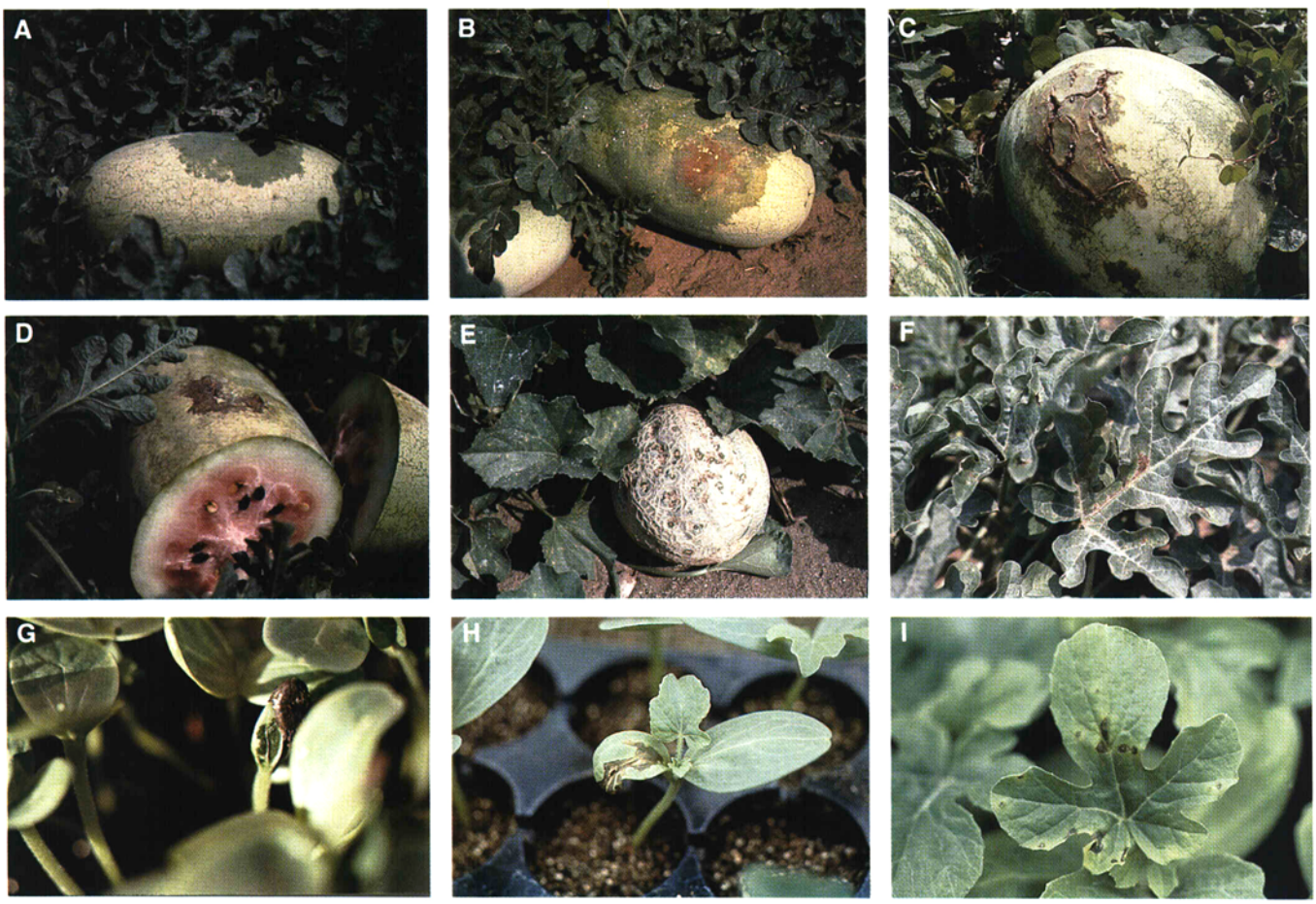


Fig. 1. Symptoms of bacterial fruit blotch of watermelon. (A) Typical olive green stain or blotch on fruit, (B) advanced lesion on fruit, (C) ruptured rind associated with advanced infection, (D) cross section of infected watermelon, (E) symptoms on muskmelon fruit, (F) lesions on watermelon leaves, (G) water-soaked cotyledon on a young, infected seedling, (H) typical necrotic lesion on an expanded cotyledon, and (I) lesions on true leaves of young transplants.

that "fruit blotch" was used as the common name. The same year, a scientific name (*Pseudomonas pseudoalcaligenes* subsp. *citrulli*) was assigned to a bacterial pathogen associated with a seedling disease of plant introductions at the Regional Plant Introduction Station at Experiment, Georgia (19). Schaad et al. (19) showed that the bacterium was similar to the unidentified nonfluorescent bacterium described by Webb and Goth (26). In 1979, Sowell and Schaad (21) published a paper describing the etiology of this seedborne bacterial disease of watermelons. According to the authors, they were working with the same bacterial pathogen described by Webb and Goth and reported severe problems only in the seedling stage. Goth and Webb (6) published one more paper on the disease. In 1981, they evaluated seedlings of 39 watermelon cultivars for resistance to *Pseudomonas pseudoalcaligenes* subsp. *citrulli*. They made no observations on watermelon fruit and made no reference to the Crall and Schenck paper of 1969 (2).

The connection between the seedling disease and the fruit blotch symptoms was not to be made until much later. Furthermore, until the outbreak in the Mariana Islands in 1987, fruit blotch on watermelon appeared to be little more than a curiosity.

Wall and Santos (23) ushered in the modern age of bacterial fruit blotch with their abstract and presentation at the 1988 annual meeting of the American Phytopathological Society. For the first time, the scientific name *Pseudomonas pseudoalcaligenes* subsp. *citrulli*, the common name fruit blotch, and watermelon fruit with characteristic symptoms were associated with the same phenomenon. In a subsequent presentation and abstract, Wall (22) connected the seedling disease and the fruit symptoms and accurately described parts of the disease cycle. Wall et al. (25) later published a brief report that further documented their work and concluded that the pathogen was seedborne. Interestingly, they cited the paper of Schaad et al. (19). Wall and Santos (24) described their research in greater detail in a paper that accompanied their presentation at the 1989 College of Arts and Sciences Research Conference.

By the time Wall presented his research in 1989 at the annual APS meeting in Richmond, Virginia, the first fruit blotch epidemics in commercial watermelon fields in the United States had taken a toll in Florida, South Carolina, and Indiana. Hopkins (7,8) reported his observations in 1989 and demonstrated differences in susceptibility to fruit infection among watermelon cultivars. Latin and Rane (14) reported the occurrence of bacterial fruit blotch in Indiana and later published an abstract on their initial investigations into the disease (17). Somodi et al. (20) described the biochemical characteristics of the pathogen and raised doubts concerning the taxonomic classification of the bacterium. Willems et al. (27) reclassified the pathogen from the genus *Pseudomonas* to *Acidovorax* in 1992. Rane and Latin (18) described the association of the pathogen with seed and, in a popular article (13), warned of the danger of this seedborne pathogen to transplant growers. After these initial reports and descriptions of the disease, research on the etiology and epidemiology of the bacterial fruit blotch continued (4,9,10,12), and reports of bacterial fruit blotch outbreaks in other states appeared (1,3).

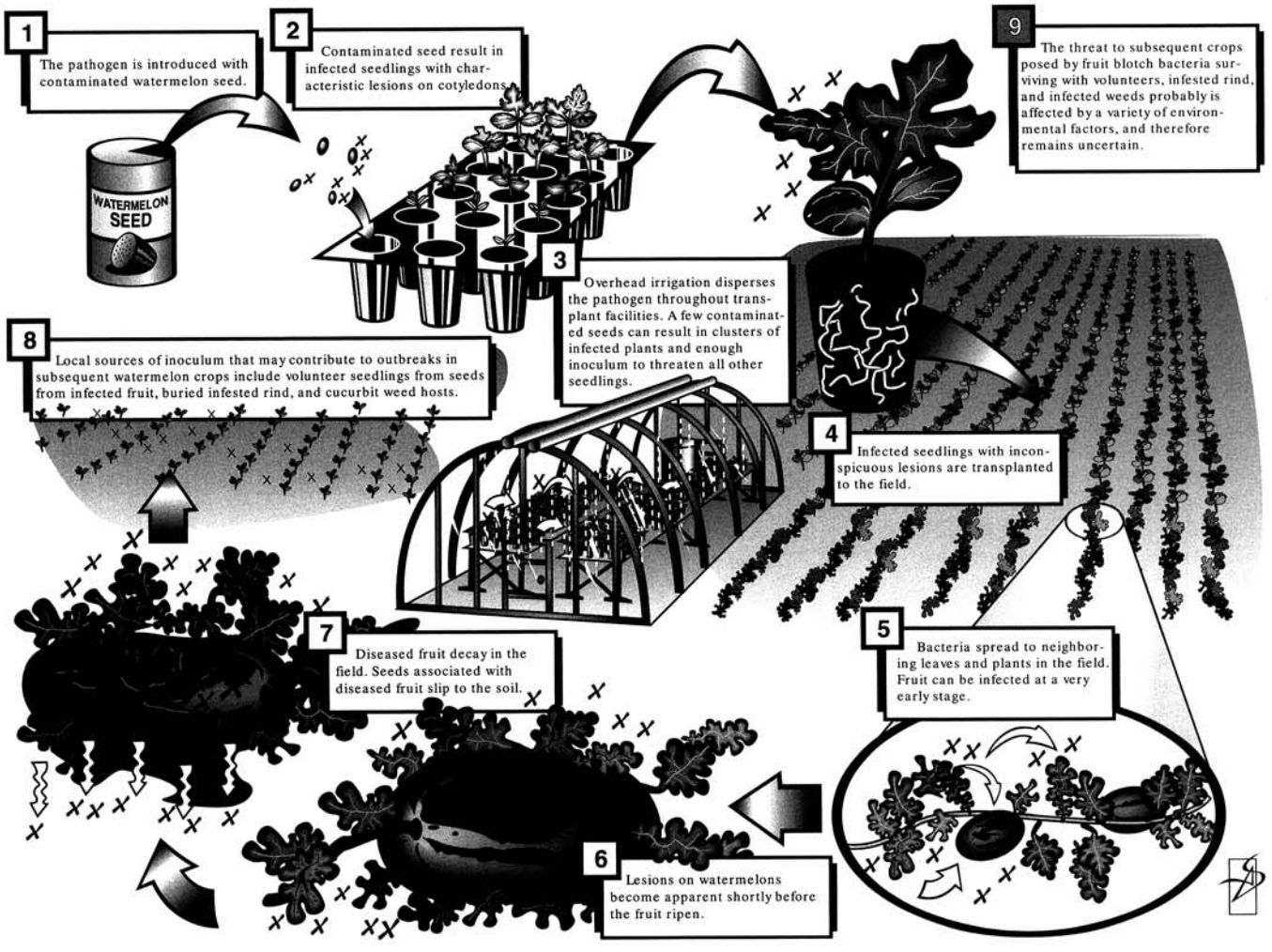


Fig. 2. Disease cycle of bacterial fruit blotch. Steps 1 through 9 illustrate the cycle from the introduction of the pathogen with contaminated seed to the survival of the pathogen in local fields.

Bacterial Fruit Blotch and the Uninformed Public

There is a sensational nature about this disease, although the economic losses do not approach the magnitude of losses associated with other modern epidemics such as citrus canker and southern corn leaf blight. The newness of the disease, the gruesome appearance of infected fruit, and the severe losses sustained in affected fields all contributed to an unusual amount of attention from newspaper and television reporters. In May of 1994, after outbreaks were discovered in transplant production facilities in southeastern states, national wire service reporters wrote headlines warning consumers of "exploding watermelons." It is unclear how the term "exploding" became associated with fruit blotch symptomatology (it was rumored to have been used by television reporters in Georgia in 1992), but it played well in the press and appeared in numerous widely circulated newspapers. Several plant pathologists, including the authors, were cited in many of the articles. Although we insisted that explosions are not part of the fruit blotch syndrome, the headlines suggested otherwise. Fortunately, the story died before the prime months of watermelon consumption, and there appeared to be no reduction in sales, despite the foreboding headlines. Working with an uninformed news media and the consequences of their inaccurate reporting can be a major frustration encountered by those of us with extension responsibilities. For the record, we are not aware of any fruit blotch–related injuries to workers or consumers!

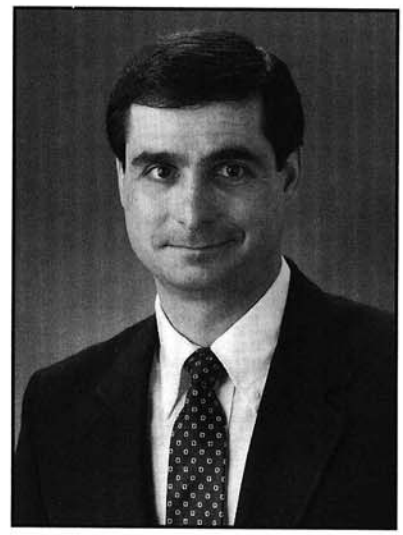
Litigation regarding bacterial fruit blotch is another source of frustration. A number of growers who sustained heavy losses due to bacterial fruit blotch have filed suit against seed companies for selling contaminated seed. They claim damages associated with the crop failure. The value of a watermelon crop differs from one region of the country to another; therefore, it may be difficult to determine what constitutes a reasonable claim. However, regardless of where the epidemic occurs, it is likely that the magnitude of the loss will be much greater than the original cost of the seed, which is the liability of the seed company stated on the label of the seed package. Various seed companies have responded to grower claims differently. Plant pathologists are often called as expert witnesses when the parties cannot agree on a settlement and the complaint goes to court. The courtroom experience is interesting and exciting but also somewhat frustrating. Because most citizens are not well-versed on the nature of plant disease, the jury is likely to be unaware of different types of pathogens, mechanisms of dispersal, and modes of survival and transmission. Therefore, an expert witness (plant pathologist) is guided by the attorney under direct examination to educate the jury on the basics of plant disease and, in particular, the disease in question.

Fruit Blotch as a Threat to the Industry

Bacterial fruit blotch of watermelon and the resulting litigation has been devastating to the entire watermelon industry. Because of the pending lawsuits and the risk of future litigation, most of the major watermelon seed companies suspended the sale of seed in the United States in the fall of 1994, thus threatening the very existence of the watermelon industry in 1995. The possibility that there would be no watermelon seed for the 1995 season captured the attention of the growers as well as of the national media. In the later months of 1994, all but two or three of the companies resumed the sale of seed, but they required growers to sign forms stating that the company was not liable for the possible presence of the bacterial fruit blotch pathogen on the seed. Some companies also labeled their seed "not for sale" in certain states where they felt the risk of disease loss or litigation was greatest.

Most companies have begun assaying their seed lots for the pathogen by using a 10,000-seed-growout test under conditions favorable for symptom development, as recommended by researchers. The label on commercial packages of watermelon seed now includes a statement that the seed lot tested negative for the fruit blotch pathogen, but that absence of the pathogen cannot be guaranteed. Changes in seed handling and packaging practices have increased production costs. The destructive testing (growouts) of expensive hybrid and triploid watermelon seed is especially costly. These costs are passed on to the user, resulting in substantial increases in the price paid for watermelon seed by farmers.

A positive consequence of the bacterial fruit blotch epidemics is that various parts of the watermelon industry are uniting to work toward the common goal of control-



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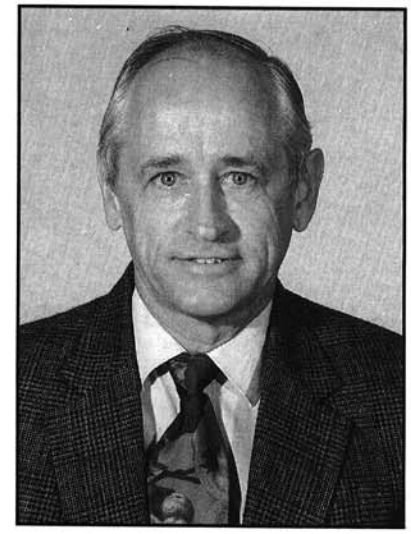

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ling this disease. The seed companies, transplant growers, watermelon growers associations, private researchers, and public research and extension personnel have assembled to support and conduct research to investigate the disease and to explore all possible management options. As a direct result of meetings initiated by industry, a color bulletin co-authored by eight university scientists from four states was produced for educational purposes and distributed with each purchase of watermelon seed in the United States. The sense of cooperation is encouraging, and the future of the industry is perhaps not as grim as it appeared in the summer of 1994.

Future of Bacterial Fruit Blotch

Bacterial fruit blotch is no longer considered a new disease. The disease cycle is fairly well understood. Etiological and epidemiological research, supported by state agricultural experiment stations and industry, continues at public institutions. Short-term strategies for disease management have been proposed and, in many cases, successfully implemented. Debate over long-term disposition of this pathogen on a national and perhaps global scale is about to begin.

Growers often ask how long they will have to deal with bacterial fruit blotch. The answer to the question probably will vary with different regions of the country. In the watermelon production regions of the southeastern United States, uncertainty regarding the perennation of the pathogen exists because cucurbit weeds that survive in ditch banks and wooded areas may harbor the bacteria. Crop rotations in the South generally are long enough to eliminate the threat from infested residue. However, infected volunteer plants could provide a source of inoculum for subsequent crops, especially in areas adjacent to fields that sustained fruit blotch epidemics. The potential for survival on wild cucurbits or epiphytically on nonhost species may help explain the incidence of fruit blotch in Florida each year since 1989.

The situation is different in Indiana, where outbreaks occurred only in 1989, 1992, and 1994, years in which the disease was clearly associated with contaminated watermelon seed. Wild cucurbits are uncommon in the Midwest, and it is likely that the disease can be avoided if seed distributed to farmers is not contaminated. Although crop rotations are generally shorter in the Midwest, growers can sufficiently increase the interval between susceptible crops to eliminate the threat of fruit blotch from local inoculum sources such as infested rind and infected volunteer plants. Consider that bacterial fruit blotch also occurred in Iowa in 1989 but has not occurred since. It happened that the Iowa farmer purchased transplants from an Indiana grower who had a contaminated lot of seed and who suffered outbreaks of the disease in his transplant production facilities and his fields. The Iowa farmer has not observed fruit blotch in his fields since 1989, when it was introduced with the infected plant material. If long-term survival in soil or crop residue were a trademark of this pathogen, then fruit blotch epidemics would occur annually in midwestern production regions. Outbreaks that were reported in Delaware and Maryland in 1989 also have not recurred, supporting the contention that introduction of the pathogen with infested or infected plant material is essential for outbreaks in northern states.

Sanitation practices in transplant production facilities also can effectively eliminate the fruit blotch threat. The two Indiana growers who sustained initial fruit blotch outbreaks in their plant houses in 1989 have not had a recurrence, nor have the six other Indiana growers who planted contaminated seed and had plant houses with infected seedlings in 1992. At the time of this writing (April 1995), no new outbreaks of fruit blotch have been reported, although many of the transplant facilities that were affected in 1994 resumed production in 1995.

Assuming that the host range of *A. a.* subsp. *citrulli* is limited to cucurbits, and that survival within infested rind and volunteer melon plants is not long-term, one must ask whether eradication of the disease from U.S. production regions is an attainable goal. It is possible, but it will take a concerted effort by growers, transplant producers, and most importantly, the seed industry to eliminate the threat of bacterial fruit blotch in the U.S. watermelon industry.

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