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Disease Control in Tomato Transplants Produced in Georgia and Florida

The transplant industry in southern Georgia began in 1908 when a few growers in the Tifton area started to produce field-grown plants for sale to home gardeners and producers in more northern areas (1). Attempts to establish the industry on a commercial scale began in 1914 when an Indiana tomato packing company arranged with several Tifton area growers to plant 771 kg of seed for transplant production. This first commercial venture was not totally successful because growers had limited experience producing transplants on such a large scale and because a late freeze injured plants. However, the growers learned as additional plantings were attempted during 1915–1917. The commercial industry was established successfully in 1918, when plantings were extensive in the Tifton area.

Southern Georgia was especially suitable for tomato transplant production because plant harvest coincided with recommended transplanting dates in the northeastern and midwestern states. Field-grown plants were desirable because they were sturdier and less susceptible to transplant shock than plants from greenhouses or cold frames. Another major advantage was their competitive price.

By 1934 more than 1,000 ha in Tifton and surrounding counties were devoted to tomato transplant production, and 150 million transplants were shipped to northern fruit producers. Production continued to increase and reached 4,112 ha by 1946. By the late 1940s and early 1950s, more than 90% of transplants for production of processing tomatoes in the central and Atlantic states were grown in the South, mostly in southern Georgia.

Transplant Production

Until recently, the southern United States has been the major source of field-grown tomato transplants for the northeastern states and southern Canada. In Georgia, fields are seeded during February through late April, and plants are shipped within 65 days, depending on the cultivar, seeding date, environmental conditions, and management practices. Transplants usually follow a cover crop of rye, which is harrowed and deep-turned with a moldboard plow. Strips of rye are left as windbreaks to reduce damage by windblown sand. Coated tomato seeds are planted a few millimeters deep with a precision seeder so that seeds are spaced approximately 1 cm apart in double zigzag rows, with four or five double rows per bed. Depending on conditions, fields are irrigated three or four times daily to soften the soil and ensure that seedlings emerge properly (12). Plants are harvested when they attain marketable size. Plants are clipped (Fig. 1) several times to maximize yield, reduce labor costs, and promote uniform plant growth, which allows a single harvest.

Transplants are harvested when foliage is dry and the soil is not excessively wet. Before harvest, the plant beds are undercut with a steel bar to loosen the roots. Usually, plants are pulled by hand and soil is removed from the roots by vigorous shaking or slapping the roots against the worker's leg. Plants are packed loose with bare roots in wooden crates. Bundling of plants and wrapping of roots in wet moss, once a standard practice, has been discontinued because loosely packed plants separate fairly easily and survive very well. Usually 1,000 plants are packed per crate. Crates of plants are taken to a packing shed, then transferred to environment-controlled tractor-trailer trucks and shipped to northern markets.

The tomato transplant industry offers a unique perspective on disease control. Most tomato transplants are produced within guidelines of a state-governed inspection program. Plants produced under the certification program must meet requirements of crop rotations, seed treatment, insect control, horticultural standards, and disease control (a theoretical zero percent tolerance applies to some diseases). Diseases in other crops are economically important because they affect quality or lower yields, but diseases in tomato transplants can result in certification rejection and complete crop loss. Plants without a phytosanitary certificate cannot be exported to Canada. Adverse weather conditions in the north and/or south often delay harvest. A short growing season combined with a favorable climate for disease development makes producing field-grown transplants a hectic, risky, and tense business.

Containerized Transplants Grown in the Greenhouse

Although once used exclusively for fresh market crops, container-grown tomato transplants in recent years have found a place in the processing tomato market. More expensive than field-grown transplants, containerized transplants grow more uniformly and survive equally well under most field conditions. Seeds are planted into individual cells of expanded or hard polystyrene containers. The cells are inverted cones or pyramids with open bottoms to permit air-pruning of roots. Planted flats are watered and placed in a germination chamber for 3 days. The trays then are

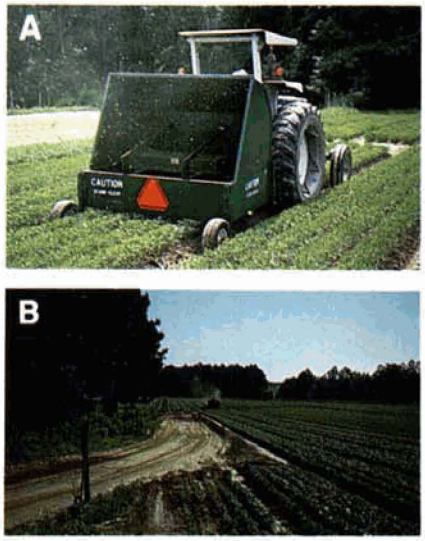

Fig. 1. The practice of clipping tomato transplants to promote uniform growth and maintain plant quality has the potential for disseminating bacterial pathogens in the (A) particulate debris and (B) aerosols generated.

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placed in a growing house and watered two to six times a day. Fertilizer in the potting mix is sufficient for growth to the cotyledon expansion stage. Liquid fertilizer is applied overhead on a 3-day schedule, then less often during the finishing stage. Transplants are hardened for field planting by reducing nitrogen concentration and frequency of watering. Gradual lowering of the growing house temperature aids in the hardening process when plants are grown for a winter crop. In spring, as heat units increase, seedlings in the greenhouse are harvested 2–3 weeks earlier than field-grown transplants.

The process of hardening plants before transplanting can result in the development of necrotic spots of unknown etiology that are currently being attributed to stress. Symptoms of diseases of biotic origin, including bacterial spot, may be atypical under these conditions or may be masked by the stress-related symptoms. Isolation from these lesions often yields diverse microorganisms other than pathogens. Thus, identification of the cause may be hindered by the presence of secondary microorganisms.

Overhead irrigation of plants enhances both spread and development of diseases on seedlings in containers. In Florida, some transplant production facilities are located close to field production areas, and movement of pathogens into the production area by mechanical means, aerosols, insects, or windblown rain can result in disease on seedlings. Movement of bacteria from these sources is always a threat in Florida, where tomatoes are grown from August through May. The situation in Georgia is different because most transplant production (field or greenhouse) is far removed from fruit production areas.

Regardless of location, thorough sanitation is the key for a successful disease control program. All frames, trays, and tools should be disinfested with chlorine, bromine, or formalin solutions before reuse. Pathogen-free soil or potting mix is essential for control of soilborne pathogens. Control of weeds in and around the greenhouse is recommended. Air circulation and watering schedules are managed to minimize the duration of leaf wetness without causing water stress. Plants should be handled as little as possible to avoid mechanical transmission of bacteria and viruses. To prevent transmission of tobacco mosaic virus, workers should never smoke while handling plants. Pesticide recommendations for disease control in the greenhouse are similar to those for field-grown transplants except that certain chemicals are not registered for greenhouse use.

Early Troubles in the Transplant Industry

From the early years until the late 1960s, growers produced transplants on "new" or recently cleared land and moved to new sites every 2 to 3 years, primarily to avoid weed, nematode, and disease problems. Shifting cultivation provided a measure of pest control but did not lend itself to increased efficiency. The area planted often was excessive to ensure an adequate supply. Irrigation used on a small scale was often not available because systems were moved from location to location. Stumps and debris in newly cleared land interfered with mechanization of operations. Nutrition problems were common because native soils were acid and infertile. Nonuniformity of plant size was a common problem.

The scarcity of new land coupled with the need to improve plant yield, quality, and size uniformity led to major changes during the late 1960s and 1970s. Shifting cultivation sites was changed to the more efficient system of permanent production sites on previously cultivated land. Precision seeding, high plant populations, prescription fertilization, irrigation, pesticides, and clipping became common practices and led to marked increases in yields per hectare. By the 1980s, about 900 million tomato transplants were produced on less than 2,000 ha within a 80-km radius of Tifton, Georgia.

Early enthusiasm for southern-grown transplants soon was tempered by the onset of diseases that could not be ignored. As early as 1925, New York growers were advised to get a "guarantee of health" with the purchase of southern transplants. In 1929, New Jersey growers were complaining about collar rot (caused by *Alternaria solani* Sorauer) and other diseases on plants from the South. In 1933, Maryland growers reported that bacterial canker occurred in some fields where southern-grown plants were used. Over the years, disputes about diseases caused more friction between southern transplant growers and northern tomato producers than any other issue.

In those early years, attempts to control transplant diseases were hampered by a paucity of information on the basic etiology and epidemiology of the causal organisms, lack of chemicals and technology to control diseases, and ignorance of plant diseases by both southern transplant growers and northern fruit producers. Ignorance and mistrust on the part of both groups resulted in counterproductive actions rather than solutions to mutual problems. Southern growers denied responsibility, contending that the pathogens originated in the North in association with seeds and that important diseases in the South were already present in northern fields. In 1929, O. C. Boyd, attempting to establish seedborne inoculum as the culprit in the transplant disease problems, planted 42 seed samples from grower stocks in Georgia fields where no tomatoes had been previously grown and shipped the harvested plants to cooperators in New Jersey and Delaware. These workers observed several diseases (early blight, nail-head spot, bacterial spot, bacterial canker, and Septoria leaf spot) in both southern and northern fields and concluded that seeds were indeed a major source of primary inoculum. This early work established the importance of seedborne inoculum and emphasized the need for full cooperation of all parties in finding solutions to common disease problems.

Between 1942 and 1976, early blight, bacterial spot, bacterial wilt, and southern blight were the most important diseases responsible for the revocation of transplant certification. Root-knot nematodes also caused sizable losses (23).

Recent Problems in the Transplant Industry

Foliar fungal diseases such as late blight and early blight that were so destructive in the early years of the industry have become manageable with the advent of effective fungicides and efficient spray equipment. Bacterial diseases that have occurred sporadically over the years have been the most difficult to control. These diseases and tomato spotted wilt virus have received increased attention in recent years.

Bacterial wilt. Bacterial wilt continues to defy control, and the only effective measure is to avoid infested soils. The causal organism, *Pseudomonas solanacearum* (Smith) Smith, is a complex species that is divided into strains, biotypes, and races without universal agreement as to what constitutes a valid subspecies description. In southern Georgia, race 1 predominates and occurs frequently on tomato and potato but does not infect tobacco often.

Under favorable conditions, *P. solanacearum* can survive in soil for extended periods in the absence of host plants and in association with weed hosts, some of which are present in transplant fields in southern Georgia. Survival in soil in Georgia transplant fields has been unpredictable. Sometimes the bacterium has disappeared rapidly when new fields were brought into transplant production, but long-term survival has been observed in other transplant fields. In most soils, infestation of *P. solanacearum* is highest in the top 30 cm of the profile, but low populations have been recovered as deep as 60–75 cm (19). Well-drained soils with good water-retention characteristics are conducive for the pathogen's survival. Other soil conditions that promote survival are moderate to high temperatures and low to moderate pH. Soils that allow desiccation of the pathogen or promote antagonistic organisms are detrimental to survival. The role of antagonistic organisms in the survival of the pathogen is

still poorly understood. Because infection and disease development are favored by high temperature (optimum 30–35 C) and high moisture, bacterial wilt is most severe on tomato transplants late in the spring. Sometimes transplants grown in soils known to be infested with the bacterium escaped infection if harvested early enough in the season (18).

Bacterial speck. Bacterial speck (Fig. 2A) first became significant in 1978, when the disease resulted in the rejection of some 160 ha of transplants. Circumstantial evidence suggested that the outbreak resulted from the introduction of *Pseudomonas syringae* pv. *tomato* (Okabe) Young et al on seed. The bacterium was recovered from 19 of 1,566 samples of commercial tomato seed used for transplant production in Georgia during 1979–1982 (20).

Although *P. s. tomato* survived epiphytically on weed species in northern Georgia and elsewhere, the significance of its survival on weeds in southern Georgia transplant fields is questionable. The bacterium has not been isolated from weeds or volunteer crop species growing in transplant fields despite repeated attempts (20). Also, there is no evidence that the bacterium survives in soil or host debris in southern Georgia. In field tests at Tifton, the bacterium was not detected in artificially infested soil (10^8 cfu/g) or in buried host tissue assayed 15 days after burial in the field. The bacterium survived less than 45 days in nondecomposed tissue left on the soil surface.

Once introduced into a transplant field, *P. s. tomato* has the potential to spread rapidly by splashing or windblown rain. It also may be disseminated by the clipping process or through aerosols generated during clipping (Fig. 1). Infection and disease development are favored by free moisture and low temperatures (18–22 C). Little disease occurs when mean temperatures are 25 C or higher. Streptomycin and copper bactericides are useful in preventing bacterial speck on tomato transplants and are recommended as part of the disease control program for transplant production (2).

Bacterial spot. Bacterial spot (Fig. 2B), caused by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye, is one of the most destructive pathogens of tomato transplants in Florida and Georgia and, unlike some other bacterial diseases, is a persistent problem. Known as a pathogen of tomato since 1912, the bacterium attacks leaves, fruits, and shoots. Severe defoliation results in yield losses, and spotting may make fruit unmarketable. On greenhouse-grown transplants, lesion development is usually restricted to leaflets. Lesions begin as light brown spots 1–2 mm in diameter and often show water-soaking. They may be difficult to discern from leaf spots caused by other phytopathogenic bacteria or some physiological disorders. Isolations from lesions yield many saprophytic bacteria, which may preclude isolation of the target organism.

Maintaining disease-free transplants is an integral part of reducing field losses from bacterial spot and can be accomplished by addressing the source(s) of primary inoculum and environmental factors that promote disease development. Exclusion of the pathogen from transplant production areas is the main control measure. The disease may become established on transplants from seed infestations or by dissemination from nearby infested fields. The pathogen even has been recovered from the hands of workers who harvest plants. The bacterium also has been detected in aerosols downwind from clipping and harvesting operations (22). Although rain and irrigation have been associated with increasing numbers of airborne bacteria in other areas of the world, airborne *X. c. vesicatoria* cells declined over transplant fields in Georgia after rain or irrigation, which had a "washing out" effect (22).

Although *X. c. vesicatoria* can be seedborne, the importance of contaminated seed in the epidemiology of the disease is relatively unknown. Several semiselective media (10,21,24) recently developed may be useful in elucidating the role of seedborne inoculum. The bacterium can survive on crop residues for 3–6 months and on tomato volunteers for up to 12 months (16). Weeds and soil do not appear to serve as significant reservoirs for long-term survival of the pathogen.

Bacterial spot is difficult to control by chemicals. No bactericide is completely effective, which is necessary when tolerance of disease is zero. Streptomycin and copper bactericides have been used over the years to reduce the incidence of bacterial spot, but resistance to these materials is common (17,25). In the mid-1960s a combination of copper with mancozeb or maneb was found to be more effective than copper alone. In Florida and Georgia, spray recommendations for many years have included copper-mancozeb mixtures for control. In vitro studies have shown that copper-mancozeb combinations are more toxic to copper-resistant strains than copper alone. Apparently the mixture releases more soluble copper than a copper suspension alone, which may partially explain the increased toxicity (17).

New copper compounds devised to increase the copper activity include liquid formulations to increase the soluble copper and granular forms with reduced particle size to provide better coverage. In addition, the new formulations are easier to apply through irrigation systems (chemigation) (Fig. 3). However, all of the formulations have to be mixed with maneb or mancozeb for maximum benefit against copper-resistant strains.

Syringae leaf spot. Syringae leaf spot (Fig. 2C), caused by *Pseudomonas syringae* pv. *syringae* van Hall, was reported first in transplant fields in 1980 and initially was confused with bacterial speck. However, syringae leaf spot was quickly determined not to be a significant threat like bacterial speck (15), and the Georgia Department of Agriculture (GDA) continued certification of plants with the disease. This decision created a problem because certification inspections historically were based on symptoms, and the GDA was not equipped to identify bacterial pathogens in the laboratory. In a cooperative effort, University of Georgia and GDA scientists developed appropriate laboratory procedures that allowed identification of these organisms (13,14). At first, *P. s. syringae* was distinguished from *P. s. tomato* by traditional microbiological methods. *P. s. tomato* does not utilize erythritol or DL-lactate, whereas most strains of *P. s. syringae* do. More recently, indirect immunofluorescence, ice nucleation assays, growth and fluorescence on various substrates, and analysis of fatty acid methyl esters by gas-liquid chromatography have been used. Also, a temporal distribution of the two diseases in southern transplant fields aids in their identification (26). Syringae leaf spot occurs primarily during March and April, whereas bacterial speck appears primarily after the first of May, but because the two diseases overlap in time, isolation and analysis in the laboratory remain the only way to ensure accurate certification.

Opportunistic xanthomonads. The need for rapid identification of bacterial pathogens for certification purposes precluded testing for pathogenicity. Before 1987, any xanthomonads recovered from lesions from either tomato or pepper were assumed to be the bacterial spot pathogen. In 1987, however, it was found that xanthomonads isolated from mixed infections with either *P. s. syringae* or *P. s. tomato* were frequently nonpathogenic. The nonpathogenic xanthomonads were always positive for starch hydrolysis, were pectinolytic when grown on crystal violet pectate medium, and had distinct fatty acid methyl ester profiles, all of which distinguished them from strains of *X. c. vesicatoria* (11). Because there was no accepted nomenclature for these bacteria, they were categorized as opportunistic xanthomonads, and the plants harboring opportunistic xanthomonads alone or in combination with *P. s. syringae* were certified.

The similarity among symptoms of bacterial speck, bacterial spot, and syringae leaf spot and the need to separate opportunistic xanthomonads from *X. c. vesicatoria* required that GDA personnel rely on isolations and identifications in the laboratory. It is ironic that syringae leaf spot, a disease of only "cosmetic"

importance, and an opportunistic xanthomonad served as the impetus for beneficial reform in the certification program.

Bacterial canker. A recent outbreak of bacterial canker, caused by *Clavibacter michiganensis* subsp. *michiganensis* (Smith) Davis et al, resulted in further revision in the certification program. Although a major problem during the early years of the industry, bacterial canker had been of little concern until an outbreak was traced to southern transplants in 1984. Feelings of mistrust reemerged, and some northern producers accused southern transplant growers of dumping inferior and diseased plants on the market for an easy and quick profit. The southern transplant growers insisted they were not to blame and either accused seed companies of selling contaminated seed or claimed that the disease already occurred in the North. The same cause of friction between the different parties of earlier generations occurred again.

The origin of inoculum in diseased fields in the North can be difficult to ascertain once an epidemic has occurred. An obvious conclusion is that the disease originated from a local source of inoculum or that it came on the transplants. One striking feature of diseased plants in the 1984 epidemic was that infection began at the clipped end of the main stem. This was strong circumstantial evidence that the disease originated in the South, where the clipping occurred. However, all transplants associated with the 1984 epidemic were certified as "disease-free" on the basis of their appearance at the time of inspection. Symptoms did not appear on transplants in the southern fields. We now know that clipping plants with latent infections readily disseminates the bacterium throughout transplant beds, and the transplants remain symptomless until transplanted.

A weak link in the inspection process was how to detect symptomless plants with latent infections. The problem was not necessarily one of unavailable techniques. The pathogen can be detected as early as 2 days after inoculation by pressing stem sections onto selective media (Fig. 4) (8). A continuing problem, even with the use of semiselective media, was how to accurately and rapidly identify suspect bacteria recovered from symptomless transplants as well as from soil, seed, weeds, and irrigation water. Traditional microbiological tests required too much time and labor for routine testing on a large scale and almost always needed to be confirmed by pathogenicity tests that took several weeks to complete. Tobacco was inconsistent in the expression of a hypersensitive response when challenged with different strains of *C. m. michiganensis*, but four-o'clock (*Mirabilis jalapa* L.) proved to be a suitable substitute (6). Hypersensitive response occurred independently of temperature within the range tested (18–43 C) and consistently for more than 40 strains tested.

Another useful method for characterizing suspect strains was analyzing fatty acid methyl esters. In particular, the presence of anteisopentadecenoic acid

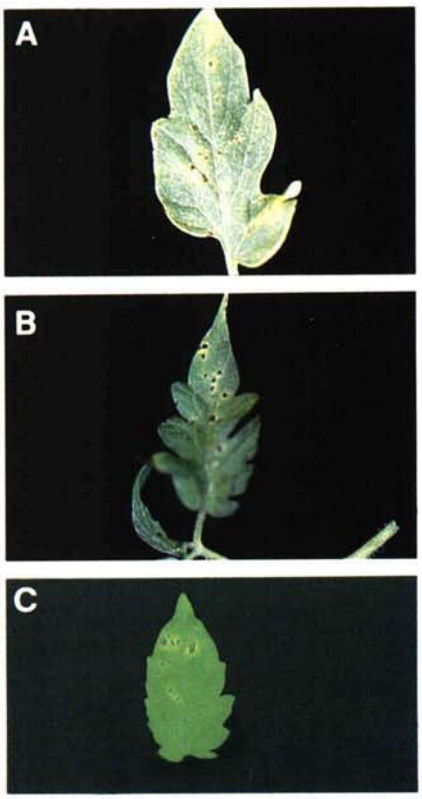

Fig. 2. Leaf spots on tomato leaflets include (A) bacterial speck caused by *Pseudomonas syringae* pv. *tomato*, (B) bacterial spot caused by *Xanthomonas campestris* pv. *vesicatoria*, and (C) syringae leaf spot caused by *P. s. syringae*.

Fig. 3. Experimental application of a liquid formulation of cupric hydroxide through a center pivot simulator to control bacterial spot of tomato.

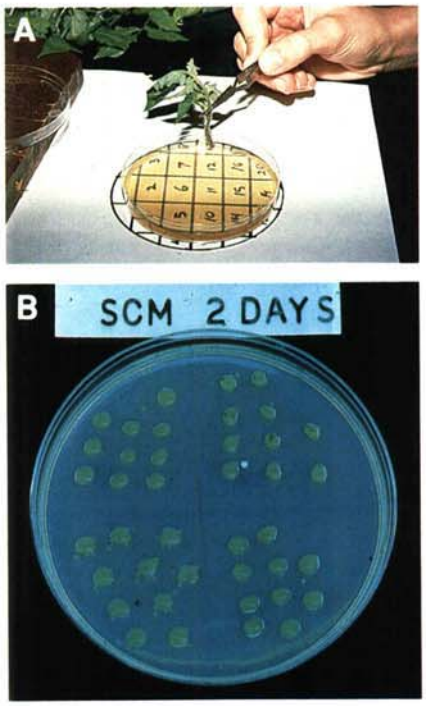

Fig. 4. (A) In the stem printing technique, a piece of tomato stem is gently squeezed and the freshly cut surface is pressed on a semiselective medium. (B) These colonies of *Clavibacter michiganensis* subsp. *michiganensis* were recovered within 2 days after seedlings were clip-inoculated.

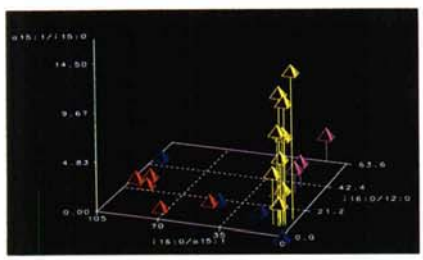

Fig. 5. Three-dimensional plot, based on ratios of percent content of selected fatty acid methyl esters, of relationships of U.S. strains (yellow peaks) and Republic of China strains (pink peaks) of *Clavibacter michiganensis* subsp. *michiganensis* with saprophytic *Clavibacter*-like bacteria (red and blue peaks).

Fig. 6. Isolation of *Clavibacter michiganensis* subsp. *michiganensis* from a tomato seed from a commercial seed lot: (Left) Basal medium, i.e., CNS without antibiotics, and (right) semiselective CNS medium.

Fig. 7. Bronzed leaf surface and ring spots are early symptoms of tomato spotted wilt virus infection.

(an unsaturated branched-chain fatty acid designated as a15:1) was highly specific for *C. m. michiganensis* when compared to profiles of saprophytic bacteria with similar appearance on semiselective media (9). Subjecting the data to principal components analysis or plotting ratios of key fatty acids on a three-dimensional graph has the potential of identifying specific strains or at least arranging strains into common groups (Fig. 5).

Finally, the bacteria could be detected and identified by ELISA, either by analyzing expressed sap from stems or by pressing the cut end of a stem directly into ELISA wells (8). Although this method may be the most practical for screening a large number of samples in the shortest amount of time, a living culture is not obtained for confirmation, and new serotypes could escape detection. The main problem of certifying all transplants to be free of latent infections, regardless of the methodology, is the time, labor, and cost required for processing and analyzing a sample size large enough to ensure that the test results and subsequent certifications are meaningful. Such an undertaking to test symptomless plants for potential latent infections appears impractical.

The source of primary inoculum for the 1984 bacterial canker epidemic was never determined. However, *C. m. michiganensis* has been detected in several seed lots since 1984 (7). Because the bacterium does not appear to survive in transplant fields in southern Georgia, it is likely that seedborne inoculum was responsible for the 1984 outbreak. Research during the 1930s showed that both seed treatment and foliar-applied chemicals could be used in the transplant industry to minimize disease problems. Some of the early seed treatment chemicals included copper compounds, Semesan Jr. and Improved Semesan Jr. (with hydroxymercurichlorophenol as the main ingredient), and corrosive sublimate (mercuric chloride). The mercurial compounds were of particular interest because of their effectiveness against bacteria and the prevalent seedborne bacterial pathogens. Organic mercurials such as Ceresan became widely used until banned in the 1970s. It is tempting to speculate that the loss of the mercurial seed treatment chemicals is responsible for new outbreaks of certain bacterial diseases such as canker in recent years.

Recent steps taken by the industry as a whole have been based on the premise that seeds are important sources of primary inoculum for bacterial pathogens in transplant production. Interest has been renewed in developing seed extraction and seed treatment methods for eradication of *C. m. michiganensis* (3,5). To improve detection efficiency, the GDA has increased its seed sample size from 200 to 30,000 and replaced a nonspecific test incapable of detecting *C. m. michiganensis* with a complex test that includes grinding the seed, centrifuging it at different speeds to concentrate the bacteria and clear debris, and plating it onto various semiselective media designed specifically for detection of *C. m. michiganensis* (Fig. 6), *X. c. vesicatoria*, and *P. s. tomato* (4,7). Also, the disinfestation of clipping mowers and the use of mowers designated for specific plantings have become common practices in transplant production to prevent spread to noninfected plantings should a contaminated seed lot escape detection.

The latest threat: Viral diseases. Until 1989, plant viruses were of little consequence in the transplant industry. The mosaic virus diseases that occur frequently on greenhouse and field-grown tomato are observed very infrequently on tomato transplants in the fields of southern Georgia. The low incidence of viruses in Georgia transplants has been attributed to the absence of seedborne inoculum, low activity of aphid vectors in early spring when transplants are grown, and limited handling of plants until harvest (1).

Tomato spotted wilt (Fig. 7) was first observed on transplants in 1989 and has been of concern since. To date, the spatial distribution of plants infected by tomato spotted wilt virus (TSWV), when plotted over time and studied by doublet analysis or ordinary runs analysis, has been random. Graphs of disease incidence vs. distance from field perimeters were used to compare plant/disease dispersal gradients within and between fields. The observed dispersal gradients were interpreted as evidence of a local source of inoculum and of local movement of thrips from areas immediately adjacent to the fields. In addition, the gradients did not flatten over time. On the basis of these analyses, we concluded that secondary dissemination was inconsequential to TSWV epidemics. This was of particular importance to the transplant industry because clipping was thought to be a potential method of disseminating the virus.

Another threat in Florida, but not yet in Georgia, is a new disease of tomato caused by a geminivirus. Infected plants are severely stunted, and foliar symptoms range from interveinal chlorosis to brightly colored mosaic. Distortion of apical shoots is common, and petioles may droop, giving the appearance of a collapsed plant canopy. Although the disease so far has been restricted to Florida, it has been found in greenhouse-grown transplants and staked tomatoes in counties along the Georgia border. The vector, the sweetpotato whitefly (*Bemisia tabaci* (Gennadius)), is found in Georgia. It is difficult to predict whether this geminivirus will become yet another serious threat to the southern field-grown transplant industry.

The Future of Transplant Production

For years, tomato producers in the northeastern and central United States and south central Canada have depended on southern-grown tomato transplants to provide early field transplantation and production. The number of field-grown transplants produced has varied over the years because of fluctuations in the total number of hectares of tomatoes planted in the north, the rise and fall in popularity of direct seeding, and competition with container-grown plants in greenhouses. Currently in northern states, the popularity of mechanized planting requiring container-grown plug plants has caused a decline in field transplant production to the point that the field-grown transplant industry in southern Georgia is near extinction. The concern of disease problems in field-grown transplants, as occurred with bacterial canker in 1984 and with tomato spotted wilt in 1989 and 1990, has contributed to the demise of the industry.

The expectation that transplant producers should provide absolutely disease-free transplants and that there will be no disease problems in greenhouses is unrealistic. Disease will continue to occur in plants regardless. Zero tolerance in reality is a managerial decision and is related only partly to technological advances in detection and identification methods. Mostly, disease-free certification continues to be based on visual inspection. Escapes, latent infections, and epiphytic populations will continue to cause problems. The best one can hope for is to minimize losses by following recommended cultural and disease control practices, to use seeds produced and treated by a reputable company, and to have seeds and plants inspected and tested by a regulatory agency such as the GDA.

Literature Cited

1. Borders, H. I. 1953. Transplants grown in the South. Pages 463-465 in: U.S. Dep. Agric. Yearb. Agric.
2. Conlin, K. C., and McCarter, S. M. 1983. Effectiveness of selected chemicals in inhibiting *Pseudomonas syringae* pv. *tomato* in vitro and in controlling bacterial speck. Plant Dis. 67:639-644.
3. Dhanvantari, B. N. 1989. Effect of seed extraction methods and seed treatments on control of tomato bacterial canker. Can. J. Plant Pathol. 11:400-408.
4. Fatmi, M., and Schaad, N. W. 1988. Semiselective agar medium for isolation of *Clavibacter michiganense* subsp. *michiganense* from tomato seed. Phytopathology 78:121-126.
5. Fatmi, M., Schaad, N. W., and Bolkan, H. A. 1991. Seed treatments for eradicating *Clavibacter michiganensis* subsp. *michiganensis* from naturally infected tomato seeds. Plant Dis. 75:383-385.
6. Gitaitis, R. D. 1990. Induction of a hypersensitivelike reaction in four-o'clock by

Clavibacter michiganensis subsp. *michiganensis*. Plant Dis. 74:58-60.
7. Gitaitis, R. D. 1990. Survey of phytopathogenic bacteria recovered from commercial tomato seed and their secondary spread within tomato transplant fields. Pages 293-298 in: Proc. Int. Conf. Plant Pathog. Bact. 7th.
8. Gitaitis, R. D., and Beaver, R. W. 1990. Characterization of fatty acid methyl ester content of *Clavibacter michiganensis* subsp. *michiganensis*. Phytopathology 80:318-321.
9. Gitaitis, R. D., Beaver, R. W., and Voloudakis, A. E. 1991. Detection of *Clavibacter michiganensis* subsp. *michiganensis* in symptomless tomato transplants. Plant Dis. 75:834-838.
10. Gitaitis, R. D., Chang, C. J., Sijam, K., and Dowler, C. C. 1991. A differential medium for semiselective isolation of *Xanthomonas campestris* pv. *vesicatoria* and other cellulolytic xanthomonads from various natural sources. Plant Dis. 75:1274-1278.
11. Gitaitis, R. D., Sasser, M. J., Beaver, R. W., McInnes, T. B., and Stall, R. E. 1987. Pectolytic xanthomonads in mixed infections with *Pseudomonas syringae* pv. *syringae, P. syringae* pv. *tomato*, and *Xanthomonas campestris* pv. *vesicatoria* in tomato and pepper transplants. Phytopathology 77:611-615.
12. Jaworski, C. A., Brodie, B. B., Glaze, N. C., McCarter, S. M., Good, J. M., and Webb, R. E. 1973. Research studies on field production of tomato transplants in southern Georgia. U.S. Dep. Agric. Prod. Res. Rep. 148.
13. Jones, J. B., Gitaitis, R. D., and McCarter, S. M. 1983. Evaluation of indirect immunofluorescence and ice nucleation activity as rapid tests for identifying bacterial diseases of tomato transplants incited by fluorescent pseudomonads. Plant Dis. 67:684-687.
14. Jones, J. B., Gitaitis, R. D., and McCarter, S. M. 1986. Fluorescence on single-carbon sources for separation of *Pseudomonas syringae* pv. *syringae, P. syringae* pv. *tomato*, and *P. viridiflava* on tomato transplants. Plant Dis. 70:151-153.
15. Jones, J. B., McCarter, S. M., and Gitaitis, R. D. 1981. Association of *Pseudomonas syringae* pv. *syringae* with a leaf spot disease of tomato transplants in southern Georgia. Phytopathology 71:1281-1285.
16. Jones, J. B., Pohronezny, K. L., Stall, R. E., and Jones, J. P. 1986. Survival of *Xanthomonas campestris* pv. *vesicatoria* in Florida on tomato crop residue, weeds, seeds, and volunteer tomato plants. Phytopathology 16:430-434.
17. Marco, G. M., and Stall, R. E. 1983. Control of bacterial spot of pepper initiated by strains of *Xanthomonas campestris* pv. *vesicatoria* that differ in sensitivity to copper. Plant Dis. 67:779-781.
18. McCarter, S. M., Barksdale, T. H., and Jaworski, C. A. 1971. Reduction of bacterial wilt by early harvest of tomato transplants. Phytopathology 61:849-851.
19. McCarter, S. M., Dukes, P. D., and Jaworski, C. A. 1969. Vertical distribution of *Pseudomonas solanacearum* in several soils. Phytopathology 59:1675-1677.
20. McCarter, S. M., Jones, J. B., Gitaitis, R. D., and Smitley, D. R. 1983. Survival of *Pseudomonas syringae* pv. *tomato* in association with tomato seed, soil, host tissue, and epiphytic weed hosts in Georgia. Phytopathology 73:1393-1398.
21. McGuire, R. G., Jones, J. B., and Sasser, M. 1986. Tween media for semiselective isolation of *Xanthomonas campestris* pv. *vesicatoria* from soil and plant material. Plant Dis. 70:887-891.
22. McInnes, T. B., Gitaitis, R. D., McCarter, S. M., Jaworski, C. A., and Phatak, S. C. 1988. Airborne dispersal of bacteria in tomato and pepper transplant fields. Plant Dis. 72:575-579.
23. Ratcliffe, T. J., and Morton, D. J. 1963. Diseases affecting production of certified tomato plants in Georgia during 1942-1963. Plant Dis. Rep. 47:1065-1066.
24. Sijam, K., Chang, C. J., and Gitaitis, R. D. 1991. An agar medium for the isolation and identification of *Xanthomonas campestris* pv. *vesicatoria* from seed. Phytopathology 81:831-834.
25. Stall, R. E., and Thayer, P. L. 1962. Streptomycin resistance of the bacterial spot pathogen and control with streptomycin. Plant Dis. Rep. 16:389-392.
26. Voloudakis, A. E., Gitaitis, R. D., Westbrook, J. K., Phatak, S. C., and McCarter, S. M. 1991. Epiphytic survival of *Pseudomonas syringae* pv. *syringae* and *P. s. tomato* on tomato transplants in southern Georgia. Plant Dis. 75:672-675.

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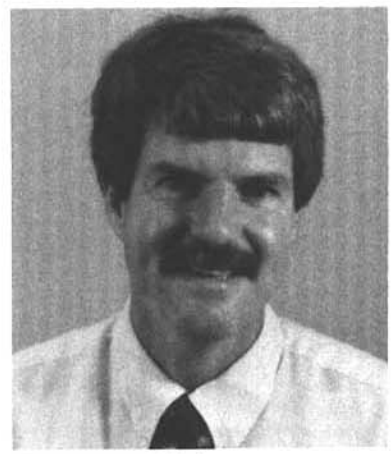

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