OCCURRENCE, EPIDEMIOLOGY, AND CONTROL OF BACTERIAL CANKER OF TOMATO IN SOUTHWESTERN ONTARIO

C.D. McKeen

Abstract

During the last decade bacterial canker caused by Corynebacterium michiganense has become a serious disease of greenhouse tomatoes in Essex County in southwestern Ontario. Canker caused losses in yield averaging 5–10% annually in 1965–71, with a few individual growers losing UP to 60% in a single crop. Perennation of the disease occurs locally. Intensive and continuous tomato cultivation in the Leamington area aids inoculum spread and together with delays in disease detection makes control of canker difficult.

Introduction

Although bacterial canker caused by Corynebacterium **michiganense** (E. F. Sm.) **H.** L. **Jens.** has been known to occur in the **U.S.A.** since 1909 (10), and has occurred with sporadic frequency in many of the tomato-growing areas of the world **(12), it** became serious in southwestern Ontario only during the 1960's. For the last 8 years (8) **it** has been a constant threat to the greenhouse tomato industry centered around Leamington, Ontario, and currently is one of the most dreaded and potentially devastating diseases. This is a report of its epidemiology and factors that render control difficult.

Observations

Occurrence of canker in greenhouse and field crops

In 1961, canker caused almost 100% infection of a 2-acre (0.8 ha) field of staked tomatoes, Lycopersicon esculentum L. CV. Trellis 22, near Leamington, Ontario. **By** mid-season of 1962, canker affected at least 30% of the plants in two staked crops, one being on the same farm that had the outbreak in 1961. In 1963, the disease affected two greenhouse crops, located on different farms from previous occurrences. One occurred in the spring and one in the fall, and although slightly less than 20% of the plants in both establishments became infected, the rapidity with which the disease spread along the rows was alarming. In 1964, canker was serious in two staked crops, one on the farm with the 1961 outbreak. Although not diagnosed with certainty, canker was also reported to have occurred in a few greenhouse fall crops.

Contribution from the Research Station, Agriculture Canada, Harrow, Ontario NOR 1G0

Layne and Rainforth (6) observed canker in 21 separate greenhouse plantings in the fall crop of 1965. This involved slightly more than **25** acres (10 ha). They also reported a new systemic fruit symptom of canker. Recognition of this symptom improved the detection of canker, and thereafter made possible more accurate records of its occurrence in greenhouse crops. The disease became more widespread in the 1966 greenhouse plantings. With concerted efforts by plant pathologists and provincial extension specialists at the Harrow Research Station who urged greenhouse growers to follow carefully the recommendations for controlling canker, lower incidences resulted in 1967 through 1969 than in 1965 and 1966. Nevertheless, canker occurred in approximately 20% of the greenhouse establishments in each of the 3 years. Generally, the numbers of infected plants were large in only one **or** two of the several greenhouses located at each site. In the 1970 crop, there was a substantial increase over the previous 3 years, both in the numbers of greenhouse crops affected and in the overall numbers of plants infected. As in previous years, canker was generally more prevalent in the fall than in the spring crop.

In 1967, Reyes et al. (9) reported canker occurring in **3** of 6 tomato fields examined in Essex County and in 1 of 4 in the adjacent county of Kent. In 1970, within a radius of 6 miles (10 km) of Leamington, I observed canker to be prevalent in 4 of **12** fields grown for the early-basket trade and in 17 of 36 fields of later-maturing crops grown for the processing industry. In 5 of 24 fields of processing tomatoes located farther from Leamington, in Essex County, canker was also found. No canker was observed in 10 field crops examined in Kent County in 1970.

Canker losses and related factors

In general, the disease has been much less serious in field than in greenhouse crops. In the latter, canker causes much

more severe symptoms in the early spring and the late autumn than during the summer. Under short day culture of low light intensity, systemically infected plants wilt severely and soon die. In contrast, during the summer months infected plants often show barely perceptible wilting of leaves. More commonly, localized tan to brownish, scorch-like necrotic areas (0.5 to 2 cm in diam) on the laminae are characteristic symptoms. In crops that become infected in late May or June, fruit symptoms are scarcely discernible.

Losses in the greenhouse crop have been variable from year to year and from greenhouse to greenhouse even at individual establishments. Losses suffered by individual growers have ranged from less than 5% to as much as 60%. In 1966, most of the 20% reduction in gross yield from the fall crop was attributed to losses from canker. The overall annual yield losses in greenhouse production in the Leamington area from 1965 to 1971 have been estimated to range from 5% to 10%.

As reported by Kendrick and Walker (5), the succulence of the crop markedly determines the severity of infection. Because of high soil fertility resulting from heavy fertilization of the spring cucumber crop, fall plantings of tomatoes following cucumbers almost invariably show greater disease severity than those following tomatoes.

The type of equipment used to irrigate crops affects symptoms, particularly those found on fruits. Where no splashing of the foliage or fruits occurs in greenhouse watering, the "birds-eye" spot is not found. In contrast, the frequent occurreqce of this diagnostic symptom in the field readily reveals infection. Birds-eye spot does not harm the quality of the processed fruit but renders those for the early-basket crop unmarketable and is thereby largely responsible for the losses sustained.

Localized i and s t invasion of greenhouse plants

The erratic spread of canker in the greenhouse crop has been extremely puzzling and has made the making of control recommendations difficult. Delays of 4-6 weeks and occasionally as long as 3 months between first outbreaks and secondary spread to adjacent plants have often been encountered. To study the cause of these delays an experiment was set up in March, 1970 with potted plants. Michigan-Ohio Hybrid plants 15 inches (37 cm) high growing in 5-inch (12.5 cm) pots of compost soil were used. Sap expressed from the brownish vascular areas of infected fruits and wilted stems was diluted with water in the ratio of 1 to 3 and then rubbed very lightly with the wetted forefinger on the surface of the stems in one series of 12 plants and on the leaves in another series of 12. In the first series inoculum was applied to one side of the stem along the first five internodes above the cotyledons, and in the second series to the upper surface of two leaflets on the third and fourth oldest leaves. Check plants were rubbed with water. Fertilizing schedules were established to maintain a low degree of plant succulence. Six weeks after inoculation, all plants were carefully repotted into 7-inch (17 cm) pots. Blister-like lesions, as reported by Layne (7) and Basu (1), developed in 7 to 10 days on the inoculated leaves. Ten to 20 days later, systemic infection occurred in all leaf-inoculated plants and caused typical unilateral wilting of leaves.

Where stems were inoculated, tan-colored pin-point lesions developed but were not usually discernible before 10 days, and in less succulent plants not before 12-14 days. Thereafter, the lesions enlarged slowly and many were only 1-2 mm in diam 25 days after inoculation. At that time they had whitish borders with tan centers. Lesions continued to enlarge slowly and in 6-8 weeks were 2.0-2.5 mm in diam. Some lesions had coalesced. All were rusty red, projected prominently above stem surfaces and had rough surfaces. After 10 weeks, only 3 of 12 plants had developed systemic symptoms. The lesions continued to increase in size and many had coalesced. Because of excessive sucker production and the care required in the subsequent handling of these potted plants, further observations on systemic invasion were not continued, although the plants were held for 5 more months to determine the longevity of bacteria in the local lesions.

Tissue fragments dislodged from discrete lesions by a superficial scratching with the tip of a scalpel blade were taken at fortnightly intervals after lesion initiation to determine the viability and pathogenicity of the contained bacteria. Microscopic examinations showed that all scrapings contained an abundance of viable bacteria. Concomitantly, when such tissue fragments were inserted into scalpel wounds in young tomato stems, vascular infections typical of canker developed. Eight-month-old lesions proved to be a good source of viable, pathogenic bacteria. Delays in disease spread appear therefore to be associated with slow development of systemic stem lesions and low rates of stem infection in well-hardened plants.

Perennation of canker in Essex County

Extensive laboratory and greenhouse experiments carried out from 1965 through 1968 established that seedborne inoculum was not the source of canker each year in tomato crops of Essex County. Also, critical examinations of many field crops set with transplants imported from Georgia, U.S.A., revealed that these transplants were not the source of infection. By 1969, circumstantial evidence clearly established that the disease was becoming endemic to the area. In several

instances in 1970, field infections were traced to transmission resulting from a previous limited handling of infected greenhouse plants, and similarly infection of greenhouse crops sometimes resulted from the handling of infected field-grown plants. I also traced infection in several processing crops to locally grown infected transplants.

Problems in controlling canker in Essex County

The concentration of 275 acres (110 ha) of steam-heated glass and polyethylene-covered houses, as well as several acres of unheated structures used during the spring for the growing of transplants, means that within an area of about 36 *sq* miles (93.6 km^2) at Leamington, tomatoes are being grown the year round. In addition, more than 1200 acres (486 ha) for the early-basket trade and about one-tenth of the 7600 acres (3420 ha) in Essex County for the processing industry are grown within the same area. To eradicate canker requires a combined effort by all the individual greenhouse growers as well as all others involved in tomato production and processing. Infection of field-grown tomatoes constitutes a potential source of inoculum for the fall greenhouse crop. As well as transmission by laborers working intermittently in both field and greenhouse crops, there is good circumstantial evidence that windblown inoculum from infected field crops may have been partly responsible for the epidemics in the 1966 and 1970 fall greenhouse crops. In both years a heavy rainstorm accompanied by winds of high velocity occurred when seedlings for the fall crop were half-grown, and sand and bits of plant debris from field crops were blown into the greenhouses.

When canker occurred in the fall greenhouse planting, it usually over-wintered and caused at least a trace of infection in the following spring crop, even where careful sanitation, including steam sterilization of planting containers and soils, had been employed. Bryan and Boyd (3), and Grogan and Kendrick (4) reported that canker bacteria overwintered in tomato debris in the field and in plantbed soil in Georgia and California, respectively. Basu (2) reported that in the absence of host debris the causal bacteria do not survive for more than 3-4 weeks in an unsterilized compost soil at 25 C. The almost continuous tomato production in the Leamington area does not require that canker bacteria survive saprophytically for more than a few days or weeks to serve as a potential inoculum source.

The capacity of canker bacteria to exist superficially on tomato stems at almost "sub-clinical" levels militates against canker control. The difficulty of recognizing canker in tomato plants growing under "hard" conditions makes detection of the disease very difficult. Also, incipient or mild symptoms on the foliage may easily be overlooked because of the similarity to injury occasionally caused by excess fertilization or by applied pesticides. Delay in detection of canker in the greenhouse crop, where regular handling of the plants in cultural operations is required, has often resulted in extensive and rapid spread of the disease. The smoldering aspect of canker presents a continuing threat to the grower, and the additional daily examinations and extra operations required to detect and prevent explosive secondary spread add significantly to production costs.

The practice of applying sprays of fixed copper at weekly intervals, especially to seedlings, transplants, and young greenhouse plants, has afforded a measure of canker control. However, it has been established that strict adherence to proper sanitation must accompany chemical use.

Acknowledgments

The author gratefully acknowledges the assistance of J. R. Rainforth, D. George, and J. C. Fisher, greenhouse vegetable extension specialists with the Ontario Ministry of Agriculture and Food, who informed the author periodically about infected greenhouse crops from 1965 through 1972. I also thank Harry Thorpe of this laboratory for technical assistance with greenhouse experiments and laboratory diagnoses.

Literature cited

1. Basu, P. K. 1966. Conditions for symptomological differentiation of bacterial canker, spot and speck on tomato seedlings. Can. J. Plant Sci. 46:525-530.

2. Basu, P. K. 1970. Temperature, an important factor determining survival of Corynebacterium michiganense in soil. Phytopathology 60:825-827.

3. Bryan, Mary K., and O. C. Boyd. 1930. Control of bacterial canker of tomatoes. Phytopathology 20:127. (Abstr.)

4. Grogan, R. G., and J. B. Kendrick. 1953. Seed transmission, mode of overwintering and spread of bacterial canker, caused by Corynebacterium michiganense. Phytopathology 43:374. (Abstr.)

5. Kendrick, J. B. Jr., and J. C. Walker. 1948. Predisposition of tomato to bacterial canker. J. Agr. Res. 77:169-186.

6. Layne, R. E. C., and J. R. Rainforth. 1966. A new symptom of bacterial canker resulting from systemic infection of tomato fruits and its

implications in dissemination and seed transmission. can. J. Plant Sci. 46:371-374.

7. Layne, R. E. C. 1967. Foliar trichomes and their importance as infection sites €or Corynebacterium michiganense on tomato. Phytopathology 57:981-985.

8. McKeen, C. D. 1972. Tomato diseases. Can. Dep. Agr. Publ. 1479 p. 33-34.

9. Reyes, A. A., J. R. Chard, A. Hikichi, W. E. Kayler, K. L. Priest, J. R. Rainforth, I. D. Smith, and W. A. Willows. 1968. A survey of diseases of vegetable crops in southern Ontario in 1967. Can. Plant Dis. Surv. 48:20-24.

10. Smith, E. F. 1910. A new tomato disease of economic importance. Science (N.S.) 31:794-796. (Abstr.)

11. Smith, W. P. C., and Olqa M. Goss. 1946. Bacterial canker of tomatoes. J. Dep. Aqr. West. Australia 23:147-156.

12. Strider, D. L. 1369. Bacterial canker of tomato caused by Corynebacterium michiganense. North Carolina Agr. Exp. Sta. Tech. Bull. 193. 110 p.