

Integrated Approach to Controlling Leucostoma Canker of Peach in Ontario

Peach (*Prunus persica* (L.) Batsch) is the third most valuable fruit crop in Ontario, Canada, following apples (*Malus domestica* Borkh.) and grapes (*Vitis* spp.). In 1988, the 3,000 ha of peach orchards produced 36,000 t of fruit with a value of approximately \$18 million (Canadian). Because of Ontario's northern location, most people are surprised to learn that peaches are grown there. The climate of the Niagara fruit-growing region, about 43 degrees north latitude, is more favorable for tender fruit production than most other parts of eastern North America because air temperature is influenced by a large, deep body of water (Lake Ontario) and a moderately sloped, adjacent raised land shelf (Niagara Escarpment) (34). Crop losses due to winter cold or spring frost are more frequent in the neighboring United States as far south as Georgia. The combination of land form and water results in large and continuous heat transfer from Lake Ontario to the fruit lands whenever the lake is warmer than the land and when winds are light or absent. This "escarpment effect" is important both day and night during December, January, and February and at night during March, April, and May. When the air is calm in spring and early summer, the cold lake and onshore breezes act to cool the land mass and delay tree development, thus reducing the probability of spring frost damage.

The Leucostoma Canker Problem

Leucostoma canker, also called perennial canker, *Cytospora* canker, and Valsa

canker, is caused by two closely related fungi, *Leucostoma cincta* (Pers. & Fr.) Höhn. (anamorph = *Cytospora cincta* (Pers.) Fr.) and *L. persoonii* (Nits.) Höhn. (anamorph = *C. leucostoma* (Pers.) Fr.). In Ontario, both pathogens infect peach trees, but in other locations only one of the two pathogens may be present. *L. persoonii* generally prefers warmer climates, whereas *L. cincta* occurs in cooler areas. Although found primarily on peach in Ontario, these fungi can cause cankers and twig dieback on plum and prune (*P. domestica* L.), sweet and sour cherry (*P. avium* L. and *P. cerasus* L., respectively), apricot (*P. armeniaca* L.), wild black cherries (*P. serotina* Ehrh.), choke cherries (*P. virginiana* L.), ornamental cherries, and apple, among other mainly rosaceous hosts. Both fungi are widespread in North America, and the disease is prevalent in northern portions of the region favorable for production of temperate fruits.

Leucostoma canker limits peach production in West Virginia, New Jersey, Pennsylvania, New York, Illinois, Michigan, Colorado, Idaho, Ontario, and British Columbia and also occurs in Europe, Japan, and South America. The disease is recognized as the most serious disorder of peach trees in the Niagara Peninsula.

Economic Impact

The most recent survey of the disease in Ontario (21) determined that 98% of the 2,000 trees examined were infected. One-half of the trees had cankers on trunks and 90% had cankers on scaffold limbs. The disease affected 30% of the tree trunk and branch circumference of

the sampled trees. Infection severely debilitates trees, and in a 1-year period, 9% of the trees were removed and 10% of the bearing surface of those remaining was cut and destroyed. The mean orchard life span in Ontario is about 10 years, of which 6–8 years are in full production of 15.7 t/ha valued at about \$14,000. If peach orchards having enhanced partial resistance to *Leucostoma* spp. are planted, or if the partial resistance now available is used with properly timed fungicide applications, the life span of the orchard might increase by 5 years and thereby expand the period of full production to 11–13 years. If that objective is achieved, current Ontario peach production levels could be sustained by reducing the number of trees planted by 50%. Annual savings in orchard establishment costs (planting 150 vs. 300 ha per year) would be more than \$2.5 million per year. If the productive life of orchards is increased by 5 years, gross income from productive orchards would increase by about \$68,000/ha.

History of the Disease

The first observations of peach tree cankers caused by *Leucostoma* spp. were made in 1900 (31) in western New York and a few years later in Missouri (28). The first report from Ontario was in 1912 (18). In a series of studies from 1912 to 1918, McCubbin (24) described the uniform occurrence of the disease in southern Ontario, established the spring-and-fall pattern of canker activity, and compared the perennial infections caused by *Leucostoma* spp. with the relatively short-lived branch infections caused by the brown rot fungus, *Monilinia*

fruticola (Wint.) Honey. During the 1930s, Willison published a series of papers that more clearly defined the etiology of the disease and the factors contributing to its occurrence and recommended control practices that continue to form the basis for management of this disease in the 1980s (35–37).

Symptoms and Etiology

The appearance of peach canker varies according to the part of the tree infected. Infections of small twigs appear as sunken, discolored areas near winter-killed buds or leaf scars (Fig. 1). Alternating concentric zones of different colors, which represent failed attempts at wound periderm formation by the host, often are visible in the bark (Fig. 2) and on the surface of the lesion (Fig. 3). Nodal infections are visible in late May or early June. The infected tissues darken with time, and amber-colored gum may ooze from them (Fig. 4) unless the twig is entirely killed. One-year-old shoots that develop in the center of the tree are quite susceptible to nodal infections, and if left untreated, infections from these shoots rapidly invade scaffold limbs and large branches. Branch cankers that result have dead twigs or twig stubs at their centers.

Cankers that form on the main trunk, branch crotches, scaffold limbs, and older branches are conspicuous symptoms that begin with exudation of a copious quantity of amber-colored gum. Gum production is a natural

response of peach trees to any irritation, but that due to *Leucostoma* infection is excessive to the point of being detrimental. As cankers age, the gum becomes dark brown to black, the infected bark dries out, and cracks open, exposing blackened tissue beneath elliptical cankers along the length of the stem (Fig. 5). *L. persoonii* is isolated from large cankers more often than *L. cincta*, which is found more frequently in infected nodes (32).

Beginning in late spring and continuing through the summer, trees grow rapidly and resist further penetration of the fungus into healthy tissues. Callus rings form around cankers during this time, but the pathogen overcomes this defense in late fall or early spring when the tree is dormant. The yearly alternation of callus formation and canker extension produces a canker with concentric callus rings. Callus rings may be indistinct and the canker more diffuse in appearance if the plant's defense responses are compromised by external conditions.

Extension of branch or twig infections may result later in leaves that turn yellow, droop, and eventually wilt and die. Dead twigs and branches usually are covered with a multitude of pinhead-sized black pycnidia erupting through the bark (Fig. 6A). Conidia are extruded in a polysaccharide matrix called a cirrus (Fig. 6B). In Ontario, pycnidia of both *Leucostoma* spp. are found continually, and perithecia of *L. persoonii* are

common in late fall, throughout the winter, and into spring. An individual pycnidium of *Leucostoma* spp. may produce up to 10,000 spores (32). Conidia initiate most new infections. The role of ascospores in the disease cycle has not been determined.

Epidemiology and Environment

Most conidia of *Leucostoma* spp. are disseminated by splashing rain; boring insects, birds, and pruning tools serve as other routes for spore dissemination (13,23,35). Conidia of *L. persoonii* may travel up to 76 m from the source (1). The dispersal gradients of *Leucostoma* spores depend on wind velocity during rain. In California, conidia may be recovered from the bark surface at almost any time of the year (2)—an expression of either frequent extrusion or considerable longevity of *Leucostoma* conidia. The former is the more likely explanation because conidia of *L. cincta* die within 6 hours when released from



Fig. 1. Nodal infections caused by *Leucostoma cincta* on 1-year-old peach shoot.



Fig. 3. Nodal lesions on 1-year-old peach shoot. Note alternating zones of varying color on the distal portion of the shoot.

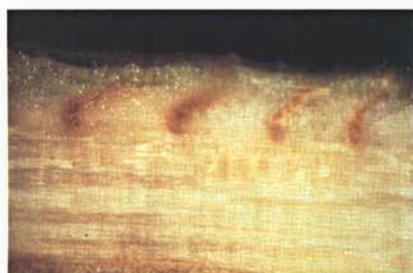


Fig. 2. Alternating zonation lines in 1-year-old peach bark inoculated with *Leucostoma persoonii*.



Fig. 4. Gum exudation at a branch lesion initiated from an infected twig.



Fig. 5. Conspicuous perennial canker characteristic of *Leucostoma* spp. on a peach scaffold limb.



Fig. 6. (A) Dead twig infected by *Leucostoma persoonii* showing a multitude of black pycnidia. (B) Closer view showing a single *Leucostoma* pycnidium extruding spores in a cirrus.

their polysaccharide matrix, become wet, and then are allowed to dry (32). Schulz and Schmidle (30) observed that both *Leucostoma* spp. sporulate all year long but produce the most spores from November through March. Spores also were numerous in June, if rainfall was sufficient. The number of spores trapped depended on the number of hours between 10 and 15 C (optimum for spore production), the duration of wetness, and the duration of relative humidity between 90 and 100%. Leupschen and Rohrbach (23) also trapped spores of *L. persoonii* throughout the year, but they found the most spores during summer months. In contrast, Jones and Leupschen (22) obtained the highest spore counts in September and October.

Cardinal temperatures for spore germination and growth of *L. cincta* are lower than those for *L. persoonii*, which grows best at 25–30 C and with a maximum of 32 C (15,27). *L. cincta* grows at temperatures as low as 4 C, grows optimally at 18–20 C, and usually will not grow at temperatures above 30 C. Ability to grow at 37 C can be used to differentiate the two species in culture (G. C. Adams, personal communication). Spore germination requires a carbon source and free water or 100% relative humidity (15).

When Schulz and Schmidle (30) studied the influence of temperature on colonization of peach by *L. persoonii*, necrosis was apparent within 3 days after inoculation at 14–20 C; enlargement of cankers was limited by the host, however. At 2–8 C, colonization was slower, but with time, the necrotic area was larger than at the higher temperatures. Schulz and Schmidle (30) concluded that the number of days with temperatures less than 10 C is related to smaller areas of initial necrosis but eventually larger areas of tissue colonization. These observations probably are related to the influence of temperature on host resistance mechanisms.

Several environmental factors influence susceptibility of stone fruits to infection by *Leucostoma* spp. Cold temperature injury increases disease severity (16,19,29,35), especially on the southwest side of the tree where alternate freezing and thawing of bark tissues are influenced by the large temperature differential between the bark and the ambient air during winter. More cankers appear on the southwest side than on other locations on the tree.

Many factors influence the rate of tissue maturation and, therefore, the susceptibility of stone fruit and other fruit trees to cold injury. Orchard management practices to be avoided are excessive fertilizing and watering and late-season cultivation.

Soil physical properties (3) and ring and pin nematodes (17) also are associated with *Leucostoma* infections of stone fruits. A high incidence of

Leucostoma canker is associated with French prune dieback in California. In areas where prune dieback is severe, soils have high clay content and poor availability of potassium (3). Prune trees that were subjected to postharvest moisture stress developed larger cankers after inoculation than nonstressed trees (4). Cankers were less frequent in trees grown with trickle irrigation applied to maintain soil moisture status above –30 kPa than in nonirrigated trees (14).

The Infection Court

The *Leucostoma* species that attack peach and other stone fruits are considered weak parasites because they cannot invade healthy, intact bark. Once inside the tree, however, they can colonize aggressively. Cankers usually are initiated in weak, dying, or dead tissue or at open wounds. The most common infection sites are pruning cuts, leaf scars, shade-weakened twigs inside canopies, insect injuries, brown rot cankers, and winter-injured buds, twigs, and bark. Rodent injuries and wounds resulting from cultivation, picking ladders, wire mouse guards, and broken limbs also may become infected. Short branch stubs, remaining from improper pruning, that do not heal provide an ideal gradient of dying tissue that is readily colonized by *Leucostoma*.

Many orchard surveys have identified pruning cuts as one of the most common infection courts for initiation of *Leucostoma* canker. Infections at pruning cuts are potentially devastating because they often are located at points critical to the architecture of the tree. The loss of one major scaffold limb, for example, represents about one-fourth to one-third of the bearing surface. Therefore, the major focus of our research has been to study wound response in peach and the relationship between wound response and disease resistance.

Wound Response and Resistance to Colonization

When an infection court is created, as when a tree is pruned, disease is most frequent and most severe when the inoculum arrives at the infection court immediately. Disease frequency and severity decline with time, until the wounded tissues express resistance comparable to that of noninjured bark (8,9). The length of this period depends on many factors. We have identified temperature, soil moisture, and peach genotype as a few of the factors that influence postwounding events and therefore the host-pathogen interaction.

Creation of a wound triggers a sequence of biochemical processes that lead ultimately to the generation of new bark (5,6). Histological studies showed that wounds resistant to inoculations with *L. persoonii* possessed a minimum of three phellem cells in the new periderm

(8) (Fig. 7). At earlier stages, wounds were susceptible to fungus invasion, although severity of the disease declined steadily beginning about 3 days after wounding prior to generation of new tissues. Periderm generation is probably only one of many possible types of resistance in peach to *Leucostoma* spp. and is best described as a type of rate-limiting or partial resistance.

Because new periderm cannot be penetrated readily by *Leucostoma* spp. unless the fungus is already well established in host tissue (39), we tested regeneration of periderm in wounds made in trunks of Redhaven peach beginning in May and continuing at 3-week intervals until August. Experiments also utilized sweet cherry and apple bark, which express moderate and high levels of resistance to *Leucostoma* spp., respectively. Orchard-grown, standard 19-year-old Red Delicious apple trees, 18-year-old sweet cherry trees, and 5-year-old Redhaven peach trees located at Jordan Station, Ontario, were used. Seven wounds per tree, about 2 mm deep into living tissues, were inflicted with a sharpened 4-mm-diameter cork borer. Apple and sweet cherry trees were wounded on the main scaffold limbs and peach trees, on the main trunk. Bark tissues surrounding the wounds were excised with a larger diameter cork borer 3, 7, 10, 14, 17, 21, and 28 days after wounding, prepared for histological examination, and examined microscopically for deposition of lignin and suberin in the new tissues produced in response to wounding. Regression analyses were used to investigate the relationship between wound responses and time post wounding, mean daily temperature during the postwounding period, and accumulated degree days.

Wound healing was most rapid in mid-July (7). The accumulation of degree-day units (base = 0 C) after wounding provided the best models for describing the observations. The models to describe lignin and suberin formation around peach bark wounds indicated that 256 and 411 degree-days were required for

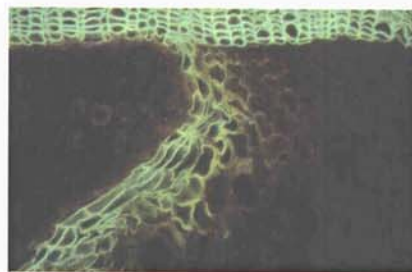


Fig. 7. Transverse section of peach bark tissue at 10 days after wounding showing the approximate degree of wound periderm formation associated with resistance of wounds to inoculation with *Leucostoma persoonii*.

Table 1. Estimates of number of days for wound responses of peach to be completed after bark injuries in March, April, May, and June at Vineland Station, Ontario*

Injury date	Estimated number of days required for:	
	Lignification	Suberization
15 March	49	62
31 March	36	49
15 April	27	39
30 April	22	32
15 May	18	27
31 May	15	23
15 June	13	21

*Based on mean daily heat unit accumulation (base = 0 C) from 1935 to 1985.

complete lignin and suberin deposition, respectively, after all dates of wounding. Lignin and suberin deposition proceeded faster in sweet cherry than in peach, and wound healing proceeded faster in apple than in sweet cherry.

Weather data from Vineland for the past 50 years were compiled and used to calculate the mean seasonal accumulation of heat units (base = 0 C). From these data, guidelines were formulated for growers who elect to prune at different times in the spring (Table 1). For example, pruning on 15 March would require 62 days for effective wound healing based on an estimated accumulation of 411 degree-days, whereas pruning on 15 May would require 27 days to accumulate 411 degree-days. It is important to realize that these estimates of wound response time are useful only as guidelines and that wound healing varies greatly with the age of the tree, diameter of the wound, age and location of the wounded tissue, and numerous other site and environmental factors.

The long periods of time required for wounds to develop resistance to wound pathogens, relative to the 5- to 14-day protective activity of most orchard fungicides, may explain why single-spray treatments have been ineffective for control of *Leucostoma* canker (20). Preliminary control strategies incorporating the use of fungicides at various intervals after pruning peach trees at different times of the year are being tested at the Horticultural Research Institute of Ontario. Implementation of a spray schedule for peach canker control for growers who prune on 15 March might require an interval of approximately 9 weeks for protection, compared with 4 weeks for those who pruned on 15 May. Currently, we recommend that pruning be delayed as long as possible in order to decrease the duration of wound susceptibility. Ideally, we would like to see the pruning operation finishing just

Table 2. Ten-point program for integrated control of *Leucostoma* canker in Ontario

1. Control insects and diseases such as oriental fruit moth, peach tree borer, and brown rot
2. Drain water away from bases of trees
3. Train trees to promote wide branch crotch angles
4. Prevent rodent injury with wire or plastic guards and thiram-latex paint
5. Prevent cold injury by avoiding practices that delay dormancy; paint tree trunks to prevent injury on southwest side
6. Prune correctly and at proper time to promote optimum wound healing
7. Surgically remove cankers on trunks and scaffolds in June or July
8. Paint pruning cuts with thiram-latex paint
9. Control leaf scar infections with fall or spring sprays for peach leaf curl
10. Apply nitrogen fertilizer before mid-April and balance it with adequate supply of potassium

as the initial sprays for brown rot blossom blight are being applied.

Soil moisture influences wound periderm formation in peach bark. In experiments to test the influence of irrigation during spring and early summer, periderm formed more slowly in nonirrigated plots than in irrigated plots of Candor peach maintained above -30 kPa with a trickle system (11) but not in plots of Redhaven peach, which is generally less susceptible than Candor to *Leucostoma* spp. The relative ability of peach cultivars to withstand water deficits and the mechanisms of water stress that influence resistance in peach to *Leucostoma* spp. have not been studied thoroughly.

Breeding for Disease Resistance

The best long-term strategy for control of peach canker is to develop cultivars with disease resistance. Constraints to implement this strategy include: 1) the land-, time-, and labor-intensive nature of conventional tree fruit breeding, 2) regional variation in cultivar performance, 3) the cultivar \times year interaction observed with most inoculation studies, 4) the lack of simple and reliable procedures for applying quantitative inoculum, 5) the lack of known resistant cultivars, and 6) the lack of knowledge regarding pathogen variation.

In 1984, we studied the susceptibility of peach cultivars relative to the events that take place in peach bark after wounding in the absence of the pathogen. In these experiments, the rate of suberin accumulation in wounds was negatively correlated with relative susceptibility of peach cultivars to *Leucostoma* spp., but only when wounds were inflicted during the months of May and June. The relative amount of suberin also was more important than the degree of new periderm development for predicting relative susceptibility (12). These findings may accelerate the selection of peach genotypes with increased partial resistance to *Leucostoma* spp. Efforts are under way to simplify the laborious histological procedures that are used to obtain suberin measurements.

Trials have been implemented to determine the heritability of wound response characters (25). During 1985, reciprocal crosses were made among V-68101 (the cultivar in our trials that accumulated suberin most rapidly) and V-68051 and NJC-95 (the cultivars that accumulated suberin less rapidly than V-68101). The seedlings from these crosses were planted the following year at two locations. Seedlings from crosses between V-68101 and several freestone cultivars with different degrees of susceptibility to the disease were included. After 1 year of study, resistance of V-68101 to canker pathogens appeared to be transmitted to the progenies, especially when V-68101 was used as the female parent. Continued field observations reinforced by laboratory suberin measurements are needed to complete this evaluation, however.

Management in Ontario

Control of *Leucostoma* spp. must take place within an integrated crop management strategy. All phases of orchard management—from the establishment of new plantings to the care of bearing orchards—must be considered. Before planting, site selection for good air and surface water drainage and distance from heavily diseased areas is important. Young trees should not be planted adjacent to older, heavily infected peach blocks, and the downwind side of older blocks should be avoided. Interplanting young trees with older diseased trees may seem to be economical, but interplanted young trees are at a much greater risk for developing *Leucostoma* canker and have a shorter productive life than young trees planted in solid blocks.

Cultivar selection for specific geographic locations is important to ensure that only the hardiest and best cultivars are planted. Nursery stock should be free from pathogens, including viruses. To minimize the stress associated with transplanting, new trees should not be excessively large. Trees should be inspected carefully after growth begins, and any dead branches should be removed.

Recommendations for care of established orchards (Table 2) are based on preventive measures designed to decrease winter injury and damage by other pathogens and insects, promote optimum plant health, and take advantage of the levels of partial disease resistance now available. For optimum control of *Leucostoma* canker, all the following practices should be observed:

1. Control insects and diseases. Oriental fruit moth, peach tree borer, and lesser peach tree borer should be controlled even in the first few nonbearing years. Control of brown rot is also important because twig infections by *M. fructicola* are often invaded and enlarged by *Leucostoma*. When blossom sprays are integrated with pruning to help control pruning-cut infections by *Leucostoma* spp., brown rot mummies should be removed so they are not

present in the tree during the next year's bloom period.

2. Protect the bases of trees. Water should be drained away from tree trunks to prevent direct cold-temperature injury to the crown. This is achieved in Ontario by mounding soil around the bases of trees in autumn.

3. Train trees properly. Trees should be trained to develop wide branch crotch angles. These are absolutely necessary for long orchard life because tissues in narrow crotch angles are more susceptible to winter injury and invasion by borers. Strong crotch angles also reduce the likelihood of splitting when trees are bearing a heavy crop.

4. Avoid rodent injury. Rabbit and mouse damage should be prevented with wire or plastic guards; these should not be so high that lower scaffold limbs are wounded when the tree sways in heavy winds. White latex paint mixed with thiram also discourages rodent feeding.

5. Prevent cold injury. Excessive fertilization with nitrogen, late application of nitrogen, or late-season cultivation and/or irrigation should be avoided. In clean cultivation management systems, cultivation should cease and a cover crop be sown no later than mid-July. Sod management with trickle irrigation, besides maintaining tree growth and increasing fruit size, increases resistance to *Leucostoma*. Southwest injury can be prevented by painting trunks and scaffolds with white latex paint.

6. Prune correctly and at the proper time. Pruning to leave the branch bark ridge has reduced the level of infection by as much as 50% when the pruning cuts were examined after 1 year (10,38). Because wound healing is temperature-dependent, pruning should be delayed until warm, dry weather begins at or around the time of budbreak. Dry weather is important during the pruning period.

7. Surgically remove cankers from trunks and scaffold limbs. This should be done during dry weather in June or July, when wound healing is fastest.

8. Cover pruning cuts with a thiram-latex paint mixture. Although evidence regarding the use and effectiveness of wound paints and sealers is conflicting, and although they are probably not required on limbs recently subjected to canker surgery during the summer (33), many growers in Ontario who prefer to prune early find that the thiram-latex paint mixture provides some degree of protection against fungal infection.

9. Use chemicals to control peach leaf curl and thus leaf scar infections, caused by *L. cincta* and thought to occur as the tree defoliates in autumn or as the second abscission layer forms in the spring. Fall or spring sprays to control peach leaf curl, caused by *Taphrina deformans* (Berk.) Tulasne, will reduce the incidence of leaf scar infections (26). Integration

of pruning with early-season brown rot control may also help to reduce the incidence of pruning cut infections.

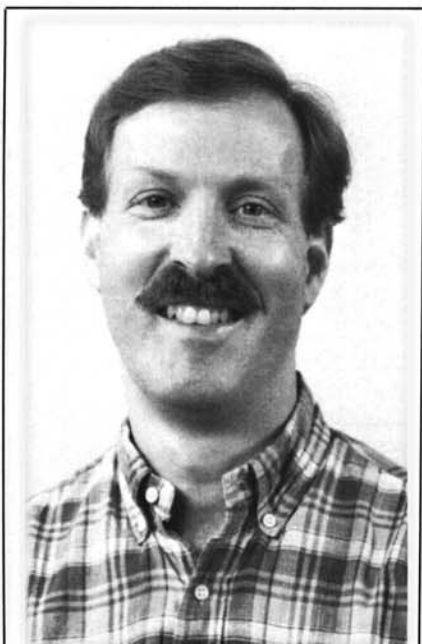
10. Apply nitrogen fertilizer in late winter or early spring (before mid-April in Ontario) to avoid inducing late, cold-susceptible growth in the fall. Balance nitrogen fertilizer application with an adequate supply of potassium. Use leaf and soil analyses to determine fertilizer requirements.

This orchard management system should keep most peach plantings free from *Leucostoma* infections for the first several years. These years are critical because young trees must become established and properly formed for optimum production. Good disease management results in extended orchard life, lower replant costs, and greater yields over the long term.

There are opportunities to improve control of *Leucostoma* canker with new cultivars having partial resistance and with fungicides applied during the time when pruning wounds are susceptible to infection. In the more distant future, development of new pathogen-resistant peach cultivars through biotechnology may be possible. We have established microembryo callus cultures of peach tissues to capitalize on the potential of somaclonal variation. Plants obtained in this manner currently are being tested in the field for increased pathogen resistance. We have isolated a low-molecular-weight protein from *L. persoonii* that causes necrosis in the bark of excised peach shoots. Introduction of this toxin to select plant material with increased resistance to the peach canker pathogens may improve selection for resistance. Improving disease management practices and research on peach breeding through the use of biotechnology hold promise for controlling peach canker in Ontario.

Literature Cited

1. Bertrand, P. F., and English, H. 1976. Release and dispersal of conidia and ascospores of *Valsa leucostoma*. *Phytopathology* 66:987-991.
2. Bertrand, P. F., and English, H. 1976. Virulence and seasonal activity of *Cytospora leucostoma* and *C. cincta* in French prune trees in California. *Plant Dis. Rep.* 60:106-110.
3. Bertrand, P. F., English, H., and Carlson, R. M. 1976. Relation of soil physical and fertility properties to the occurrence of *Cytospora* canker in French prune orchards. *Phytopathology* 66:1321-1324.
4. Bertrand, P. F., English, H., Uriu, K., and Schik, F. J. 1976. Late season water deficits and development of *Cytospora* canker in French prune. *Phytopathology* 66:1318-1320.
5. Biggs, A. R. 1984. Boundary zone formation in peach bark in response to wounds and *Cytospora leucostoma* infection. *Can. J. Bot.* 62:2814-2821.
6. Biggs, A. R. 1986. Comparative anatomy and host response of two peach cultivars



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- inoculated with *Leucostoma cincta* and *L. personii*. Phytopathology 76:905-912.
7. Biggs, A. R. 1986. Prediction of lignin and suberin deposition in boundary zone tissues of wounded tree bark using accumulated degree days. J. Am. Soc. Hortic. Sci. 111:757-760.
8. Biggs, A. R. 1986. Wound age and infection of peach bark by *Cytospora leucostoma*. Can. J. Bot. 64:2319-2321.
9. Biggs, A. R. 1989. Temporal changes in the infection court after wounding of peach bark and their association with cultivar variation in infection by *Leucostoma personii*. Phytopathology 79:627-630.
10. Biggs, A. R. 1989. Effect of pruning technique on *Leucostoma* infection and callus formation over wounds in peach trees. Plant Dis. 73:771-773.
11. Biggs, A. R., and Cline, R. A. 1986. Influence of irrigation on wound response in peach bark. Can. J. Plant Pathol. 8:405-408.
12. Biggs, A. R., and Miles, N. W. 1988. Association of suberin formation in uninoculated wounds with susceptibility to *Leucostoma cincta* and *L. personii*. Phytopathology 78:1070-1074.
13. Chiarappa, L. 1960. Distribution and a mode of spread of *Cytospora* canker in an orchard of the President plum variety in California. Plant Dis. Rep. 44:612-616.
14. Cline, R. A. 1984. Soil management and irrigation effects on peach canker in the Niagara Peninsula of Ontario. Pages 71-76 in: Proc. Stone Fruit Decline Workshop.
15. Dhanvantari, B. N. 1969. Comparative physiology and pathogenicity of *Leucostoma* spp. on peach. (Abstr.) Phytopathology 59:1023.
16. Dhanvantari, B. N. 1978. Cold predisposition of dormant peach twigs to nodal cankers caused by *Leucostoma* spp. Phytopathology 68:1779-1783.
17. English, H., Lownsbery, B. F., Schick, F. J., and Burlando, T. 1982. Effect of ring and pin nematodes on the development of bacterial canker and *Cytospora* canker in young French prune trees. Plant Dis. 66:114-116.
18. Gussow, H. T. 1912. A new disease of peaches. Page 251 in: Can. Exp. Farm Rep. 1910-1911.
19. Helton, A. W. 1961. Low temperature injury as a contributing factor in *Cytospora* invasion of plum trees. Plant Dis. Rep. 45:591-597.
20. Hickey, K. D., and Travis, J. W. 1984. Effect of preventative and post-infection fungicide treatments and the time of pruning on incidence of *Cytospora* infection on pruning wounds of peach. Pages 65-70 in: Proc. Stone Fruit Decline Workshop.
21. James, W. C., and Davidson, T. R. 1971. Survey of peach canker in the Niagara Peninsula during 1969 and 1970. Can. Plant Dis. Surv. 51:148-153.
22. Jones, A. C., and Leupschen, N. S. 1971. Seasonal development of *Cytospora* canker on peach in Colorado. Plant Dis. Rep. 55:314-317.
23. Leupschen, N. S. and Rohrbach, K. G. 1969. *Cytospora* canker of peach trees: Spore availability and wound susceptibility. Plant Dis. Rep. 53:869-872.
24. McCubbin, W. A. 1918. Peach canker. Can. Dep. Agric. Bull. 37:1-20.
25. Miles, N. W., Biggs, A. R., Svircev, A. M., and Chong, C. 1989. *Cytospora* canker resistance in peach: Germplasm evaluation and genetic improvement. Acta Hortic. In press.
26. Northover, J. 1976. Protection of peach shoots against species of *Leucostoma* with benomyl and captafol. Phytopathology 66:1125-1128.
27. Rohrbach, K. G., and Leupschen, N. S. 1968. Environmental and nutritional factors affecting pycnidiospore germination of *Cytospora leucostoma*. Phytopathology 58:1134-1138.
28. Rolfs, F. M. 1909. A disease of neglected peach trees. Mo. State Board Hortic. Annu. Rep. 2:278-283.
29. Royse, D. J., and Ries, S. M. 1978. Detection of *Cytospora* species in twig elements of peach and its relation to the incidence of perennial canker. Phytopathology 68:663-667.
30. Schulz, U., and Schmidle, A. 1983. Zur Epidemiologie der Valsa-Krankheit. Angew. Bot. 57:99-107.
31. Stewart, F. C., Rolfs, F. M., and Hall, F. H. 1900. A fruit disease survey of western New York in 1900. N.Y. Agric. Exp. Stn. (Geneva) Bull. 191:291-331.
32. Tekauz, A. 1972. The role of leaf scar and pruning cut infections in the etiology and epidemiology of peach canker caused by *Leucostoma* species. Ph.D. thesis. University of Toronto. 161 pages.
33. Travis, J. W., and Hickey, K. D. 1984. Efficacy of surgical removal and fungicide wound treatments on eradication of *Cytospora* canker on peach. Pages 60-64 in: Proc. Stone Fruit Decline Workshop.
34. Wiebe, J. 1976. What makes Niagara Peninsula weather unique? Can. Fruitgrower (March).
35. Willison, R. S. 1933. Peach canker investigations. I. Some notes on incidence, contributing factors, and control measures. Sci. Agric. 14:32-47.
36. Willison, R. S. 1936. Peach canker investigations. II. Infection studies. Can. J. Res. 14:27-44.
37. Willison, R. S. 1937. Peach canker investigations. III. Further notes on incidence, contributing factors, and related phenomena. Can. J. Res. 15:324-339.
38. Wilson, C. L., Miller, S. S., Otto, B. E., and Eldridge, B. J. 1984. Pruning technique affects dieback and *Cytospora* infection in peach trees. HortScience 19:251-253.
39. Wisniewski, M., Bogle, A. L., and Wilson, C. L. 1984. Histopathology of canker development on peach trees after inoculation with *Cytospora leucostoma*. Can. J. Bot. 62:2804-2813.