# Etiology of Canker and Dieback of Sweet Cherry Trees in Washington State

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**ABSTRACT**

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Canker and dieback were found in most sweet cherry orchards surveyed between 1985 and 1987 in eastern Washington. Cytospora canker was the principal canker disease, on the basis of disease symptoms and the recovery of *Cytospora cincta* and, infrequently, *C. leucostoma* from canker specimens. Pathogenicity tests showed that most isolates of *C. cincta* were highly virulent, colonizing over 75% of the stem circumference in 12 mo and causing cankers up to 11.5 cm long. Inoculation studies with *C. cincta* demonstrated that canker expansion was greatest from February to May; moderate expansion occurred during the summer, and little or no appreciable expansion in the autumn and winter months. In contrast, the bacterial canker pathogen, *Pseudomonas syringae* pv. *syringae*, was not isolated from cankers in orchards where disease was endemic, and inoculations in the spring (but not those in the fall) resulted in the formation of cankers, which usually remained active only until summer. It was concluded that *P. s.* pv. *syringae* and *C. cincta* do not coexist for extended periods in cankered tissues and that *C. cincta* is largely responsible for canker and dieback in sweet cherry orchards in Washington State.

Sweet cherry production has expanded extensively in eastern Washington during the last 20 years. Increasingly, there are reports of serious outbreaks of tree decline, including such symptoms as dieback, bark cankers, gummosis, and heartwood discoloration. Dieback begins in the apical branches and continues basipetally, eventually affecting the major scaffold limbs. Some orchards suffered winter freeze injury that initiated the decline in vigor, but others were commonly diagnosed to be afflicted with bacterial canker, caused by *Pseudomonas syringae* pv. *syringae* Van Hall. Despite reports that ice-nucleation-active strains of *P. s.* pv. *syringae* occur as epiphytes in many eastern Washington cherry orchards (12,13), stem cankers only rarely yielded the bacterial pathogen (D. C. Gross, *unpublished data*). This suggested three possible explanations, namely, that the bacterium survived only briefly in cankers, that it was supplanted by a secondary pathogen, such as *Cytospora*, or that another pathogen was initially the cause of infection.

Cytospora canker of *Prunus* spp., caused by either *C. cincta* Sacc. (teleomorph *Leucostoma cincta* (Fr.) Höhn.) or *C. leucostoma* (Pers.) Sacc. (teleomorph *L. persoonii* (Nits.) Höhn.), is common in many parts of the world. *C. leucostoma* is the predominant species isolated from cankers on plum trees grown in California (3), whereas *C. cincta* is reported to compose up to 98% of the strains isolated from peach trees grown in Ontario (21) and North Carolina (10). Cytospora canker is frequently misdiagnosed as bacterial canker, since many of the symptoms are indistinguishable and pycnidia may not readily form in infected bark tissue (10,19). An important distinguishing characteristic is that cankers caused by *Cytospora* spp. on peach and apricot trees are usually active for one or more years, whereas bacterial cankers are rarely active the following season (19,21). *P. s.* pv. *syringae* in cankers can sometimes be succeeded by *Cytospora* spp., however (10). Double infections with *Cytospora* and *P. s.* pv. *syringae* are apparently rare in nature; simultaneous inoculations with both pathogens usually result in larger cankers on peach and apricot trees (10,19). Oxalic acid production by *C. cincta* was shown by Endert-Kirkpatrick and Ritchie (10) to quickly acidify cankered tissues to levels inhibitory to the growth and viability of *P. s.* pv. *syringae*.

Cytospora canker is generally considered to be a serious problem on trees that are stressed or predisposed by winter injury rather than vigorously growing trees. For example, Kable et al (15) associated an unusually high incidence of Cytospora canker of sweet cherry in New York State with temperatures of the previous winter. Disease severity is also tempered by the varying levels of virulence of *Cytospora* strains. For example, Bertrand and English (3) reported that only 5 of 76 *Cytospora* isolates were highly virulent on vigorously growing French prune trees in California, suggesting that only weakened or stressed trees are prone to infection by *Cytospora* spp. (3,14).

The principal objective of this study was to identify the primary pathogen causing the cankers and dieback that have led to the widespread decline of sweet cherry trees in eastern Washington.

## MATERIALS AND METHODS

**Isolation and characterization of microorganisms from cherry cankers.** Branches with canker symptoms were collected from diseased sweet cherry (*Prunus avium* L.) trees in orchards in eastern Washington between July 1985 and August 1987. The samples were washed in cold running tap water for 5 min, and then the outer bark tissues were removed with a sterilized scalpel. Tissue segments, approximately 5 × 2.5 mm, were excised from the advancing margin of the infected bark or internal stem tissue. The excised segments were surface-disinfested by agitation for 30 sec in 0.5% NaOCl and then extensively rinsed in sterile distilled water (SDW). Fungi were isolated by placing the tissue segments on potato-dextrose agar (PDA) (Difco Laboratories, Detroit, MI), acidified PDA, or malt agar (MA) (Difco) and incubating at 23 ± 1 C for 10 days, to permit radial hyphal growth from the infected tissue. All fungal isolates were

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purified and subcultured on PDA prior to storage at 4 C.

*Cytospora* spp. were identified to genus according to Barnett and Hunter (1) on the basis of the characteristics of pycnidia formed on PDA after 30 days of incubation (24), the optimum temperature for growth on PDA after 6 days of incubation at 15, 20, 25, 30, and 36 C (3), and the presence of a zone of inhibition between pairs of strains after 16 days of incubation at 23 ± 1 C (14). Cultures of *C. leucostoma* (isolates f-7 and f-45) originally isolated from cankered plum trees in California were obtained from R. M. Bostock (University of California, Davis) for comparison to Washington isolates of *Cytospora*.

Fluorescent pseudomonads were isolated from infected tissue by soaking samples prepared as above in 5 ml of sterile potassium phosphate buffer (pH 7.1, 12.5 mM) with continuous rotary shaking (250 rpm) for 2 hr at 23 ± 1 C. Appropriate dilutions (0.1 ml) were plated on King's medium B agar (17); the plates were incubated at 25 C for 48 hr prior to identification of fluorescent pseudomonad colonies (12). Representative fluorescent colonies were purified to homogeneity and characterized with previously described key tests for identifying *P. syringae* (13). Bacteria were routinely cultivated and preserved by standard procedures (12,13). The highly virulent strain B301D of *P. s.* pv. *syringae* (13) was used as the reference strain in all tests.

**Pathogenicity tests.** In August 1986, 2- and 3-yr-old stems of three mature sweet cherry trees (cv. Bing), located at the Irrigated Agriculture Research and Extension Center, Prosser, Washington, were inoculated with 25 *Cytospora* isolates. The stems were surface-disinfested with 70% ethanol and wounded with a staple gun (Arrow T-50, Arrow Fastener Co., Inc., Saddle Brook, NJ). The staple gun was modified to include an Xpando T-50 pop rivet attachment (Arrow Fastener) from which the metal collar surrounding the steel shaft of the pop rivet had been removed. Four shots of the staple gun in a linear row created a 3- × 12-mm wound, which was 5.5 mm deep. The wounds were filled with agar plugs from 48-hr-old cultures of the *Cytospora* isolate grown on MA and were then wrapped in a single layer of Parafilm M (American Can Co., Greenwich, CT). The film was removed after 60 days. The wound sites were visually inspected for gummosis, canker, and pycnidium formation at monthly intervals. Twelve months after inoculation, canker length and the percentage of the stem circumference colonized (16) were recorded, and fungal reisolations were performed as described above.

The seasonal susceptibility of sweet cherry trees to *C. cincta* was tested using second and third-leaf trees (cv. Rainier) at Prosser, Washington. Trees were inoculated with *C. cincta* strain MTM1 at approximately 2-mo intervals, beginning in June 1986, by the method described above. Disease evaluations commenced 30 days after inoculation, continuing at 1-mo intervals until August 1987, when the trees were harvested. Within 24 hr of harvest, an attempt was made to reisolate the pathogen from both the bark and the inner woody tissue, by the methods described above, except that tissue samples were excised both 2 and 5 cm above and below the advancing margin of the canker.

The susceptibility of sweet cherry trees to bacterial canker, caused by *P. s.* pv. *syringae*, was tested in March and November under the same conditions and by the same methods described above for inoculation with *C. cincta*. The only exception was that 50 µl of either phosphate buffer or a cell suspension of *P. s.* pv. *syringae* strain B301D ($10^8$ cfu/ml) was added at the wound site. Strain B301D was grown on King's medium B agar plates for 24 hr, and cells were washed and adjusted to concentration by routine methods (13).

**Inoculation of excised cherry stems.** In March 1987, 1-yr-old stems of Bing cherry were collected from mature trees at Prosser, Washington, and stored at 5 C. Stem sections were surface-disinfested and inoculated within 48 hr after harvest, by the basic methods described by Weaver (22). The stems were cut to a length of 16 cm and then rinsed for 5 min in a stream of tap water. Surface disinfestation was accomplished by successive 10-min washes in 0.5% NaOCl, SDW, 70% ethanol, and SDW. Following disinfestation, the basal ends of the stems were sealed in melted paraffin to a depth of 3 cm and refrigerated at 5 C until they were inoculated, later that same day.

The modified staple gun was used as described above to create a wound 10 cm from the basal end of the cherry stem. Methods described above were also used to prepare and inoculate the stems with *C. cincta* MTM1. In some treatments, 25 µl of a suspension of *P. s.* pv. *syringae* B301D ($1.9 \times 10^8$ cfu/ml), prepared as described above, was added at the wound site. Controls were inoculated with either sterile phosphate buffer or MA. After inoculation, the stems were wrapped at the wound site with a single layer of Parafilm M and then placed in a sterile glass test tube with a moist cotton plug at the bottom. The tube was sealed with an aluminum foil cap prior to incubation at 15 C for 7 days. Twenty stems were prepared for each pathogen or control treatment.

At the end of the 7-day incubation period, 10 stems inoculated with *C. cincta* MTM1 were randomly selected and inoculated with *P. s.* pv. *syringae* B301D at the original wound site; conversely, stems first inoculated with *P. s.* pv. *syringae* B301D were inoculated with *C. cincta* MTM1. The methods of preparing the inocula and inoculation were as described for the initial inoculation. The control stems were treated a second time with either sterile phosphate buffer or MA. All treatments were then incubated at 15 C for an additional 8 days. Canker lengths were measured at both 7 and 15 days after the first inoculation. Attempts were made to isolate both pathogens at the end of the 15-day incubation period according to the methods described above.

## RESULTS AND DISCUSSION

**Isolations from orchard cankers.** Canker and dieback were observed in most orchards throughout the major sweet cherry production areas of eastern Washington, primarily the Yakima Valley, in orchard surveys between 1985 and 1987. Disease incidence within orchards ranged from just a few trees with mild disease symptoms to nearly all trees with light to severe levels of disease. *Cytospora* was the primary microorganism isolated from cherry cankers, being retrieved from 13 of 30 orchards surveyed (43%); the symptoms closely resembled those described by Wilson and Ogawa (26) for Cytospora canker of *Prunus domestica* L. Fluorescent pseudomonads were isolated from cankers in two of the surveyed orchards during the spring, but none of them were identified as *P. syringae*. Moreover, cankers from both of these orchards yielded *Cytospora*. These results resemble those of similar surveys of peach trees with canker and dieback in North Carolina (10), where *Cytospora* spp. were isolated from 58–76% of the samples and *P. syringae* was less frequently isolated.

*C. cincta* was the predominant species associated with Cytospora canker of sweet cherry trees in Washington (Table 1). Less than 5% of the *Cytospora* isolates were identified as *C. leucostoma* by the criteria of Hildebrand (14) and Willison (24). The *C. leucostoma* isolates grew optimally at 30 C, had pycnidial diameters less than 1.0 mm, and lacked an inhibition zone between pairs of isolates after growth on PDA. All Washington isolates of *C. cincta* grew optimally at temperatures below 30 C, had pycnidial diameters greater than or equal to 1.0 mm, and produced a zone of inhibition ranging from 7 to 30 mm between pairs of fungus subcultures. Recently, *L. cincta*, the teleomorph of *C. cincta*, was observed on cankered cherry trees in the Yakima Valley (R. Barakat and D. A. Johnson, *unpublished data*). The prevalence of *C. cincta* in sweet cherry orchards in Washington corresponds to reports that this species predominates in peach orchards in North Carolina and Ontario (6,10,21); less than 2% of the isolates were identified as *C.*

*leucostoma* in the studies of Endert-Kirkpatrick and Ritchie (10) and Tekauz and Patrick (21). Interestingly, surveys of plum orchards in California showed that *C. leucostoma* is the predominant species (3), presumably because it is favored by the higher ambient temperatures occurring in California orchards, compared to those in Washington, where the mean summertime temperature is about 22 C.

Lignicolous hymenomycetes were isolated from trees with discoloration and decay of the structural heartwood in approximately 20% of the orchards surveyed. Cultures were observed for the presence of clamp connections and other morphological characteristics of the mycelial mat and hyphae and placed into hymenomycete categories according to the scheme of Nobles (18). Minute fructifications were observed in some of the cultures, resembling those produced in vitro by *Polyporus* and *Fomes* spp. An earlier survey of wood decay fungi in Washington apple orchards revealed that 44% contained decayed heartwood resulting from hymenomycete infection (7). Thus, wood decay fungi appear to be present less frequently in sweet cherry than in apple orchards, although several of these fungi may be involved, since Shaw (20) identified 38 species as parasites of *Prunus avium* in Washington.

**Virulence of *Cytospora* isolates.** All 25 *Cytospora* isolates were pathogenic, according to inoculation tests on 2- and 3-year-old branches of mature Bing cherry trees (Table 1). Virulence varied among the isolates, however, since the mean percentage of the stem circumference colonized ranged from 8 to 100%. Nevertheless, nearly 60% of the isolates, including strain MTM1, were found to be highly virulent, colonizing over 75% of the stem circumference in 12 mo. The single isolate identified as *C. leucostoma* was also virulent, colonizing an average of 75% of the stem circumference. At the end of the 12-mo test, several branches distal to the point of inoculation were killed, showing the typical leaf flagging associated with Cytospora canker. Pycnidia were formed by at least eight of the *C. cincta* isolates, but they were found only on branches killed by the fungus. The control branches were free of infection, and the original wounds were observed to be healing normally. Moreover, *Cytospora* was not isolated from the control branches, but reisolations from cankers yielded *Cytospora* in 80% of the cases, illustrating that the fungus often dies in cankers, especially after a winter period when there is little or no fungal activity (10).

Inoculations of vigorously growing second- and third-leaf sweet cherry trees with *C. cincta* at approximately bimonthly intervals resulted in the eventual development of Cytospora canker. Regardless of the month of inoculation, canker expansion was usually greatest from March to May, with the average canker length increasing by approximately 1 cm per month. Only moderate canker expansion occurred during July and August, and little or no appreciable expansion occurred in the autumn and winter months. The expansion of cankers caused by *C. cincta* during the cool and moist spring period corresponds to other studies of *C. cincta* in *Prunus* spp. (3,6,14,23). This is attributed to ambient orchard temperatures that are close to the optimal temperature for the growth of *C. cincta*, which ranges between 20 and 25 C in vitro, depending on the strain (3). In contrast, *C. leucostoma* has been reported to be more virulent than *C. cincta* during the summer months in peach orchards in New York (14) and Canada (6,21) and in plum orchards in California (3).

*C. cincta* was reisolated in August 1987 from virtually all of the inoculated trees. It was isolated not only from infected bark and woody tissues but also from as far as 2 cm beyond the margin of the canker in some specimens. In histological studies (4,27), hyphae of *C. cincta* and *C. leucostoma* were observed to extend past the margin of the canker in both bark and woody tissues, reflecting the lack of effective boundaries in tissues at the proximal and distal margins of the canker.

**Interactions between *C. cincta* and *P. s.* pv. *syringae*.** Inoculations of second- and third-leaf cherry trees with *P. s.* pv. *syringae* B301D in the spring usually resulted in the formation of sunken, elliptical cankers with a distinct margin. After inoculation in March, for example, a 3-cm-long canker developed by mid-May, encompassing 14% of the stem circumference. Nevertheless, infections with *P. s.* pv. *syringae* were usually short-lived; the typical canker was well callused 1–2 mo later, and the bacterium could not be reisolated from the affected tissue. Such a decline in viable bacterial cells in cankers closely corresponds to the precipitous drop in epiphytic populations of ice-nucleation-active *P. syringae* to undetectable levels in sweet cherry orchards in eastern Washington, reflecting the unfavorably warm, dry conditions that prevail during the summer (12). Inoculations of trees with the bacterium in November did not result in canker formation.

Cankers caused by *C. cincta* MTM1 in excised sweet cherry stems were approximately nine times as long as those caused by a highly virulent strain of *P. s.* pv. *syringae* after 7 days of incubation at 15 C (Table 2). Subsequent counter-inoculations with the second pathogen had no notable effect on the relative severity of canker development after an additional 8 days of incubation. Stems inoculated with *C. cincta* first and then *P. s.* pv. *syringae* yielded only the fungal pathogen. Conversely, stems inoculated with *P. s.* pv. *syringae* and then counter-inoculated with *C. cincta* nearly always yielded the bacterium and rarely the fungus. Control stem treatments did not yield either of the pathogens. These results suggest that the two pathogens do not coexist in cankers for extended periods. Exclusion of the bacterium by the fungus was also observed in peach stems and was attributed to the acidification of the cankered tissue by the fungus to pH values below 4.5 (10). The antagonistic effect on the growth of

**Table 1.** Identification and virulence of *Cytospora* strains isolated from sweet cherry trees in Washington State

| Strains identified as *C. cincta* | Strains identified as *C. leucostoma* | Mean percentage of stem circumference colonized[a] |
|---|---|---|
| 14 | 0 | 76–100 |
| 8 | 1 | 51–75 |
| 1 | 0 | 26–50 |
| 1 | 0 | 1–25 |
| 0 | 0 | 0 |

[a] Mature sweet cherry trees, cv. Bing, were inoculated in August 1986, and disease was assessed in August 1987. Each strain was tested by inoculation of stems of 2- and 3-yr-old wood on three replicate trees. The mean percentage of stem circumference colonized was determined by the method of Kessler (16). The numbers show the size range of cankers caused by the corresponding number of *Cytospora* strains.

**Table 2.** Effect of inoculation sequence of *Cytospora cincta* MTM1 and *Pseudomonas syringae* pv. *syringae* B301D on canker development in excised sweet cherry stems

| First inoculation | Canker length after 7 days (mm)[a] | Second inoculation | Canker length after 15 days (mm)[a] |
|---|---|---|---|
| *C. cincta* | 37.9 ± 6.8 | *P. s.* pv. *syringae* | 43.5 ± 6.5 |
| *P. s.* pv. *syringae* | 4.2 ± 0.3 | *C. cincta* | 18.5 ± 2.5 |

[a] Mean canker length (plus or minus standard error) of 10 replicate sweet cherry stems, cv. Bing, excised in March 1987.

*C. cincta* in newly formed canker tissues heavily colonized by *P. s.* pv. *syringae* may have resulted from the production of inhibitory metabolites, such as pyoverdin$_{pss}$ (5) or syringomycin (11), which are antagonistic to the growth of *Cytospora* in vitro (D. C. Gross, *unpublished data*).

**Canker in Washington State.** Several conclusions can be made concerning the etiology and prevalence of canker and dieback diseases in eastern Washington sweet cherry orchards. First, Cytospora canker is the principal aerial canker disease occurring in orchards. Bacterial canker, caused by *P. s.* pv. *syringae*, was not found in our survey of orchards where canker and dieback were endemic. Only occasionally was bacterial canker observed in sweet cherry orchards, and this was in the spring following frost injury around the time of bloom, when high epiphytic populations of the bacterium were present (12,13). The occurrence of wood decay, which can be caused by several lignicolous hymenomycetes (20), is largely restricted to older trees and is frequently associated with trees undergoing decline due to Cytospora canker. Preliminary pathogenicity tests of representative wood decay hymenomycetes isolated from vigorously growing sweet cherry trees showed only a small region of rot within the woody tissues after several months of incubation (K. M. Regner and D. A. Johnson, *unpublished data*). Because wood decay is a slow process even in stressed trees, a long incubation period is needed for a high level of disease (2,8,9).

*C. cincta* was found to be the predominant species causing Cytospora canker. Infection and lesion development occurred primarily in the spring. This is particularly noteworthy, since *C. leucostoma* is the cause of Cytospora canker in California plum orchards, where the disease develops primarily in the summer (3). The isolates of *C. cincta*, moreover, were generally highly virulent, causing cankers on vigorously growing mature and immature sweet cherry trees. This observation supports reports from North Carolina (10), Canada (6,21), and Hungary (19) that most strains of *C. cincta* are aggressive pathogens that are not restricted to causing disease in weakened *Prunus* trees, as suggested by some studies (14,26). Nevertheless, winter injury can promote infection by *Cytospora* spp. and lower the resistance to disease development (15,25). Many of the sweet cherry orchards in eastern Washington are relatively old and have been subjected to winter injury, and this situation has undoubtedly contributed to the increase in reports of stem cankers and dieback. The proportion of orchards affected with wood-rotting hymenomycetes will also likely increase from the 20% level found in this survey as the average age of Washington orchards increases.

Finally, the low incidence of bacterial canker and the apparent inability of *P. s.* pv. *syringae* and *C. cincta* to coexist for extended periods in cankered tissues indicate a lack of interaction between the two pathogens in eastern Washington sweet cherry orchards. This does not preclude the possibility that *C. cincta* occasionally supplants *P. s.* pv. *syringae* in stem cankers, since the bacterium is reported to be commonly succeeded by *C. cincta* in canker lesions in North Carolina peach orchards (10). Nevertheless, recognition that Cytospora canker is the major canker disease in sweet cherry orchards in Washington State will lead to the implementation of improved control practices.


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