

# Bacterial canker of sweet cherry in Oregon

## DISEASE SYMPTOMS, CYCLE, AND MANAGEMENT

EM 9007 • May 2010

Robert A. Spotts, Jeff Olsen, Lynn Long, and Jay W. Pscheidt

**B**acterial canker is the number one killer of young sweet cherry trees in Oregon. This disease is particularly destructive in higher rainfall districts such as the Hood River and Willamette Valleys. It is still an important disease in drier climates such as The Dalles, the Mosier area, and Central Washington. Where conditions have been favorable for disease development, tree losses of 75% have been observed in young orchards. Under normal conditions, losses between 10% and 20% are not uncommon. In the Mid-Columbia district, sweet cherry trees have been planted beyond the area that experiences the weather-moderating effects of the Columbia River, thus increasing the risk of these orchards to spring frosts and cold winters that promote bacterial canker.

The bacterial canker pathogen also attacks pear trees, causing blossom blast and trunk cankers. Bactericide spray programs for pears are of limited value, but the disease has become less of a problem as the number of wind machines in an area has increased. The wind machines not only provide frost protection, but they also modify the microclimate in orchards to produce warmer, drier conditions that are less favorable for bacterial canker.

### Cause and symptoms

Bacterial canker is caused by the bacterium *Pseudomonas syringae* pv. *syringae*. The main symptom on young sweet cherry trees is a canker that appears slightly sunken and darker than other areas on the trunk or larger scaffold limbs (see figures 1 and 2). The inner tissue of cankers is orange to brown, and narrow, brown streaks extend into healthy tissue above and below the canker.



Photo by Robert A. Spotts, © Oregon State University.

**Figure 1. Bacterial canker at heading cut on Bing on Gisela 6.**

Robert A. Spotts, professor emeritus of plant pathology, Mid-Columbia Agricultural Research and Extension Center, Oregon State University. Jeff Olsen, Extension horticulturist, Yamhill County, Oregon State University. Lynn Long, OSU Extension horticulturist, Wasco County, Oregon State University. Jay W. Pscheidt, Extension plant pathology specialist, Department of Botany and Plant Pathology, Oregon State University.





Photo by Robert A. Spotts, © Oregon State.



Photo by Jay W. Pscheidt, © Oregon State University.

**Figure 2. Two photos showing bacterial canker of sweet cherry.**

A clear to amber ooze that may be gummy or sticky may or may not be associated with the cankers. Cankers may eventually girdle limbs or trunks. Leaves droop, turn light green to yellow, and limbs or entire trees may die during hot weather. Infected dormant buds often are killed in the Willamette Valley, a symptom known as dead bud (see figure 3).

If blossom infection occurs, cankers subsequently form on twigs and spurs, and the dead flowers remain attached on fruit spurs. Leaf and fruit infections are rare in Oregon's major fresh sweet cherry production regions. Leaf spots are at first water soaked but then become dry and brown. Fruit infections appear as small, dark, sunken lesions with distinct margins (see figure 4).

## Disease cycle

This bacterium colonizes the surfaces of many plants, including weeds and grasses commonly found in orchards. It has also been found in healthy-appearing wood and buds of sweet cherry. In the Willamette Valley, bud infections (dead bud) are common. These infections originate at the base of outside bud scales between November and February,

but a small percentage takes place through leaf scars formed when the leaves drop in the fall.

Infection of young trees occurs mainly in tissue damaged by spring frost, severe winter freezes, pruning cuts, insect injuries, or at leaf scars. Tree death is generally greatest in the first two years after planting, and trees are seldom killed eight years or more after planting. Severe infections and a high rate of tree death are related to heading cuts.

*P. syringae* populations on sweet cherry trees increase 10- to 100-fold during bloom in mid-April, the time many orchards are planted. *P. syringae* populations are low during summer, but with the return of autumn rains and cooler temperatures, *P. syringae* is detected at high levels prior to and during leaf fall in sweet cherry orchards. Infection at leaf scars can be high at this time and results in tree death. Infection of sweet cherry also increases when tissue is exposed to severe temperatures during early and mid-winter. Gummosis is associated with many cankers and is a common source of bacterial cells that can be spread by rain and insects.



Photo by Robert A. Spotts, © Oregon State.

**Figure 3. Dead buds on sweet cherry.**



Photo by Robert A. Spotts, © Oregon State.

Figure 4. Infection of sweet cherry fruit by *P. syringae*.

## Disease management

### Pruning

*P. syringae* can infect the woody tissue of sweet cherry at wound and injury sites. However, severe infections and a high rate of tree death are related to heading cuts. Since bacteria often are spread by rain, heading cuts should be made during dry weather. Heading cuts become resistant to infection after one week in summer but may take three weeks in winter. If rain occurs before cut tissues become resistant to infection, protective measures must be taken.

Scoring and summer pruning cuts are potential sites for infection. Scoring usually is done before the spring increase of *P. syringae* populations. Summer pruning is done during dry, hot weather when *P. syringae* populations are lowest. Since these wounds are potential sites for infection, growers should not make these cuts if summer rain occurs or if over tree sprinklers are used for irrigation or evaporative cooling. Bacterial canker cannot be spread in summer or winter by cutting through active cankers then immediately using the same pruning tool to make heading cuts on healthy trees. Leaf scars can become naturally infected and are another area in need of protection.

### Cultivar susceptibility

The cultivars Rainier and Regina appear more resistant than Sweetheart and Bing. In a Willamette Valley survey, Sandra Rose and Regina had less dead bud and blossom infection than Sweetheart, Stacatto, or Solamente.

### Rootstocks

Rootstock significantly affects cultivar susceptibility to bacterial canker. Death of trees on Mazzard was 30% but was 77% when trees were on Gisela 6 rootstock. While no Bing on Colt rootstock died, mortality of Bing on Gisela 6 was 90% in one study. Trees on Gisela rootstocks have shown increased susceptibility in field observations. Bing on Krymsk 5 had smaller heading cut cankers than trees on Mazzard or Gisela 6, and 43% of trees died on Krymsk 5 compared with 50% on Mazzard.

### Integrated disease management

An integrated approach is necessary for successful management of bacterial canker of sweet cherry. Because all natural and manmade injuries are susceptible to infection by *P. syringae*, injuries should be avoided whenever possible. Infection sites also should be protected until healing occurs. Bacterial canker is reduced when tree trunks are painted white to reduce sun scald-related winter injury.

Growers are encouraged to make heading, pruning, and scoring cuts only during dry weather. For this reason, pruning in the summer can help to keep infection low, especially if the disease is already present within a block. However, in large cherry orchards or where trees are planted on productive rootstocks such as Gisela, it may not be possible or advantageous to prune all trees in summer. In this case, colder, dry periods of mid-winter when temperatures are around freezing or slightly above can provide a reasonable alternative to summer pruning.

Heading cuts and leaf scars are particularly susceptible and require additional protection. Copper-based products provide poor control of bacterial canker and in some cases have resulted in more bacterial canker than in nonsprayed trees. None of the cultivar/rootstock combinations consistently showed high resistance to bacterial canker, but Bing on Gisela 6, which had a mortality of 90%, should not be planted in areas where bacterial canker is a problem.

In addition, it is important to plant cherry trees in well-drained soils and keep trees moderately vigorous through proper irrigation and nutrition. Field observations indicate that stressed trees show higher levels of infection than those with a moderate amount of vigor.



## Steps to manage bacterial canker of sweet cherry

1. Do not interplant new trees with old trees, which are major sources of *P. syringae*.
2. Keep irrigation water off aboveground tree parts as much as possible for the first 2 or 3 years after planting.
3. Avoid all types of injury, including mechanical, insect, and frost injuries. Paint all trunks white with latex paint to prevent winter injury.
4. Less disease occurs when summer pruning is used. Prune only during dry weather.
5. Remove and destroy branches and trees killed by *P. syringae*.
6. For recommendations on resistant cultivar/rootstock combinations, see discussion in text.
7. Locate the orchard in an area less likely to be affected by frost and slow drying conditions.
8. Provide optimal soil conditions for growth of sweet cherries, including attention to pH and nutrition. Application of excess nitrogen, especially late in the growing season, may promote late-season growth that is susceptible to low-temperature injury in early winter, followed by bacterial infection.
9. Control weeds, especially grasses. They often support large populations of *P. syringae*. Clover and vetch groundcovers support lower populations. Consider clean cultivation of row middles for the first 3 years.
10. Strains of *P. syringae* resistant to copper-based bactericides are widespread in the Mid-Columbia area. Copper sprays may result in more bacterial canker and should not be used.
11. Test for and control plant pathogenic nematodes before planting. High populations of ring nematode have been associated with more bacterial canker.
12. In high infection areas, plant trees later in spring to avoid cool, wet conditions.

## For More Information

- Kennelly, M.M., F.M. Cazorla, A. de Vicente, C. Ramos, and G.W. Sundin. 2007. *Pseudomonas syringae* diseases of fruit trees: progress toward understanding and control. Plant Disease 91:4-17.
- Spotts, R.A., K.M. Wallis, M. Serdani, and A.N. Azarenko. 2010. Bacterial canker of sweet cherry in Oregon—infection of horticultural and natural wounds, and resistance of cultivar and rootstock combinations. Plant Disease 94:345-350.

---

© 2010 Oregon State University. This publication was produced and distributed in furtherance of the Acts of Congress of May 8 and June 30, 1914. Extension work is a cooperative program of Oregon State University, the U.S. Department of Agriculture, and Oregon counties. Oregon State University Extension Service offers educational programs, activities, and materials without discrimination based on age, color, disability, gender identity or expression, marital status, national origin, race, religion, sex, sexual orientation, or veteran's status. Oregon State University Extension Service is an Equal Opportunity Employer.

---

Published May 2010.