Brown Rot

Phillip M. Brannen
Department of Plant Pathology
University of Georgia
Athens, GA 30602

Guido Schnabel
Department of Plant Pathology & Physiology
Clemson University
Clemson, SC 29634

Monilinia fructicola (Wint.) Honey is one of several species of *Monilinia* that cause brown rot of peach. Other species of this genus have not been reported in the southeastern United States. Brown rot was first described in 1796 in Europe. Fungi that cause brown rot are distributed worldwide. The species that causes the disease in the Southeast is also found throughout the Americas, New Zealand, Australia, Africa, Japan, and Argentina. Brown rot was recognized in the United States as early as 1807, but it was not determined to be caused by a fungus until 1855.

Symptoms

The brown rot fungus can infect fruit, shoots, and flowers of peaches and other stone fruits. Spores infect flower and fruit tissues. Stems are usually infected by invasion of the fungus from infected blossoms and fruit. Direct infection of leaves and stems by spores is rare in the Southeast; such infections are more likely to occur in plum and certain ornamental *Prunus* species.

Blossoms

Blossom blight, the bloom-infecting stage of brown rot fungus, can invade through any part of the flower (Figure 1); following infection, diseased tissues turn dark brown. Almost immediately, tan- to buff-colored tufts of spores appear on infected tissues.

Figure 1. Brown rot blossom blight. Note production of grayish-tan spore masses on the twig at the base of the blighted blossom.

When a full flower crop is present, blossom blight seldom causes significant crop loss in peach. Under conditions of a reduced flower crop, as with spring frost or bloom thinning, blossom blight can result in unacceptable crop loss. Bloom sprays are critical when flower numbers are significantly reduced. When a full flower crop is present, the primary benefit of blossom blight control is reduction of inoculum levels, which will often reduce pre-harvest brown rot levels. Blossom blight infections are generally more prevalent in blocks where severe brown rot occurred in fruit the year before.

Ordinarily, less than 1% of blossoms will be affected by brown rot. However, the confluence of high inoculum levels and environmental conditions conducive to blossom blight can lead to heavy losses. For example, spring crop losses that prompt abandonment of disease control can lead to heavy overwintered brown rot inoculum. In Georgia, blossom blight has infected up to 40% of blooms when a wet bloom period followed this type of inoculum buildup. Even this unusually high level of blossom blight does not directly result in crop loss. However, destructive levels of brown rot inoculum often build up in these blocks, and heavy crop losses can occur before and during harvest.

Stem Cankers

When flower or fruit pedicels are infected, the brown rot fungus frequently spreads into twigs and small branches and forms a brown, collapsed lesion known as a canker. These depressed, elliptical areas may enlarge until the entire stem is girdled. After girdling, the shoot tip withers and dies, while the leaves remain attached. A drop of gum is present when brown rot cankers are formed. During moist weather, tufts of spores may be visible on the cankers. Lesions of *Phomopsis amygdali* (constriction canker or Phomopsis twig blight) are often confused with brown rot lesions. However, there are distinctive differences. Lesions of constriction canker are generally found at buds or nodes, whereas

brown rot lesions are found at a blossom site. Very limited gum exudate is found with constriction canker lesions, but profuse gumming is observed with brown rot. Constriction canker lesions also form zonate patterns as they develop.

Fruit

Brown rot results in four types of fruit infections in the Southeast: infections of aborted, non-abscised fruit (buttons) (Figure 2); infection of late-thinned fruit on the orchard floor; infected green fruit (Figure 3); and infection of mature fruit during harvest and handling operations (Figure 4). After harvest, the development of fruit mummies provides a significant overwintering mechanism. Fruit that is thinned at or after pit-hardening provides a good growth medium for the fungus. Inoculum from thinned fruit after pit-hardening can dramatically increase infection of mature fruit. Fruit that is thinned prior to pit-hardening will quickly disintegrate.

Figure 2. Brown rot on an aborted fruit ("button").

Figure 3. Brown rot on insect-damaged green fruit. Note small insect feeding puncture in the center of the rotted area.

Figure 4. Brown rot on a ripe fruit. Note the production of spore masses in more or less concentric rings.

Latent infections of immature fruit can serve as a source of inoculum for subsequent fruit rot. High levels of latent infection are more common following blossom blight infections. In addition, insect- or cold-damaged green fruit are more susceptible to brown rot. Research on plums suggests that fruit-to-fruit contact may also predispose fruit to infection by *M. fructicola*. Unfortunately, detection of the pathogen on any fruit is based on the presence of masses of gray to brown fungal spores. Unless sporulation occurs, infection of these fruit is not easily detected.

Infection of maturing fruit just prior to harvest is the most destructive phase of the brown rot fungus. The first indication of pre-harvest infection is the development of small, superficial, circular brown spots, sometimes called pen-rot. Under optimal conditions, spots can enlarge at a rate of 0.02 to 0.04 inches per hour. As lesions enlarge, the skin of the fruit is ruptured with tufts of gray to light tan spores, frequently formed in concentric rings (Figure 4).

Figure 5. Rhizopus rot on a ripe fruit.

Figure 6. Anthracnose rot on ripe fruit.

Before sporulation occurs, the fruit rot lesions of brown rot, Rhizopus rot, and anthracnose are more difficult to separate from one another. When infected by *Rhizopus nigricans* (Figure 5), the skin of rotted fruit slips off very easily, and the flesh is softer and more watery than that of brown rot-infected fruit. Brown rot fruit infections are smooth. Anthracnose infections (*Glomerella cingulata*) (Figure 6) form a depressed, circular spot on the fruit surface. Anthracnose lesions are marked by the occasional presence of cream to salmon-pink concentric rings of spores; also, a cone of rotted flesh can easily be separated from the healthy flesh.

Mummies

Brown rot completely permeates a peach fruit. As the disease cycle progresses, the fruit tissue is darkened by thick-walled fungal cells. Fruit shrivel to produce a wrinkled, very dark brown to black “mummy,” which may remain attached (Figure 7) or drop to the ground (Figure 8).

Figure 7. Mummy of a brown-rotted peach fruit hanging in the tree.

Figure 8. Mummy of a brown-rotted peach fruit that has fallen to the ground.

Figure 9. Cup-like brown rot apothecia that form on partially buried peach mummies on the ground. These fungal structures produce the spores that result in some of the primary infections in the spring.

Brown rot mummies that are partially buried within a couple of inches of the surface in moist soil may produce spore-bearing apothecia during the next bloom period. Apothecia (Figure 9) are smooth, fleshy, brown to reddish brown, cup-like structures that vary in size from 1/4 to 3/4 inch in diameter. The cup-like structures arise on a stalk that may be up to 2 inches in length. A slight jarring of the apothecia will result in a small cloud of visible spores. Apothecia are seldom seen in Georgia, but they increase in importance as one moves north through the Carolinas.

Disease Cycle

The brown rot fungus overwinters predominantly as mummies or in cankers. Each year, the disease cycle is initiated by primary spores produced either in apothecia or infected tissues, including mummies on the tree. The air-borne spores infect blossoms of peach and other susceptible plants such as wild plum.

Infection can take place at temperatures as low as 41°F, but the optimum temperature for infection is 77°F. Moisture, in the form of exudate on stigmata, wounds, or free water as dew or rain, is important for infection. Infection can take place at relative humidities (RH) as low as 94%, but higher humidity levels are conducive to higher infection levels. Under optimal conditions of moisture and temperature, infection takes one to six hours.

Almost immediately after symptoms appear on the blossoms, and subsequently the stems, brown rot will begin producing spores. This spore production is important for secondary infections. If sources of secondary inoculum can be eliminated or avoided, disease control opportunities improve. Spore production on infected flowers can continue through early June during dry seasons. Sporulation of infected blooms may occur over an even longer period during a wet season.

Even without continued spore production on infected blossoms, the disease cycle can continue if infected buttons or damaged, infected fruit are present. Fruit that has been removed during thinning, at or after pit-hardening, can also provide a continuous source of spore production. Infected fruit can provide a bridge, assuring abundant inoculum from blossom to mature fruit infection.

Mature fruit are very susceptible. Assuming that a continuum of spores is present, infection of maturing fruit is probable. Given the short period of time required for infection, the high likelihood of optimal conditions for infection (moisture and temperature), and the overwhelming spore numbers that result from a continuum of infection, pre-harvest brown rot infections readily reach epidemic level.

Rain splashing is an important means of spore dissemination. Spores can also be released in copious quantities through bumping and brushing of leaves and stems by wind. Insects such as driedfruit beetles (nitidulid beetles) can also be important in the spread of brown rot.

Control

Pre-harvest brown rot is a very serious disease. A season-long management strategy is normally best. The most critical times for control of brown rot are during bloom and prior to harvest. A minimum-risk, protective fungicide program requires two to three applications during bloom and two tothree applications prior to harvest.

Sanitation is a key to management of brown rot. The proximity of readily available inoculum sources (i.e., wild plums, off-type trees where the disease was not controlled the previous season, otherwise abandoned *Prunus* species, and mummies from the previous season) all but assures high inoculum carryover. Crops abandoned after a destructive frost often experience a tremendous build-up of brown rot inoculum on the few remaining fruit. These blocks will require special attention the following spring.

Wild plum thickets are an important source of spores for both primary and secondary infections. Wild plums bloom before and during peach bloom, and the fruit mature continuously from early to late in the season, providing a continuing source of inoculum. Removal of wild plums adjacent to a peach orchard is an important sanitation procedure.

Research on plums suggests that thinning fruit to reduce fruit clustering and application of fungicides effective against brown rot before fruit contact occurs might reduce fruit losses from brown rot. Additional hand-thinning to remove buttons would be ideal, but is probably not practical. When buttons are abundant, orchards should be watched more closely for brown rot. An intensive pre-harvest spray program should be used if brown rot becomes evident.

Brown rot losses can continue after harvest during storage and transit. See the discussion of post-harvest diseases for additional information on this phase of brown rot.

References

Biggs, A. R. and J. Northover. 1985. Inoculum sources for *Monilinia fructicola* in Ontario peach orchards. Canadian Journal of Plant Pathology 7: 302-307.
Burr, T. (ed.). 1985. Proceedings of the Brown Rot of Stone Fruits Workshop. Special Report No. 55, New York State Agricultural Experiment Station, Geneva.
Byrde, R. J. W. and H. J. Willets. 1977. The brown rot fungi of fruit - their biology and control. Pergamon Press, New York.
Clayton, C. N. 1942. The germination of fungus spores in relation to controlled humidity. Phytopathology 32: 921-943.
Corbin, J. B. and J. M. Ogawa. 1974. Springtime dispersal patterns of *Monilinia laxa* conidiain apricot, peach, prune, and almond trees. Canadian Journal of Botany 52: 167-176.
Corbin, J. B., J. M. Ogawa and H. B. Schultz. 1968. Fluctuations in numbers of *Monilinia laxa* conidiain an apricot orchard during the 1966 season. Phytopathology 58: 1387-1394.
Emery, K. M., T. J. Michailides and H. Scherm. 2000. Incidence of latent infection of immature peach fruit by *Monilinia fructicola* and relationship to brown rot in Georgia. Plant Disease 84: 853-857.
Kable, R. E. 1975. The frequency and timing of benomyl sprays for control of brown rot *(Monilinia fructicola)* in canning peaches. Journal of Horticultural Science 50: 143-150.
Landgraf, E. A. and E. I. Zehr. 1982. Inoculum sources for *Monilinia fructicola* in South Carolina peach orchards. Phytopathology 72: 185-189.
Michailides, T. J. and D. P. Morgan. 1997. Influence of fruit-to-fruit contact on the susceptibility of French prune to infection by *Monilinia fructicola.* Plant Disease 81: 1416-1424.
Scott, W. M. and T. W. Ayres. 1910. The control of peach brown rot and scab. USDA Bulletin 174: 1-31.
Sutton, T. B. and C. N. Clayton. 1972. Role and survival of *Monilinia fructicola* in blighted peach branches. Phytopathology 62: 1369-1373.
Tate, K. G. and J. M. Ogawa. 1975. Nitidulid beetles as vectors of *Monilinia fructicola* in California stone fruits. Phytopathology 65: 977-983.
Weaver, L. O. 1950. Effect of temperature and relative humidity on occurrence of blossom blight on stone fruits. Phytopathology 40: 1136-1153.
Wilson, E. E. and G. A. Baker. 1946. Some aspects of the aerial dissemination of spores with special reference to

conidia of *Sclerotinia laxa.* Journal of Agricultural Research 72: 301-327.
Zehr, E. I. 1995. Constriction canker. Pages 31-32 in: Compendium of Stone Fruit Diseases. J.M. Ogawa, E.I. Zehr, G.W. Bird, D.F. Ritchie, K. Uriu and J.K. Uyemoto (eds.). APS Press, St. Paul, MN.