

FUNGAL GUMMOSIS

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Peach tree fungal gummosis was initially observed in the United States near Fort Valley, Georgia, during the 1960s. It subsequently spread to other production areas of the Southeast. The disease was independently discovered at about the same time in Japan, where it is described as peach blister canker or *ibokawa byo*, and was later reported in China and Australia. This disease, characterized by symptoms associated with bark lenticels, contributes to a general decline of trees.

CAUSAL ORGANISM

Symptoms of fungal gummosis are caused by a physiological race of *Botryosphaeria dothidea* (Moug.:Fr.) Ces. & De Not. (syn. *B. berengeriana* De Not., anamorph *Fusicoccum aesculi* Corda) specific to peach. Other *Botryosphaeria* species have been reported to cause peach gummosis in the United States; however, these species are primarily wound invaders that are not known to cause infections at lenticels. *B. rhodina* (Cooke) Arx is relatively rare on peach. *B. obtusa* (Schwein.) Shoemaker is very common in the southeastern United States, but its importance as a peach pathogen is unclear. *B. obtusa* is localized in dead tissue and outer bark (rhytidome) and is absent or infrequent in the newly infected cortex and phloem.

SYMPTOMS

The earliest fungal gummosis symptoms appear on young bark of vigorous trees as blisters 1 to 6 mm in diameter, generally each with a lenticel at its center (Figure 1). These raised areas are due to an abnormal multiplication of plant cells (hyperplasia) in response to the causal organism at the lenticel. Removal of outer bark with a knife reveals diseased tissue at the lenticel margin surrounded by the hyperplastic tissue. Blisters may be observed late in the season when infection occurs or the following spring. By the end of the second season, the area of necrosis surrounding lenticels has enlarged, and the hyperplasia is often less visible or absent. Some second season necrotic lesions exude resins. Lesions that appear in the second season after infection may or may not be preceded by the formation of blisters. Beginning when trees are 2 or 3 years old, sunken necrotic lesions encircling lenticels can be seen on the trunk and major branches (Figure 2). Typically, copious resin exudate is associated with lesions at multiple sites (Figure 3). Lesions 2 cm or more in diameter on the oldest bark may coalesce to form large cankers (Figure 4). Phloem and cortex are primarily affected; however, necrosis may extend to the xylem.

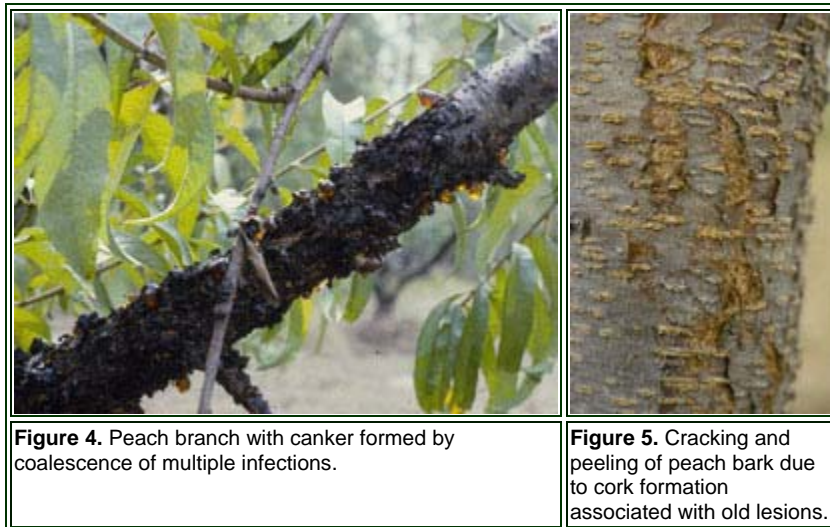


Figure 1. Peach branch with blisters caused by the fungus *Botryosphaeria dothidea*.

Figure 2. Three-year-old peach trunk with bark section removed to show multiple necrotic lesions.

Figure 3. Trunk of peach tree with multiple infection sites exuding resin.

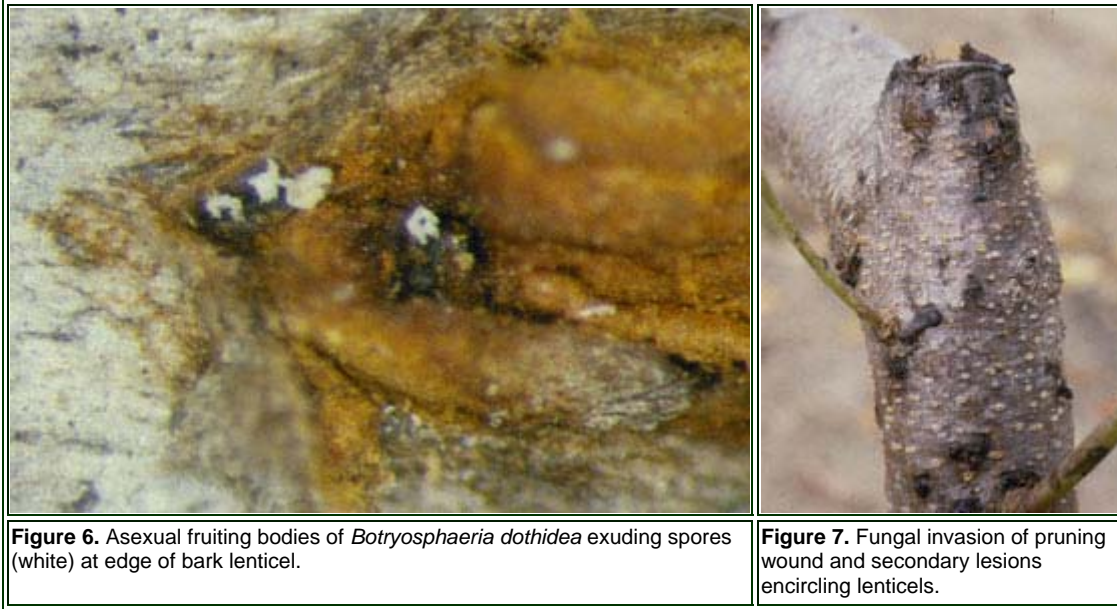
Figure 4. Peach branch with canker formed by coalescence of multiple infections.

Figure 5. Cracking and peeling of peach bark due to cork formation associated with old lesions.

Peach orchards that are poorly managed or water stressed are more likely to have severely infected trees. In its most severe form, the gummosis kills branches or entire trees. When growing conditions are optimal and inoculum levels are low, trees produce a cork layer (periderm) below the infected area, and the diseased tissue peels away from the tree (Figure 5). After repeated infections, the bark becomes rough and scaly.

DISEASE CYCLE

The fungus overwinters in diseased bark and in dead and dying wood, where it produces an abundance of spores (Figure 6). It spreads within the orchard mainly by dispersal of conidia in rainwater. In the southeastern United States, asexual spores of the fungus are present from March through October. Infections at lenticels develop from March through August, but May through July is the key infection period. The fungus also invades through wounds, causing cankers. Cankers may remain active for more than one year and lead to secondary infections at lenticels (Figure 7). Blossoms, leaves, and fruit are not infected.



CONTROL

Dead wood should be removed during winter pruning, and pruned wood should be taken from the orchard or destroyed mechanically (e.g., with a flail mower) to reduce inoculum from these sources. Summer pruning encourages fungal gummosis, because available inoculum can germinate and colonize wounds rapidly during this time. Abstaining from summer pruning and alleviating water and nutrient stress can reduce disease incidence and severity. Fungicides such as captan, applied to bark thoroughly and repeatedly (e.g., biweekly) during the key infection period (May through July), can be effective, but this may not be economically feasible. Use of this intensive fungal gummosis program for the first three years after planting trees generally prevents the non-wound invasion of the trunk and major scaffold branches.

Peach varieties vary in their resistance or susceptibility to fungal gummosis. For instance, Summergold is highly susceptible, whereas Harbrite has a relatively low susceptibility. In the long term, the best solution to this disease is the development of host resistance. Peach trees are evaluated for resistance to fungal gummosis within the breeding program at the USDA-ARS, Southeastern Fruit and Tree Nut Research Laboratory, Byron, Georgia.

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