

Botryosphaeria Diseases of Apple and Peach in the Southeastern United States

Two or more *Botryosphaeria* species cause canker and fruit rots on both pome and stone fruits. *B. dothidea* (Moug. ex Fr.) Ces. & de Not. (syn. *B. ribis* Gross. & Dug.) and *B. obtusa* (Schw.) Shoemaker cause major diseases of apple and peach, and *B. rhodina* (Berk. & Curt. apud Cke.) v. Arx has been associated with a canker and fruit rot disease of peach. In the United States, these diseases are primarily a problem in the Southeast, although not restricted to that region.

B. dothidea has recently been reported on apple trunks in Chile but is of minor importance there (9), and the fruit rot phase has not been detected. Although of minor importance in the southeastern United States until 12 years ago, the canker phase has recently been associated with declining orchards.

These fungi are also involved in a serious canker disease of peach called fungal gummosis and in minor postharvest fruit rots in Georgia and South Africa. The peach gummosis canker disease is spreading to other southeastern states and also occurs in Japan and mainland China. Other fruits affected by these facultative parasites include cherry, almond, currant, gooseberry, grape, and blueberry.

Activities of these fungi are temperature-dependent, which apparently accounts for many regional differences in disease severity and epidemiology. In the Carolinas, *B. obtusa* sporulates from mid-March to August, too late to infect apples before bloom (13); prebloom infection does occur in Georgia and in the coastal plain of Virginia, however. Late-season apple infection by *B. dothidea* and *B. obtusa* occurs in all of the southeastern states. North of the Shenandoah Valley, these fungi do not pose an economic threat, although unthrifty or poorly pruned trees may be infected by *B. obtusa*. Infection often follows fire blight caused by *Erwinia amylovora* (Burrill) Winslow et al; the fungus colonizes dead tissue and expands the cankers until large limbs are girdled and killed. In the South, *B. obtusa* invades fire-blighted tissue but damage is restricted to twigs, probably

because the warmer climate is less favorable for disease development by this fungus. Infection sites provided by poor pruning in the South are generally colonized by *B. dothidea*, which is favored by higher temperatures.

The Pathogens

B. dothidea has undergone a number of name changes. De Cesati and de Notaris established the genus *Botryosphaeria* in 1863 and listed *B. dothidea* among the nine species. Putterill in 1919 described apple tree canker and proposed the name *B. mali* because of observed differences from *B. ribis*. Further studies indicated that both fungi were similar and there was no basis for a new species. Birmingham described a canker disease of apple trees caused by *Dothiorella mali* E. & E. that developed the chromogenesis characteristic of *B. ribis chromogena*, the currant cane blight fungus. Inoculations of apple fruit with *D. mali* produced a rot similar to that caused by *B. ribis*. The *B. mali* reported by Putterill and *D. mali* were concluded to be the same. Von Arx and Müller synonymized *B. ribis* with *B. dothidea* in the reclassification of amerosporus Pyrenomyces. *B. dothidea* has been gradually accepted since the reclassification, but now some taxonomists believe it should be called *B. berengeriana*. *D. mali* and *Fusicoccum* sp. are generally accepted imperfect stages of *B. dothidea*. *D. mali* can be distinguished from other *Botryosphaeria* spp. by the macroconidia, which are generally hyaline, nonseptate, and $16\text{--}31 \times 4\text{--}8 \mu\text{m}$. Microconidia are also hyaline and $2\text{--}3 \times 1 \mu\text{m}$. The pycnidia are typically compound. The ascospores of *B. dothidea* are hyaline, ovoid, and $17\text{--}28 \times 7\text{--}21 \mu\text{m}$.

The nomenclature of *B. obtusa* is just as confusing. *Sphaeropsis malorum* was described in 1879 by Peck as the causal organism of black rot and frog-eye leaf spot (Fig. 1). Hesler in 1913 described the fungus on apple twigs and determined it belonged to the genus *Physalospora*. In 1954, von Arx and Müller placed this species in the genus *Botryosphaeria*. The imperfect stage, *S. malorum*, forms uniloculate pycnidia. Conidia are nonseptate at maturity but are melanized, oblong, faintly echinulate, and $20\text{--}26 \times$

$9\text{--}12 \mu\text{m}$. Ascospores of *B. obtusa* are fusiform and $25\text{--}33 \times 7\text{--}12 \mu\text{m}$.

A third *Botryosphaeria* sp., *B. rhodina* (syn. *Physalospora rhodina* (Berk. & Curt.) Cooke), shares this cloudy nomenclature. Pycnidia may be uniloculate or multiloculate. The conidia are oval, melanized and monoseptate at maturity, longitudinally striate, and $18\text{--}30 \times 10\text{--}18 \mu\text{m}$. The conidial state has been reported on many hosts under many names, including *Botryodiplodia theobromae* Pat. (sycamore canker), *Diplodia gossypina* Cke. (cotton boll rot), *D. natalensis* Evans (citrus stem-end rot), *Lasiodiplodia theobromae* (Pat.) Griff. & Maubl. (cashew inflorescence blight), and *L. triflorae* Higgins (plum wilt). The ascigerous state is not easily separated from *B. obtusa*. *B. rhodina* ascospores are hyaline, fusiform, and $24\text{--}42 \times 7\text{--}17 \mu\text{m}$.

For simplicity, we will refer to these species by their perfect stage names, although the ascigerous stages are less frequently encountered.

Isolation of *Botryosphaeria* spp. is not difficult when proper techniques are used. The fungi are heat-sensitive and are killed if the host tissue is flamed. Surface treatment with 0.5% sodium hypochlorite, 10% ethanol, or Radas solution (mercuric chloride) has provided the best isolation results. Incubation under fluorescent lights for 2 weeks is required for sporulation on potato-dextrose agar at room temperature (21–25 C).

Botryosphaeria Canker on Apple

History. *Botryosphaeria* (Bot) canker on apples was of relatively minor importance in the United States until 1952, when *B. ribis* was isolated from severely cankered apple trees in southwestern Indiana. Cankers continued to develop and by midsummer 1953, loss of scaffold limbs was severe. Shay and Sitterly concluded that drought favored development of the disease. In 1956, canker development caused by *B. ribis* was reported in New York orchards that had sustained extensive winter injury.

In 1972, following a severe snowstorm and abnormally low temperatures, *B. dothidea* was detected by Taylor on apple in central Georgia. Most of the damage was associated with frost cracks on

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winter-damaged tissue on the main trunks. Various chemical and paint treatments were initiated. Surveys conducted during the mid-1970s showed a reduced number of trunk cankers, with most active cankers associated with pruning wounds. Since 1977, however, disease severity has greatly increased, possibly because of severe winters and prolonged summer droughts.

B. obtusa causes a minor twig canker on apple in Georgia. Paddock first described *B. obtusa* canker and established that the fungus was the same as that causing black rot of apple fruit. Both *B. dothidea* and *B. obtusa* have increased in incidence over the past decade. Abnormally severe environmental conditions may have contributed to the increased incidence, but many management practices have also changed over this period. Summer pruning and disposal of pruned wood on the orchard floor by mowing may be involved in increased inoculum and disease incidence.

Etiology. *B. dothidea* has been described as both a wound and a nonwound pathogen. Although wounded apple trees have shown a greater incidence of infection and larger cankers, wounding is not necessary for infection (4). The fungus is able to infect through lenticels but does not move into the cortex until moisture stress is applied (6). Infection has been confirmed on cut stems 72 hours after inoculation. Invasion through wounds does not require moisture stress.

Small sunken, reddish lesions on the stem are the first symptoms expressed by infected apple. If infection is associated with a pruning wound, the sunken area is observed first at the margins of the cut. This symptom is associated with the collapse and invasion of the cortical cells by *B. dothidea* mycelium. The bark becomes depressed and blisters often form. The blisters crack, rupturing the epidermis, and sometimes exude a watery substance. The periderm of the bark then peels back (Fig. 2). The blisters are produced by separation of the periderm from the disorganized cortex. Pycnidial initials appear in the periderm and, simultaneously, parenchyma cells rapidly divide. Pycnidia can usually be observed 6 weeks after infection of young, succulent tissue. Amount and duration of

rainfall have been shown to be the most important factors determining the time and amount of inoculum released (13).

Elliptic cankers develop and eventually girdle smaller stems. The presence of mycelium in the xylem vessels but not in the xylem ray cells explains the rapid vertical and slow lateral development of the cankers (4). One limb may be affected or the entire tree may show cankers on the main trunk. The physical presence of the mycelium and tyloses in the vessels may explain the lack of vigor of infected limbs and foliage and the symptoms of nutrient deficiency.

Information on the development of *B. obtusa* cankers is sparse. This fungus, a secondary invader of fire-blighted twigs, is important in the South primarily as a source of inoculum for the fruit rot phase. The trees successfully compartmentalize the fungus. In the Northeast, the delay in warm weather probably permits further invasion of trees low in vigor.

Control strategies. During the mid-1970s, *in vitro* tests showed that benomyl and captafol inhibited growth of *B. dothidea*. Trunks were sprayed with benomyl to protect against infection and painted with white outdoor water-based latex paint to discourage winter and frost damage. These practices were partially effective in reducing disease incidence, but tree decline continued in some areas. Research conducted in the late 1970s showed that five trunk applications of benomyl (2.4 g/L, or 2 lb/100 gal) at 2-week intervals beginning in the spring inhibited growth of *B. dothidea* in existing cankers (5).

Most cankers on mature apple trees are associated with poor pruning techniques (Fig. 3A) or with trunk damage resulting from poor equipment handling or environmental injury (Fig. 3B). Proper pruning techniques are important to ensure rapid callus formation. Late-summer pruning after harvest is receiving more emphasis now, and wounds made at this time of year provide ideal infection courts for *B. dothidea*. In the apple-growing areas hardest hit by *B. dothidea*, most orchard managers now apply benomyl (2.4 g/L, or 2 lb/100 gal) after summer pruning to prevent infection before callus formation.

Starkey and Hendrix showed that flail mowing is an effective alternative to removing prunings from the orchard. This process separates the bark from prunings and facilitates bark decomposition by saprophytes. A rotary mower is not satisfactory.

Until apple cultivars more tolerant of the extreme weather conditions in the Deep South become available, canker diseases caused by *Botryosphaeria* spp. will continue to plague the industry.

White (Bot) Rot of Apple Fruit

History. In 1919, Putterill inoculated apple fruit with an isolate of *B. dothidea*

obtained from cankered apple wood, and fruit rot resulted. Fenner in 1924 made collections from apple-growing areas throughout the United States and demonstrated that *B. dothidea* causes a fruit rot with symptoms similar to those of black rot produced by *B. obtusa*. Severe fruit rot was experienced in 1951 in southwestern Indiana. This widespread disease was concluded to be climate-dependent. Without control measures, losses can reach 100%.

Etiology. Infection of apple fruit by *B. dothidea* usually results in a soft, light-colored rot—thus the common name, white rot (Fig. 4). Sometimes white rot is indistinguishable from black rot caused by *B. obtusa*, and isolation is the only way to differentiate the two diseases. Fruit rot symptoms usually are not observed until midsummer, since infection generally does not occur until 4–6 weeks before harvest (8). Wounding is necessary for fruit infection. Inoculated fruit does not rot until the sugar content reaches approximately 10.5%. We do not know whether sugar content or other concurrent physiological changes such as pH are responsible for increasing the likelihood of infection and enhancing pathogen development. Also, the peak spore production period coincides with this period in fruit development (13).

Control strategies. Benomyl is one of the most effective fungicides for controlling white rot. Season-long benomyl applications at 2-week intervals are effective but costly. Core rot caused by *Alternaria* spp. and spider mite infestations can become problems with this program. Growers in Georgia are now advised not to spray with benomyl until the sugar content approaches 10.5%. In middle Georgia, where white rot is most common, this usually occurs in early July, or about a month before harvest. Control measures recommended for Bot canker should also be followed to reduce inoculum for fruit rot.

Black Rot of Apple Fruit

History. Peck in 1879 demonstrated that black rot of apple fruit was caused by *B. obtusa*. Black rot was a major problem in 1912 in the Southeast and resulted in 25–50% fruit loss. In 1951, black rot caused more fruit loss in Georgia than all other diseases combined. The conclusion of early studies was that a wound was needed for rot to occur, but studies by Taylor in 1955 showed that wounding was not a prerequisite for fruit infection. He concluded that infection by *B. obtusa* could occur from the time of bud break until harvest.

Epidemiology. In Georgia, inoculum is produced on dead apple wood throughout the year. Spores are released during rainy periods when temperatures are above 15 C. Buds become infested in Georgia during December through February (1). When moisture is adequate,

infection may occur as early as the silver-tip stage of bud phenology.

Spore-trap results from North Carolina indicate that spores are released too late to cause blossom infection. Ascospores are present from mid-March through May and conidia, from mid-March through August (13). For this reason, black rot infections occur only after bud break in North Carolina.

In Georgia, a second peak of conidial release occurs in June (1). This inoculum develops on twigs killed by fire blight in the early spring and on mummified apples that have not fallen. Rotted apples can be observed under these twigs in trees that have not been sprayed with fungicides (Fig. 5). The distribution of the pathogen suggests rain-splash dispersal from these twigs, but insects may also carry spores to the tree from prunings on the orchard floor.

Initial symptoms on fruit are brown lesions that, after early season infections, often originate at the calyx end (Fig. 6). As the rot progresses, lesions may turn dark and almost black. Concentric rings may form on mature rotted fruit. The rot

is usually firm and may encompass one-half to two-thirds of the apple. Fruiting bodies may be produced on the apple but often occur after the fruit has begun to dry and mummify. Infected fruit often ripens and drops prematurely from the tree. Secondary infections may result in small, discrete lesions on the fruit at harvest (Fig. 7).

Control strategies. Because of varying climatic conditions, trees in some areas escape bud infections and orchards can be maintained with a fungicide spray program to control later infections. In Georgia, where silver-tip stage infections are important, fungicide applications should begin at bud swell. Captan has been shown to control *B. obtusa*, but fungicides such as dodine and ferbam should be applied in combination with

captan when scab, rust, or mildew is a problem. Because of long residual, captan applied at bud swell may replace up to five applications of captan from early prepink through petal fall.

Sanitation and protective pesticides are the key to black rot control. Fire blight control is important in discouraging *B. obtusa* inoculum buildup. Protective sprays alone cannot take the place of good sanitation practices, and sanitation alone cannot provide satisfactory control.

Peach Tree Fungal Gummosis

History. Fungal gummosis was first noticed in central Georgia in the late 1960s, and in 1974, *B. dothidea* was identified as the causal agent (14). The onset of the peach gummosis epidemic coincided with changing cultural practices for orchard weed control as part of the 10-point program to control peach tree short life. The use of a herbicide in the row with sod middles is now the predominant practice. Pruning wood left on the orchard floor is no longer

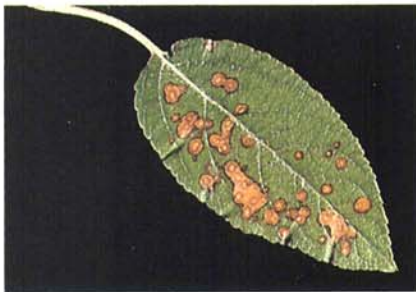


Fig. 1. Frog-eye leaf spot of apple caused by *Botryosphaeria obtusa*.



Fig. 2. Peeling, papery epidermis characteristic of Bot canker of apple caused by *Botryosphaeria dothidea*. Bot canker can cause the entire tree to decline.



Fig. 3. Bot canker of apple associated with (A) poor pruning techniques and (B) winter or mechanical damage.



Fig. 4. White rot of apple caused by *B. dothidea*.



Fig. 5. Twigs of apple tree killed by fire blight and invaded by *B. obtusa*. These cankers provide some of the inoculum for black rot fruit infections.

repeatedly disked into the soil for rapid decomposition but typically is mowed with a bushhog. The bark of the prunings provides ample substrate for sporulation of *Botryosphaeria*.

Surveys of central Georgia conducted from 1980 to 1984 determined that three species of *Botryosphaeria* were present in most gummosis cankers (3). Wound inoculations of healthy trees with *B. obtusa* or *B. rhodina* produced symptoms indistinguishable from those of *B. dothidea* (2). Despite some etiological differences, the major infection court for all three species is pruning wounds. Thus it seems reasonable to consider all three species as causal organisms of the same disease.

Etiology and epidemiology. All three species sporulate on prunings, on dead wood left in the tree, and on the surface of cankers. Any necrotic bark tissue will support the development of pycnidial stromata. Spores of one or more of the three fungi are always present in the orchard.

Weaver (15) showed that *B. dothidea* conidia are present from March through December. This species can penetrate wounds or lenticels. Lenticular infections are most common below existing cankers, areas presumably subject to high levels of inoculum during rain runoff. These infections are often successfully compartmentalized, remaining 1–2 cm in diameter. Most lesions are sunken, but some are raised and contain either gum or callus.

Under field conditions, a seasonal succession of fungi occurs in the cankers: *B. dothidea* predominates in summer and fall and *B. obtusa* predominates in winter and spring (3). Whether this succession is due to climatic preference or to changes in the host physiology is not clear. Monthly isolations from a population of infected lenticels showed the same trend in species dynamics. *B. obtusa* and *B. rhodina* apparently do not penetrate lenticels and probably invade lesions induced by *B. dothidea*.

B. dothidea is not isolated from cankers in January. Whether this species must reinfest the cankers annually is not known. The activities of *B. obtusa* in winter and spring may prevent compartmentalization by extending the season of fungal activity beyond that of vigorous tree growth. If only one fungal species were present, gummosis would perhaps be much less serious in Georgia. In California, *B. dothidea* causes cankers of almond but the pathogen is successfully compartmentalized, possibly because of greater host vigor, other host differences, or the absence of *B. obtusa*.

On peach, the hyphae colonize the stem cortex and are repeatedly walled-off by phellogen formation. The flaking layers of phellem, bound together by mycelium and gum deposits, give old cankers the characteristic swollen and crusty appear-

ance (Fig. 8). Hyphae occasionally penetrate xylem vessel elements, depending on the type of wound invaded, but systemic movement is usually accomplished by growth within the cortex, especially along the vascular cambium and in the area of phloem ray dilation. Vascular discoloration therefore does not extend deep into the wood. Gum exudes from cortical cracks and lenticels (Fig. 9). The cankers have no definite margins, and the fungus can be isolated as much as 30 cm (1 ft) beyond visible symptoms.

In the field, *B. obtusa* sporulates during the winter and conidia infest nearly half of the buds in an infected orchard where inoculum is ample. A low percentage of these buds becomes infected before opening in the spring. Unlike infection with *Cytospora* spp., which readily invade cold-damaged buds, *B. obtusa* infection is not enhanced by cold damage. These bud infections do not significantly increase the amount of fungus present in systemically infected

trees. Because most 1-year-old twigs on severely infected trees already have latent infections, an additional 3–5% infection through buds is not significant. The importance of bud infections in newly planted orchards is now being investigated. Infections through the buds also initiate the latent fruit infections by *B. obtusa* described by Rittenburg and Hendrix (12). Approximately 5–10% of surface-sterilized fruit contain this species soon after bloom, but these fruit abscise by the end of May. A low percentage of fruit may also become infected by *B. dothidea* or *B. rhodina* later in the season and express postharvest symptoms (Fig. 10). Symptoms of these minor fruit rots are similar to those of peach anthracnose caused by *Glomerella cingulata* (Stonem.) Spauld. & Schrenk. The flesh is slightly sunken but remains firm, and the epidermis clings to the peach. At present, these rots do not occur frequently enough to warrant fungicide applications.



Fig. 6. Black rot of apple caused by *B. obtusa*. Lesions often originate at the calyx end.



Fig. 7. Late-season secondary infection with *B. obtusa* produces small, discrete lesions on mature apple fruit.



Fig. 8. Peach tree fungal gummosis cankers, with swollen layers of flaking bark and dried gum exudate.



Fig. 9. Gum exuding from cortical cracks and lenticels on peach tree is first symptom expression of gummosis.



Fig. 10. Lesions resulting from peach fruit rots caused by *Botryosphaeria* spp. are firm, sunken, and usually less than 4 cm in diameter and resemble those of anthracnose caused by *Glomerella cingulata*.

Host-parasite interaction. One poorly understood aspect of peach gummosis is the role the gum reaction plays in disease development. Circumstantial evidence supports two intriguingly opposite interpretations. Recent evaluations of cultivars and selections for resistance to gummosis indicated that cultivars most prone to gum formation were generally colonized less by *B. dothidea* and *B. rhodina*. The converse was true of trees inoculated with *B. obtusa*, which induces very little gum. Pusey et al (10) showed that isolates of *B. dothidea* from central Georgia induced less gum initially, but gumming persisted longer than with isolates from other areas. Reilly and Okie (11) found that trees pruned and inoculated on 15 August produced gum longer than trees pruned and inoculated on 2 March but that reisolation rates were higher in March-pruned than in August-pruned trees. Of the three species present in most cankers, *B. rhodina* induces the most gum and the most extensive necrosis when inoculated on healthy stems. Recently, Copes (7) showed that the total number of spores produced increased with temperature over the range of 15–35 C. Temperatures in Georgia at the time *B. rhodina* spores are released are usually high enough for growth and infection. Yet this aggressive species is much less

common in the field than *B. dothidea* or *B. obtusa*. Does copious gum induction limit the colonization of trees by *B. rhodina*?

These data suggest that levels of fungus colonization are generally lower in trees that gum profusely. This is open to opposing interpretations: 1) Gum production may be a resistance reaction that in some way limits fungus spread and 2) if the loss of carbohydrates through gumming and the ensuing xylem dysfunction result in more tree stress than the direct parasitism of the fungi, lack of gum production may be a tolerance reaction. Resolving this dilemma would greatly simplify the search for resistant cultivars.

Control strategies. Since *Botryosphaeria* spp. sporulate profusely on dead branches left in trees or on the orchard floor, removal of this food base is essential to reducing the inoculum level. Experiments are in progress to determine if flail mowing is as effective for peach wood as for apple wood. Preliminary data indicate that chopping should be done soon after pruning, rather than after harvest, to allow sufficient time for decomposition.

The usefulness of fungicide sprays at bud swell is being evaluated for protection of new plantings. These sprays have been found ineffective in orchards

already heavily infected, where the growth of fungi within the tree keeps pace with tree growth. In inoculation tests (11), benomyl-captan sprays applied immediately after pruning reduced infections. We are currently evaluating postpruning sprays under circumstances of long-term field inoculum pressure.

Increasing plant vigor by reducing stress caused by nutrient and, especially, moisture deficits is expected to reduce disease impact. Experiments designed to test the effects of management practices on disease development are under way.

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