Atropellis Canker of Pines

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Atropellis canker is the general name for a widespread disease of pines caused by four species of the genus *Atropellis* Zeller and Goodding. These cankers (fig. 1) are similar to those caused by some of the rust fungi, but are easily distinguishable by the presence of "blue-stained" wood beneath the affected bark and the absence of bark rupture by aecial blisters. *A. pinicola* (Zeller and Goodding) occurs on four species of soft pines, including eastern and western white pine and sugar pine, and on three hard pines: shore, lodgepole, and Scotch. *A. piniphila* ((Weir) Lohman and Cash) attacks the soft pine *Pinus albicaulis,* but has also been reported on six hard pines including jack, shore, lodgepole, and ponderosa. Similarly, *A. tingens* (Lohman and Cash) is found on eastern white pine and on nine hard pines including jack, pitch, red, slash, and loblolly. It has not been reported on longleaf pine. *A. apiculata* (Lohman, Cash, and Davidson) has been reported only on Virginia pine.

Distribution and Importance

The range and importance of Atropellis canker is indicated by the range and importance of the host tree species.

A. pinicola is most common in the Pacific Northwest and British Columbia, but extends southward into California and eastward into Idaho. Trees of all sizes and ages are affected, but most cankers are found on branches of trees up to 40 feet high. Symmetrical growth of smaller trees is prevented by cankers which kill the leader where it is of small diameter, and trees under 5 inches in diameter at breast height are frequently killed by girdling trunk cankers.

A. piniphila is common throughout the range of lodgepole pine in Alberta and British Columbia, Canada. It occurs on the same host in Idaho, Montana, Oregon, and Washington, and on ponderosa pine in southern Arizona and southern New Mexico. It occurs rarely, if at all, on either host from central Arizona and New Mexico northward through the central Rocky Mountains to the northern limits of ponderosa pine. Trees of all sizes and ages are affected, but mortality is caused by girdling of small stems. Long multiple cankers (fig. 1, *C*) are common on the trunks of infected trees, but branch cankers are infrequent except in northern Alberta where they are common in quite open stands.

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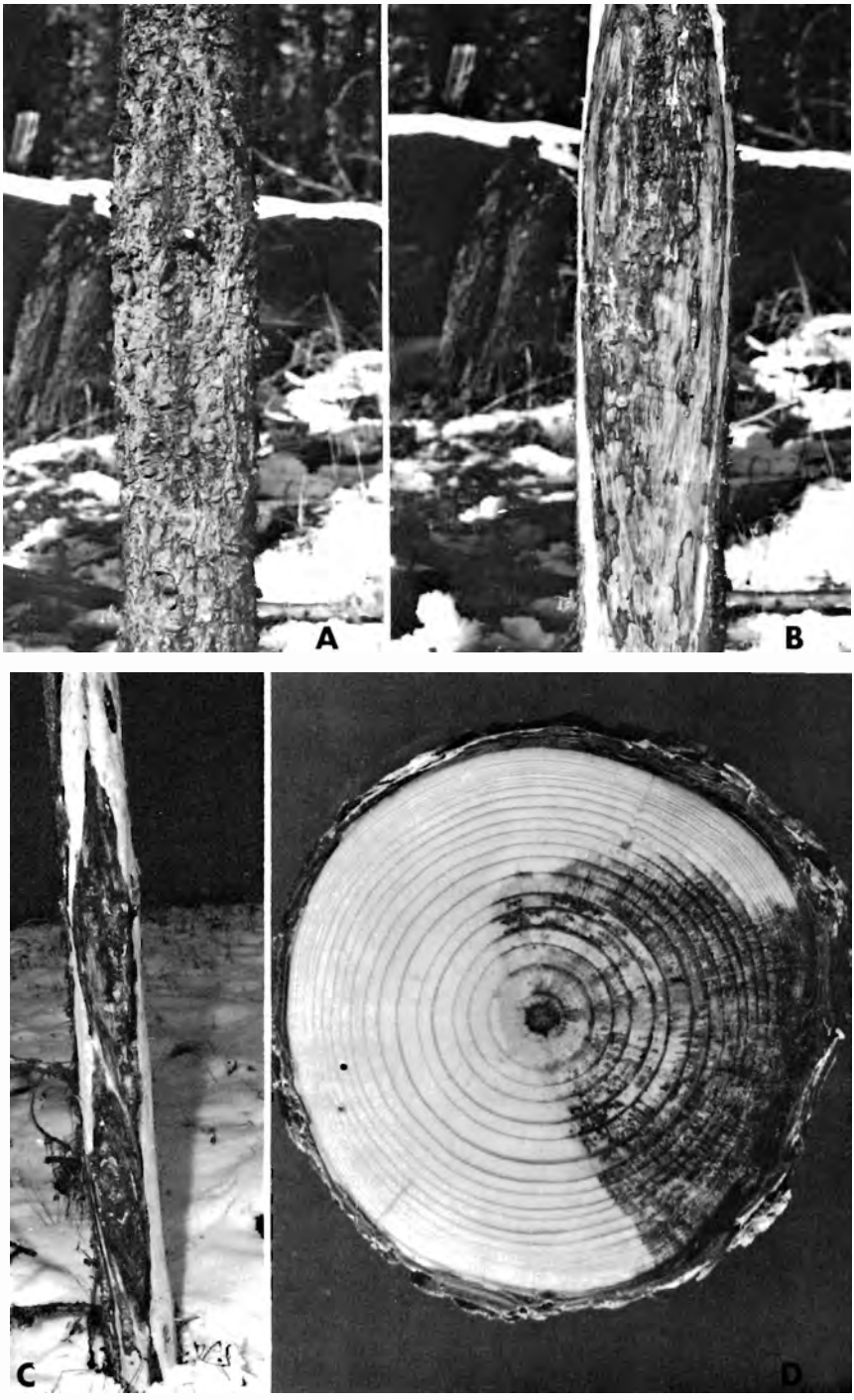

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Figure 1.—*Atropellis piniphila* cankers: (*A-C* on lodgepole pine.) *A*, Canker with bark intact (note dead branch stub in center and bright, unstained wood where branch has been broken off); *B*, canker with bark removed to show extent of stained wood beneath; *C*, multiple cankers with bark removed; *D*, cross-section through old canker on ponderosa pine showing stained wood.

A. tingens is widely distributed on hard pines in the eastern United States from the New England and Lake States southward to the Gulf States. Trees of all sizes and ages are affected and the resulting cankers quickly kill small branches; perennial target-type cankers are formed on the main stem and large branches.

A. apiculata has been reported to be associated with cankers on the main stems, twigs, and small branches of young Virginia pines in North Carolina and Virginia, but its range may be more extensive. Nothing has been recorded on the damage caused by *A. apiculata*, but it is probably similar to that of *A. tingens*.

Life History

Species of *Atropellis*, with the exception of *A. apiculata*, have been studied in enough detail to indicate that their life histories are similar. Although little is known about *A. apiculata*, its development, with minor exceptions, seems to be similar to the others.

During the growing season, spore-bearing structures of two types are produced on the surface of the bark over cankers except those caused by *A. apiculata:* (1) Black, unstalked or short stalked, globose, multiloculate stromata, about 1/32 inch in diameter when fully expanded, in which conidia (asexual spores) are produced, and (2) black, unstalked or very short stalked apothecia (fig. 2), about 3/32 inch in diameter when fully expanded, in which ascospores (sexual spores) are produced. (Only the latter type of fruiting structure

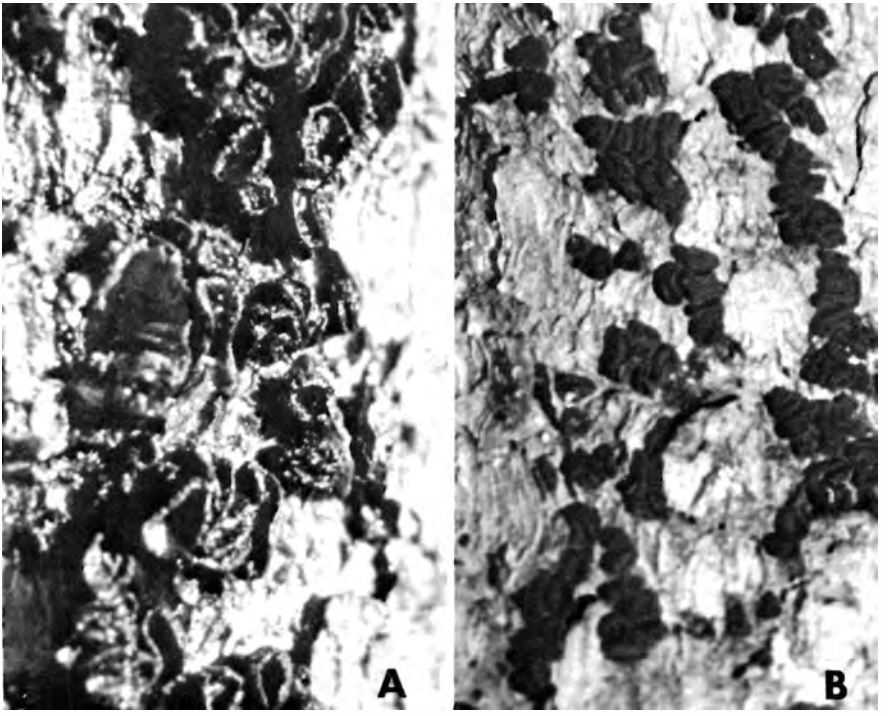

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Figure 2.—Apothecia of *Atropellis piniphila: A,* Apothecia are moist and open (about 4 times natural size); *B,* apothecia are dry and closed (about 2.5 times natural size).

has been reported for *A. apiculata.*)

Conidial stromata appear on infected host bark about the same time as the apothecia. Their exact role has not been determined, but evidence that they precede apothecial formation, and their similarity to structures in related fungi, suggest that they may function as spermatia and may be involved in a sexual role from which ascospores result. Conidial stromata may be mistaken for apothecia during dry weather, but are easily recognized when moist by the creamy, mucilaginous mass of spores which fills the cavities.

Apothecia are cup-shaped when moist and open (fig. 2, *A*) or roughly globular when dry and closed (fig. 2, *B*). They closely resemble similar structures formed by members of the genus *Cenangium,* but may readily be separated from them by a simple chemical test. Apothecia of *A. apiculata* will turn a 5 percent solution of potassium hydroxide chocolate brown; all other species of *Atropellis* show a blue-green reaction. No reaction occurs when the apothecia of *Cenangium* species are similarly treated.

Ascospores are wind-disseminated in summer or early fall during rainy weather. They germinate under favorable conditions of moisture and temperature, and the resulting mycelium penetrates undamaged bark or leaf scars of susceptible hosts. Most infections occur in the axils of branches or twigs (*A. pinicola*) or on the main stem in the region of the nodes (*A. piniphila*); however, infection by *A. tingens* is thought to occur within the needle fascicle or through the base of the needle sheath. Infection does not occur through dead twigs, dead branches or wounds, nor does the fungus grow saprophytically in the dead wood of branches killed by girdling cankers.

Within the host the fungus grows through the bark and wood, staining the latter a dark bluish black (fig. 1, *B, C, D*). Longitudinal growth is essentially the same in both directions, and averages between 1 and 2 inches a year. Circumferential growth, however, is much slower, averaging only about 1/4 inch annually. On ponderosa pine in the Southwest, where cankers 10 feet or more in length are common, a much faster rate of longitudinal extension for *A. piniphila* in indicated. Cankers caused by *A. tingens* enlarge rapidly the first few years, but then slow down and seem to stop after about 10 years. This is not true for cankers caused by *A. pinicola* and *A. piniphila.*

On small, suppressed trees conidial stromata are produced near the center of some 1-year-old stem cankers, but both these and apothecia are found on 2-year-old cankers. On large trees in open stands, stem cankers may be 15 to 25 years old, or older, before either form of fruiting occurs. Stromata continue to form on 2- or 3-year-old infected tissue, throughout the life of the canker, and the apothecia may continue to produce ascospores for a short time after the death of the host.

Symptoms and Signs

The most obvious symptoms of *Atropellis pinicola* are dead branches or "flags" which are often mistaken for blister-rust "flags" on soft pines; similar "flags" on hard pines are often caused by *A. tingens.* These are most conspicuous in spring and early summer. Copious resin flow is associated with *A. piniphila* cankers on lodgepole pine, but not on ponderosa pine.

Cankers caused by all species are similar (fig. 1). Affected tissues become somewhat sunken, but there is not an extreme shriveling nor separation from the surrounding bark. However, longitudinal

splits are common in the bark of cankers caused by *A. piniphila* on lodgepole pine. Cankers caused by *A. pinicola* and *A. piniphila* very frequently have a dead branch or branch stub in the center (fig. 1, *A*), suggesting that infection occurs through the branch. When the dead branches are split, however, it is apparent from the stained wood that infection usually occurs at or near the axil. Similarly, cankers caused by *A. tingens* usually have a dead needle cluster in the center, fixed to the branch with a gob of pitch, suggesting that infection occurs through the needle. However, sectioned needles, examined microscopically, show no hyphae or other indications of the fungus. Thus, infection must occur at or through the base of the needle. Some 1-year-old cankers may show signs of the disease by the presence of conidial stromata, but usually these and the apothecia appear in second and subsequent years.

Discolored wood of young cankers is roughly wedge-shaped in cross-section, with the point of the wedge toward the center, but older cankers lose this character (fig. 1, *D*).

Damage

Except in central and northern Alberta, where infection is common in open stands, dense, stagnated pure stands seem to more frequently provide conditions favorable for infection by *A. pinicola* and *A. piniphila*. Spacing has little effect on the occurrence of cankers caused by *A. tingens*. When the fungus becomes well established, a high percentage of stems may be cankered. The low productivity of stagnated stands is thus amplified if an abundance of *Atropellis* infection is present.

Many trees support multiple cankers (fig. 1, *C*). Single cankers may girdle and kill small suppressed trees, and multiple cankers can kill larger trees, but the impact of mortality on the value of the stand is usually insignificant in overstocked stands.

Malformed stems and discolored, resin-soaked wood make the cankered portion of trees undesirable for lumber. An important form of damage results from the adhesion of infected bark to the underlying wood; this creates debarking problems in the preparation of pulp. Additionally, the resinous wood associated with cankers degrades the pulp, and the blue-black stain adds to bleaching costs. Further degrade results from the abnormal tissues produced by the host. There are large increases in the proportion of bark periderm and xylem parenchyma, while tracheid production is diminished and the tracheids are abnormally short and thin-walled.

Control

No control measures have been developed for Atropellis canker in the eastern United States. In the western United States, control recommendations are designed to minimize initial amounts of inoculum and its subsequent rate of formation. Sources of inoculum have been traced to infected trees not killed by wildfires. Consequently, these fire-residuals should be felled within a few years after the establishment of new reproduction in the burned areas, and adjacent stands of infected unburned trees should be harvested or sanitized by removing as many infected trees as possible. Infected immature stands should be thinned to prevent a high incidence of multiple stem infections.

Widely infected merchantable stands should be clearcut in strips or large blocks. If infection is present only in spots, these pockets need to be clearcut. In either case, all small unmerchantable trees should be destroyed, whether they appear to be infected or not, because

small cankers in the crowns of trees may be easily overlooked.

Infected unmerchantable mature stands should be destroyed, because inoculum is produced very rapidly on stem infections of small, suppressed trees. Such stands constitute a potential hazard to nearby healthy stands. Since incidence is greatest in pure stands, cultural practices to encourage stands of mixed species should be beneficial. Use of non-susceptible tree species to regenerate clearcut patches or strips will isolate diseased from healthy stands and offers possibilities for restricting the disease.

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