19. White Pine Blister Rust

Lee E. Riley and Judith F. Danielson

Hosts

White pine blister rust, caused by the fungal pathogen *Cronartium ribicola*, is an exotic, invasive disease that is native to Asia. It was introduced to both the east and west coasts of the United States in the early 1900s on infected eastern white pine nursery stock imported from Europe.

Blister rust has a complex life cycle that requires alternate hosts for spread of the disease. The life cycle includes five spore forms that most commonly alternate between five-needle pines and currant or gooseberry (*Ribes* spp.) leaves, although Indian paintbrush and snapdragon have recently been determined to serve as alternate hosts as well.

All five-needle pines are susceptible to the fungus, although some species are more susceptible than others. Of the species native to North America, western white, sugar, eastern white, and whitebark pines are most susceptible; limber and southwestern white pines are moderately susceptible; and the susceptibility of bristlecone pine is under investigation. Blister rust is the most important exotic pathogen of forest trees worldwide, and has been particularly devastating to large forested areas and forest stand structure in North America.

Distribution

After introduction from Europe on both coasts of the United States, the pathogen spread throughout the entire range of white pines in North America. Although less of a problem in the East, the disease is found from the Atlantic Provinces of Canada south through Georgia and as far west as Minnesota and Iowa. In Western North America, the pathogen is infecting white pine species from British Columbia and Alberta through the Intermountain West and Pacific Northwest, south through the Rocky Mountain Region, California, and the Southwest, and into Mexico, and is actively continuing its spread in elevation and latitude. It is most severe where conditions are cool and moist for extended periods in late summer and early fall.

Damage

Although young seedlings are more susceptible to infection by *C. ribicola* than older trees, mortality is rare in 1- or 2-year-old nursery stock because of the length of the disease cycle. If spores are present and conditions are optimal, seedlings may become infected during the first growing season. Some mortality may occur in the second year or in any holdover or transplanted stock. Depending on the genotype, mortality is quite likely to occur in infected seedlings following outplanting. Blister rust is a major cause of mortality in regenerating and outplanted five-needle pines, making reestablishment of these species extremely difficult (fig. 19.1).

Figure 19.1—*Recent blister rust mortality on 13-year-old outplanted sugar pine in southwestern Oregon.* Photo by Judith F. Danielson, USDA Forest Service.

Diagnosis

Pines. The infection court is needles; spores send germ tube hyphae through stomata, forming mycelia that grow down into the stem cambium. Within a few months of infection, diagnostic yellow, orange, or red flecks may (or will) appear on the needles, sometimes with necrotic spots in the center (fig. 19.2). After initial infection, the fungal hyphae grow through the needle and into the branch, eventually into the stem of the seedling. The established mycelium forms an orange-brown canker on stem tissue that continues to develop and expand. A slightly swollen or cankered area may form on the seedling branch about 12 to 18 months after the initial infection. This area may be characterized by browning bark with yellowish discoloration at the border of the area, followed by a distinctive spindle-shaped swelling (fig. 19.3). Approximately 1 year following formation of the swelling, pycnial lesions that are characterized by orange to yellow blisters will form (fig. 19.4). These pycnia will eventually produce aeciospores that will infect the alternate host species.

Figure 19.2—*(A) and (B) Needle spots on western white pine seedlings infected with blister rust; (C) infected versus noninfected seedlings.* Photos A and B by Richard Sniezko, USDA Forest Service, photo C by Judith F. Danielson, USDA Forest Service.

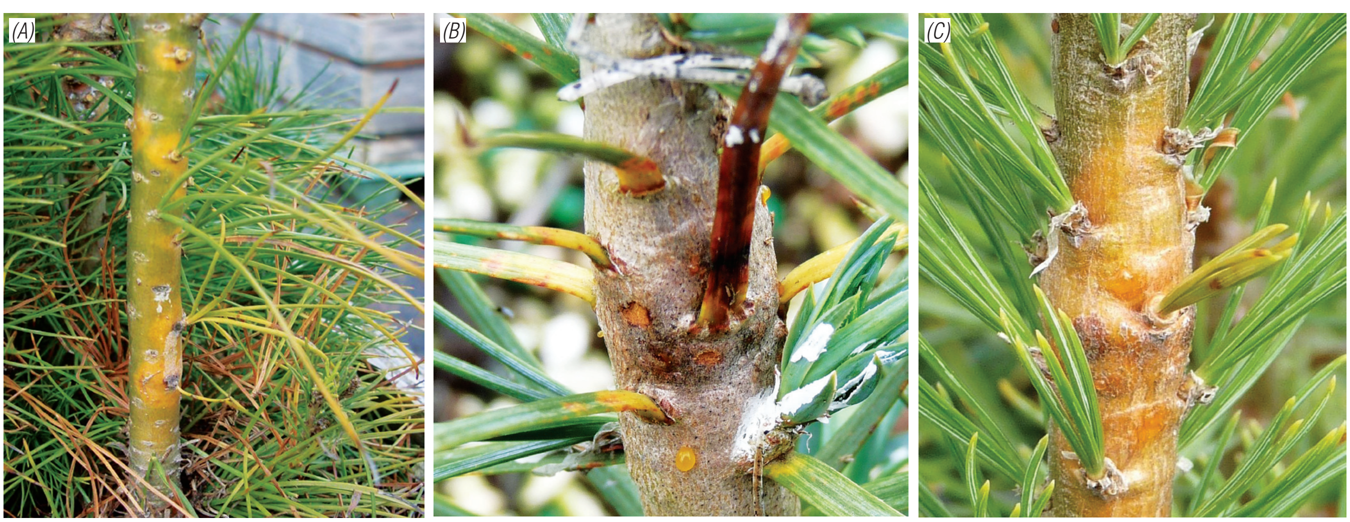

Figure 19.3—*Stem cankers on (A) western white pine, (B) southwestern white pine, and (C) limber pine infected with blister rust.* Photos by Judith F. Danielson, USDA Forest Service.

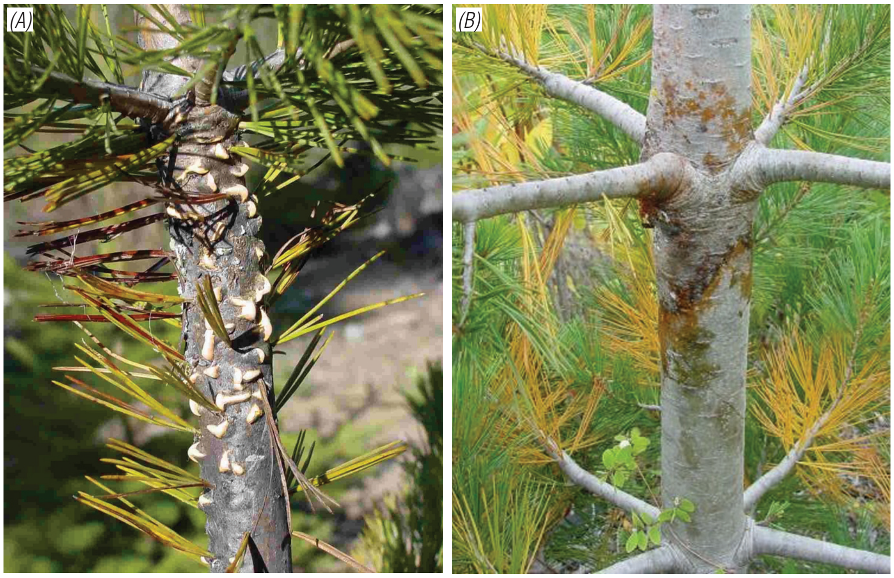

Figure 19.4—*(A) Pycnial colonies on western white pine; (B) active stem canker on western white pine.* Photo A by Richard Sniezko, USDA Forest Service, photo B by Robert Danchok, USDA Forest Service.

Diagnosis of rust infection in the nursery may not be possible. Seedlings should be carefully monitored during lifting and packing, but infection may not be evident until seedlings have been outplanted.

***Ribes* species.** Although resistant varieties of *Ribes* species do exist, most are susceptible to rust infection. In spring, urediniospores can be found on the underside of infected *Ribes* leaves (fig. 19.5). These spores are characterized by yellow to orange blisters, which are seen easily without magnification. These urediniospores will reinfect the *Ribes* leaves throughout the summer, thereby creating a buildup of inoculum. In late summer to early fall, hair-like structures, called telial columns, will form from the old uredinial blisters (fig. 19.6). These structures eventually release basidiospores, the spore form that infects pines under the proper conditions. Infected *Ribes* plants, either in areas surrounding the nursery or on plants grown as a nursery crop, will create a high-hazard or high-risk situation for five-needle pine seedlings growing in the nursery.

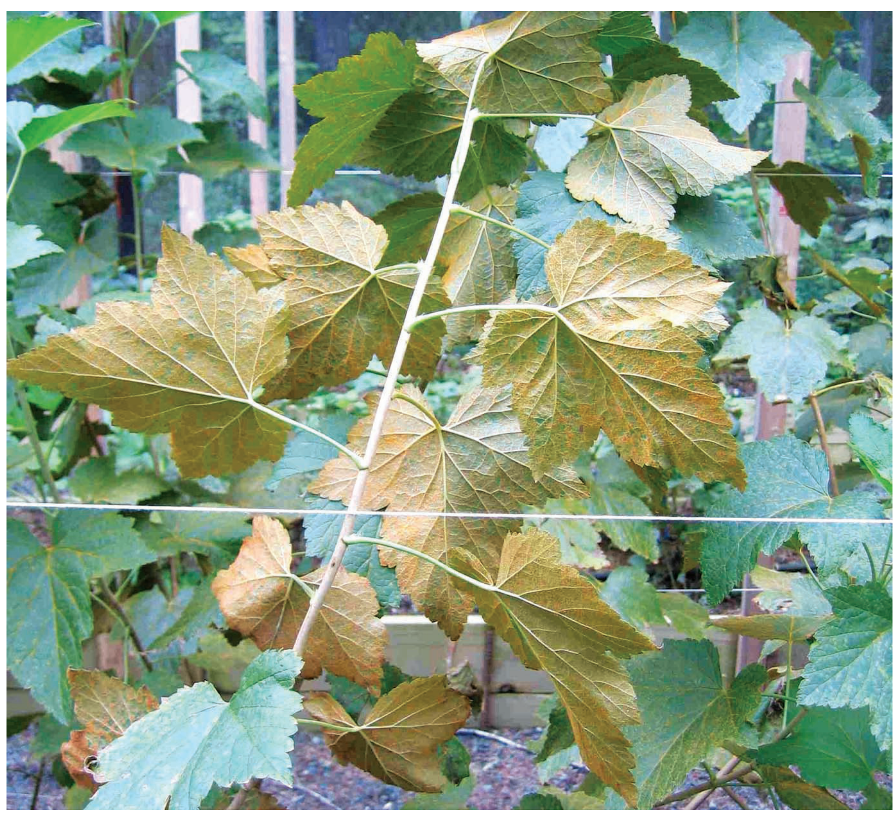

Figure 19.5—Ribes nigrum *infected with* Cronartium ribicola *in August.* Photo by Judith F. Danielson, USDA Forest Service.

Biology

The spread of *C. ribicola* is favored by moist, cool conditions. It is much less of a problem in nurseries located in dry environments. In late summer, basidiospores are released from the infected host plants (most commonly *Ribes* spp.). They can infect pine needles of any age. Spore germination and infection require 48 hours of 100 percent relative humidity at temperatures below 20 °C (68 ºF). During this period, spore germ tubes penetrate the needles through the open stomata.

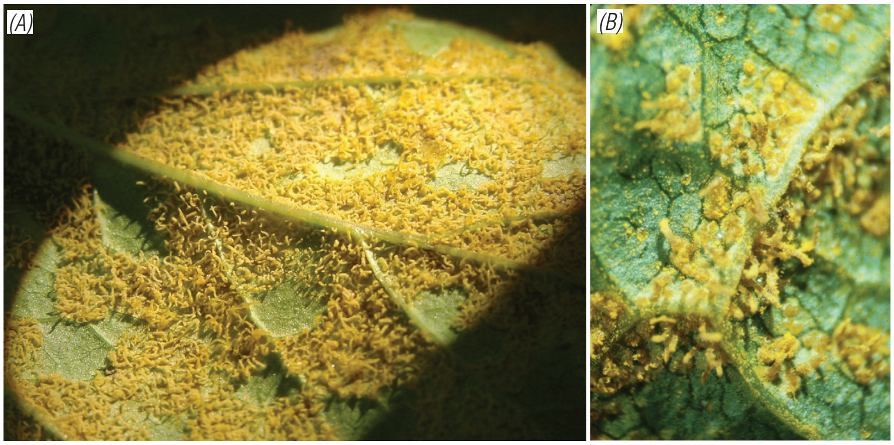

Figure 19.6—*(A) and (B) Telial columns on* Ribes *species leaves.* Photos by Robert Danchok, USDA Forest Service.

During the spring following infection, fungal hyphae grow through the needles and into the branch to form the branch swelling or canker. About 1 year later, pycnia and pycniospores are formed on the canker, which lead to the development of aecia and aeciospores in the same tissue during the following year. Aeciospores are hardy, highly infective, and can be disseminated up to 500 km (300 mi) by wind to infect the alternate host. After the alternate host is infected, urediniospores are produced on the leaves, causing reinfection of the host and a buildup of inoculum.

In late summer to early fall, telial columns are formed on the alternate host leaves, in which the teliospores germinate and produce the basidiospores that infect the pines.

Rust infection can be transferred between *Ribes* during the uredinial stage of the fungus, but rust does not transfer between pines at any stage in the life cycle. The rust is an annual infection on its herbaceous hosts, and is perennial on its pine hosts.

Control

Prevention

Since basidiospores are relatively short-lived and usually infect pines within 100 m (328 ft) of origin, host plant removal in a 300-to-500 m (984-to-1,640 ft) area is recommended to reduce the rust hazard. *Ribes* eradication has been attempted throughout both the Eastern and Western United States where white pines are native. The program resulted in various success rates because one infected *Ribes* plant per hectare can provide the necessary inoculum for a serious disease problem. *Ribes* eradication programs were somewhat successful in the East, but much less so in the West.

The best alternative for disease prevention in the nursery is to sow seeds from resistant genotypes that have been tested in long-term rust resistance breeding programs. Seed orchards of eastern white pine, sugar pine, and western white pine have been developed and are continually being improved by government agencies in Canada and the United States from the survivors of breeding and artificial disease inoculation programs. Seeds from these orchards are producing reforestation stock with higher viability than wild woods run seeds. Because the incidence of resistance is very low in native pine populations, even progeny of these programs may show incomplete or low resistance to blister rust. Resistant genotypes, or genotypes displaying partial resistance, are available for most breeding zones in eastern and western white pines and sugar pines, with research currently underway to produce resistant whitebark, southwestern white, limber, and bristlecone pines. Because of the ability of *C. ribicola* to mutate, it is important to maintain a broad array of resistance mechanisms in outplanting stock, including slow-rusting mechanisms, for balance. Planting of infected seedlings, a majority of nonresistant seedlings, or seedlings of unknown provenance almost completely ensures the eventual failure of the planting.

Cultural

If diagnosis is possible, all infected seedlings should be removed before or during lifting and packing. If high value, older seedlings are infected and infection has not reached the stem of the seedlings, pruning to remove the infection can be moderately effective.

If *Ribes* species are also grown as a crop in the nursery, all plants should be closely monitored and infected seedlings or infected leaves removed as soon as possible.

Chemical

Fungicides in the ethylene-bisdithiocarbamate class of compounds have been found to be effective in controlling rust fungi. Triadimefon, a systemic chemical in the triazole class, has shown some longer term effectiveness in protecting seedlings from white pine blister rust infection.

Selected References

Agrios, G. 1997. Plant pathology, 4th ed. San Diego, CA: Academic Press. 635 p.

Berube, J.A. 1996. Use of triadimefon to control white pine blister rust. Forestry Chronicle. 72: 637–638.

Boyce, J.S. 1961. Forest pathology. New York: McGraw-Hill. 572 p.

Danchok, R.; Sharpe, J.; Bates, K.; Fitzgerald, K.; Kegley, A.; Long, S.; Sniezko, R.; Danielson, J. 2003. Operational manual for white pine blister rust inoculation at Dorena Genetic Resource Center. [Unpublished document]. Located at: Cottage Grove, OR: USDA Forest Service, Dorena Genetic Resource Center.

Goheen, D.; Goheen, E. 2000. Forest insects and diseases: Natural Resources Institute. [Unpublished document]. Located at: Central Point, OR: USDA Forest Service, Forest Health Protection, Pacific Northwest Region, and the Dorena Genetic Resource Center.

Hunt, R. 1997. White pine blister rust. In: Hansen, E.M.; Lewis, K.J., eds. Compendium of conifer diseases. St. Paul, MN: American Phytopathological Society: 26–27.

McDonald, G.I.; Hoff, R.J. 2001. Blister rust: an introduced plague. In: Tomback, D.F.; Arno, S.F.; Keane, R.E., eds. Whitebark pine communities: ecology and restoration. 193–220.

Patton, R.F.; Cordell, C.E. 1989. White pine blister rust. In: Cordell, C.E.; Anderson, R.L.; Hoffard, W.H.; Landis, T.D.; Smith, Jr., R.S.; Toko, H.V., tech. coords. Forest nursery pests. Agriculture Handbook 680. Washington, DC: USDA Forest Service: 75–77.