

## **After a century, white pine blister rust still a factor**

**By Ellen and Don Goheen**

The white pine blister rust story is an old story in forest pathology. In western North America, it is nearing its 100th anniversary, yet it is still a very important story. The pathogen is well established in areas where it has long been resident, it still causes substantial damage and mortality on high risk sites, and it continues to spread into new environments where, in some cases, it has found new hosts.

*Cronartium ribicola*, cause of white pine blister rust, is native to Asia and had spread to become widespread in Europe by the beginning of the 20th century. It was first described on eastern white pine (*Pinus strobus*) in the Baltic provinces of Russia in 1854. The fungus was introduced into both eastern and western North America on diseased European planting stock around the beginning of the 20th century. In the West, introduction resulted from one shipment of infected eastern white pine seedlings shipped to Vancouver, British Columbia, from France in 1910. The pathogen became established in the wild, but was not recognized until the 1920s by which time it had already spread throughout the western white pine region and infected sugar pine in southern Oregon. Its forays into higher elevations occurred slightly later and the fungus is still on the move with relatively recent establishment in southwestern white pine in New Mexico, limber pine in Wyoming and Colorado, and within the last few years, discovery of infection in Rocky mountain bristlecone pine in southern Colorado.

White pine blister rust is a disease of five-needle pines, white pines in the subgenus *Strobus*, including the northwestern species whitebark pine (*Pinus albicaulis*), western white pine (*P. monticola*), sugar pine (*P. lambertiana*) and limber pine (*P. flexilis*).

Sugar and western white pines are extremely valuable timber trees. They also have significant scenic, wildlife and watershed values. They possess a number of unique virtues including high growth potential, ability to reach great ages and sizes, frost hardiness and resistance to root disease. Whitebark pines are keystone species in the high elevations. It and limber pine (which occurs more extensively in more arid high elevation sites in the interior west) provide important food for wildlife, are critical to watershed integrity, and are incredibly valuable aesthetically. All of the five-needle pines contribute substantially to ecological diversity. Alternate hosts of the fungus are currants and gooseberries in the genus *Ribes* and species of *Pedicularis* and *Castilleja*.

On infected pines, white pine blister rust causes formation of resinous lesions, eventually girdling the host at the point of infection. This results in branch and top mortality of large trees, and, in the case of main stem infections on smaller trees, entire tree death. Large infected trees that are not killed immediately by the fungus itself may be predisposed to infestation by mountain pine beetle (*Dendroctonus ponderosae*) as a result of infection. Heavy infection of the *Ribes* hosts may cause defoliation. Impacts to other alternate hosts are yet undetermined.

*Cronartium ribicola* has a complex life cycle involving five spore types and requiring both pine and *Ribes* (or other alternate) hosts for its successful completion. Basidiospores of *C. ribicola* infect pine hosts during summer

and fall. Infection takes place through needles of any age. The relatively delicate, short-lived basidiospores are wind dispersed, generally infecting hosts within 100 yards, but capable of being moved longer distances in clouds and fog. For successful spore germination and infection of pine needles to occur, there must be 48 hours with 100 percent relative humidity and temperatures not exceeding 68 degrees F. Following germination and successful penetration, a sparse mycelium develops and grows from the needle into the bark of the stem. Twelve to 18 months later, a slightly swollen, cankered area first becomes visible. Two to three years after initial infection, pycnia and pycniospores are produced on the cankers. They are noninfective and have a sexual function. After an additional one to two years, in the spring, aecia with aeciospores are produced in the same location on the cankers. The relatively tough aeciospores are wind disseminated over considerable distances and infect leaves of alternate hosts. Infection of the alternate hosts is favored by moist conditions. Two weeks after initial infection, uredinia are produced on leaves. Urediniospores produced from the uredinia reinfect *Ribes* throughout the summer causing buildup of inoculum. In late summer to early fall, hairlike telial columns emerge from the old uredial pustules. Teliospores germinate in place on these columns and produce basidiospores, starting the process over again. The entire life cycle requires three to six years for completion.

The initial spread of white pine blister rust in North American forests was phenomenally rapid. The rapidity of spread was due to the combination of numerous highly susceptible hosts, the close proximity of primary and alternate hosts, the favorable environmental conditions that prevail here, and the fact that *C. ribicola* spores are windborne. Aeciospores, in particular, are effectively transported very long distances, sometimes up to 300 miles. Spread is episodic and is much more dramatic during years with moist summers and falls. The disease is often not as severe in dry microsites as in moist ones.

Losses caused by white pine blister rust have been exceptionally great. For example, western white pine was once the dominant species on five million acres in the Inland Northwest. It is no longer. Also, it is believed that five-needle pine crown cover has been reduced by at least 50 percent in Southwest Oregon. Economic loss has occurred as reduced value in salvaged mortality, unsalvaged mortality in mature stands, loss of site potential due to killing of immature trees, and value loss through top killing. Ecological losses have never been well evaluated, but have been substantial. Forest pathologists throughout the West have identified the decline of five-needle pines to be a critical concern.

Nature has provided some natural controls for white pine blister rust. A native fungus, *Tuberculina maxima* can be found parasitizing blister rust-infected tissue, stopping or slowing canker growth. Even rodents do their share to control the disease, chewing on the sugar-laden infected tissue and girdling the cankers. Unfortunately, these controls will never be enough to truly slow the pathogen down. Heroic and costly historic efforts aimed at eradicating *Ribes* or killing cankers on infected trees with chemicals were well intentioned, but singularly unsuccessful in the West.

The most effective approach to control white pine blister rust involves planting pines with various levels of resistance to *C. ribicola*. Programs to identify and screen apparently resistant pines or to breed trees for increased resistance show great promise though *C. ribicola's* ability to mutate means that precautions to maintain as many kinds of resistance mechanisms as possible in the resistance breeding efforts are necessary. Adding value to resistant stock by pruning in young stands may be worthwhile to: 1) remove cankered branches before the fungus reaches the main stem; and 2) modify the microclimate in the lower crowns.

White pine blister rust is a permanent resident in North America and many efforts are in place to facilitate the survival of white pine species in the presence of the disease and minimizing its ecological, economic and aesthetic impacts. These efforts are focused on restoring white pines where they have been lost, sustaining white pines in the presence of disease, and planning mitigating actions when the rust spreads into new areas. However, there are many challenges facing land managers interested in managing white pines. These include the loss of potentially resistant trees to bark beetles and fire, the lack of planting opportunities, reluctance to plant white pines because of concerns about survivability, the presence of the disease in land areas with allocations where traditional silvicultural approaches to management are controversial, and the potential impacts of climate change, particularly in high-elevation forests. That a pathogen requiring more than one host to complete its life cycle, with five spore stages, and very strict environmental requirements for portions of its life cycle remains so successful even after nearly 100 years should, at the very least, continue to increase our concerns regarding the potential impacts related to the introduction of non-native pathogens to our forests.

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