TREE NOTES

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White Pine Blister Rust in California

John Kliejunas[1] and David Adams[2]

[1]USDA Forest Service, Pacific Southwest Region, Vallejo, CA 94592; [2]California Department of Forestry, Retired, Davis, CA

Background

White pine blister rust, caused by the fungus *Cronartium ribicola*, is a disease of 5-needle pines (white pines) and its alternate host *Ribes* (currants and gooseberries). The fungus is an invasive, exotic pathogen native to Eurasia. Exotic forest pests like *C. ribicola* can have profound effects on forest ecosystems and continue to have significant economic effects long after their arrival.

The rust was introduced into Vancouver, British Columbia in 1910 on a shipment of eastern white pine (*Pinus strobus*) nursery stock grown in and shipped from France. The native *Ribes* became infected, leading to permanent establishment of the disease. By 1929, the pathogen had spread 250 miles southward to the California border where it was infecting sugar pine at one location on the Six Rivers National Forest and at one location along the east fork of Indian Creek on the Klamath National Forest. In the next 15 years, the rust spread another 250 miles south through the Sierra Nevada to Dodge Ridge on the Stanislaus National Forest. The known southern limit remained at Dodge Ridge until 1968, when the location of the rust was extended 150 miles south to the Mountain Home Demonstration State Forest area in Tulare County. The newly found infection centers there had originated in 1961 and intensified in 1964 and 1967. Additional surveys in 1970 found three small centers in the Tyler Meadow area of the Greenhorn Mountains, Sequoia National Forest, and intensification of the rust in Mountain Home Demonstration State Forest and areas of the Sequoia-Kings Canyon National Park, and the first infection center on the Sierra National Forest.

The known southern extent of white pine blister rust in California had been at the southern end of the Sierra Nevada at the head of the Bear Creek drainage, just north of Evans Flat, on the Greenhorn Ranger District, Sequoia National Forest for a number of years. Sugar pine in the Breckenridge Mountain area, at the southern tip of the Sierra Nevada, were reported infected in 2002 (Fig. 1).

Blister rust is now prevalent throughout many of the sugar pine and high elevation white pine stands of northern California, but has not yet been reported in the Tehachapi Mountains or south.

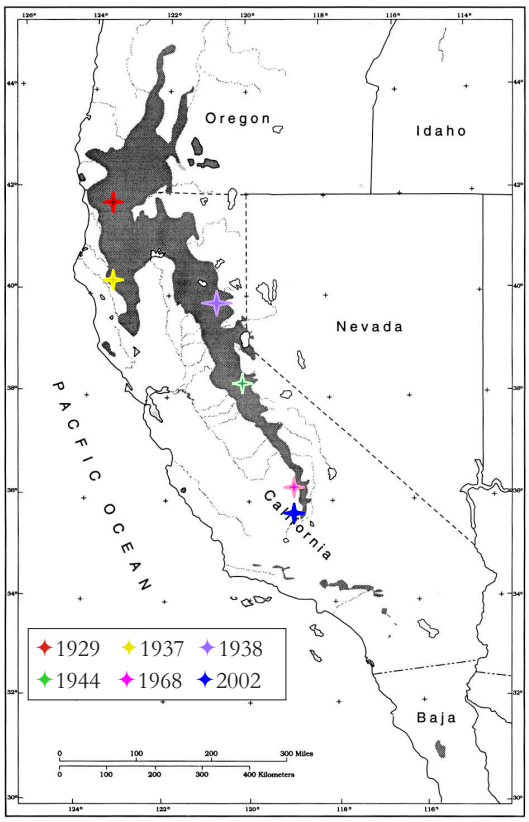

Figure 1. Spread of white pine blister rust in California. The shaded area depicts the range of sugar pine.

The pathogen has not yet stabilized geographically, and continues to spread and intensify in California and elsewhere.

Disease Cycle

Cronartium ribicola is an obligate parasite (a parasite that needs a living host to survive) that attacks all native North American white pines. In California, sugar pine (*Pinus lambertiana*), western white pine (*P. monticola*),

whitebark pine (*P. albicaulis*), limber pine (*P. flexilis*), bristlecone pine (*P. longaeva*), foxtail pine (*P. balfouriana*), and all species of *Ribes* are potential hosts. The fungus needs two hosts to survive, spending part of its life on 5-needled pines and the other on *Ribes*.

The disease cycle, which takes 3 or more years to complete, is presented in Figure 2.

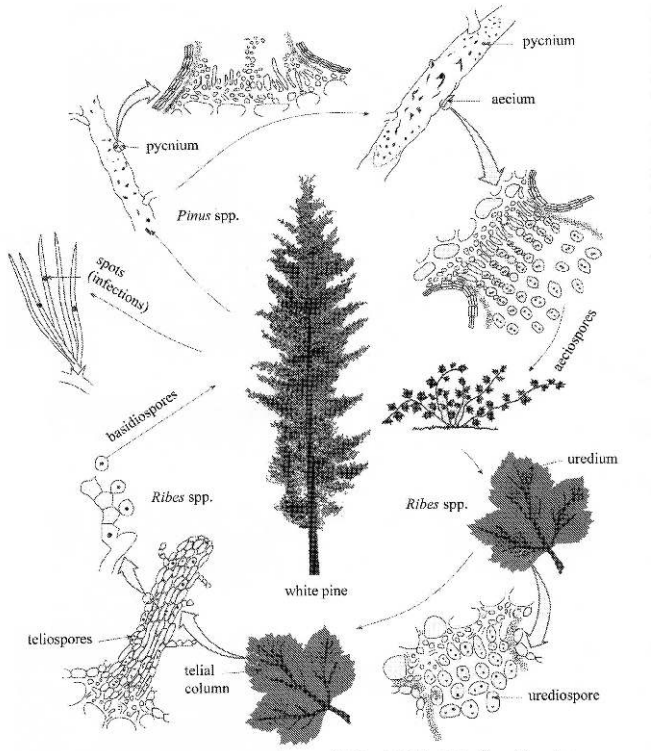

Figure 2. Disease cycle.

The rust has five spore stages, three of which are important to its spread. Thick-walled spores (aeciospores) produced by the fungus in the spring on pine bole or branch cankers are wind-disseminated to *Ribes* where they infect the leaves. Spores (urediospores) produced in orange pustules on the underside of the leaves re-infect *Ribes* throughout the summer, resulting in an intensification of the rust. In the late summer and early fall, telial columns consisting of teliospores are formed on *Ribes* leaves. The teliospores germinate in place to produce thin-walled, delicate basidiospores, which are wind-disseminated to pines and infect current year needles through stomata. Following pine infection, the fungus grows from the needle into the cambial tissue of the branch and forms a canker. After 2 to 3 years, pycnia appear as small blisters on the bark. The pycnia discharge small droplets of honey-colored liquid containing pycniospores. Aecia containing aeciospores are then produced on the cankers and are spread to *Ribes* to continue the cycle.

Although blister rust may spread hundreds of miles from pines to *Ribes*, its spread from *Ribes* back to pines is usually limited to a few hundred feet due to the fragility of the basidiospore. Blister rust is perennial on white pines and annual on the deciduous foliage of *Ribes*.

Environmental conditions are critical for successful infection and limit disease establishment in most years. However, the perennial nature of the fungus on white pines affords infection of *Ribes* every year. Abundant moisture and cool temperatures favor infection of both hosts, and must coincide with spore dispersal for infection to occur. At least 48 hours of 100% humidity and a temperature near 20°C (68°F) are needed for infection of pines. In California, the sites where these conditions frequently occur are usually in cool moist areas such as stream bottoms or around meadows. When these periods of favorable environmental conditions occur, high incidence of infection can result (wave years). On the average, wave years have occurred in northern and central California at approximately ten-year intervals. In areas less favorable for rust, the frequency of wave years decreases.

Symptoms and Signs

Infection of pines may result in branch mortality, top kill, and tree mortality. Cankers develop on branches. Active branch cankers enlarge annually as the fungus invades additional tissues and moves toward the bole. Infections girdle and kill the branches, resulting in bright red "flags" in the crown (Figs. 3, 4). These are the visible symptoms of white pine blister rust infection. Branch cankers within 61 cm (24 inches) of the bole will eventually reach the bole and form cankers.

Figures 3 and 4. Branch flagging.

The signs of infection on white pines and *Ribes* leaves are distinctive.

A honey-colored ooze consisting of pycnia (Fig. 5), then pustules of powdery orange spores (aecia), develop on the cankers in the spring (Fig. 6).

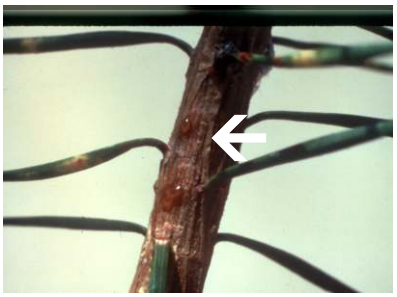

Figure 5. Pycnia.

Figure 6. Aecia.

Trunk cankers are often diamond-shaped, with dead roughened bark and greenish-yellow to orange margins (Fig. 7).

Figure 7. Trunk canker.

As the disease develops, cankers will have dead centers surrounded by a series of zones containing pustules of aecia, drops of pycnia, and a yellowish zone of discoloration. Bole cankers that girdle the stem kill the tree above the canker.

On *Ribes*, uredia appear in the spring as slightly raised yellow spots on the lower side of the leaves, petioles, and young stems (Fig. 8). During late summer and early fall, golden-brown, hairlike, telial columns up to 2 cm (0.8 inches) long appear on the underside of the leaf (Fig. 9). These columns bear microscopic spores (basidiospores) that infect white pine needles in the late fall and early winter—an ideal time for infection of white pines.

Figure 8. Uredia.

Figure 9. Telia.

Impact

Economic impact has been considerable on sugar pine and western white pine. Both species are major timber species and key components of mixed conifer forests. Infection of seedlings and saplings results in direct mortality. Infection of larger trees may result in additional stress, resulting in bark beetle attack and subsequent mortality. Ecological damage has been severe on high elevation species, including whitebark and limber pines.

Management Considerations

Various strategies are available to the resource manager to reduce or mitigate current and potential impacts of white pine blister rust on the white pine resource. The presence of the disease, or potential for the disease, does not preclude the management of 5-needle pines to meet recreational, esthetic, wood production, or other objectives.

Ribes Eradication

Early blister rust control efforts involved extensive, and costly, eradication of *Ribes*. Dense populations of *Ribes* increase the probability of having a high production of basidiospores and infection of local pine populations. Removal of nearby *Ribes* may reduce the incidence of disease on pine trees, but is not usually feasible or effective over large areas. Reducing *Ribes* to a few bushes per acre may be effective in limiting rust infection. The unsuccessful efforts were discontinued in the 1960s. The emphasis shifted from direct control to genetic screening of planting stock for rust resistance.

Rust Hazard Zones

Although a hazard-rating system developed in the Intermountain area for western white pine may be useful there, no system exists in California for reliably estimating the probability and severity of blister rust infection. In the 1970s, the rust in the southern Sierra Nevada moved rapidly into and intensified in upland sites. The rust there occurs much higher in trees than observed in the northern and central Sierra Nevada.

Pruning

The majority of infections occur on pines within a few feet of the ground, presumably because of less air movement and higher relative humidity that facilitates spore germination and infection. Pruning of lower limbs of eastern white pine and western white pine has been used as a treatment for reducing white pine blister rust infections. However, this practice is not used in California. Infections often occur high in the crowns, especially in the southern Sierra Nevada. Pruning has not been successful at Mountain Home or

at LaTour Demonstration State Forests. Crop trees pruned to 18 feet in natural stands in the southern Sierra Nevada had slightly more rust infection than untreated trees following wave year infections.

Removal of branch cankers before they develop into bole cankers can increase the probability of tree survival. Surveys of mixed conifer stands in the northern Sierra Nevada indicated that pruning is biologically feasible for increasing tree survival where rust levels are not excessive. Pruning to a height of 18 feet (5.5 meters), removing no more than one-half of the tree's crown, was recommended. Pruning trees with cankers within 4 inches (10.2 cm) of the bole is unlikely to benefit the tree and is not recommended.

Disease Resistance (Genetic Control)

The selection and improvement of rust-resistant pines is the primary means of managing the disease. Because this fungus is an invasive species, native trees have not evolved with it and few white pines are resistant to infection. There is also evidence that different strains of *Cronartium* exist, some with the ability to overcome natural pine resistance. Major gene resistance (MGR), inherited by a single dominant gene, triggers a hypersensitive reaction when leaf tissues are invaded by the pathogen. MGR to *C. ribicola* attack has been found in a small percentage of sugar pine, western white pine, and southwestern white pine. Rust capable of neutralizing this gene exists in some blister rust populations at low but varying frequencies. Two races that specifically neutralize MGR in sugar pine and western white pine have been confirmed. Both races appear to have limited distributions. MGR is probably not a useful form of long-term resistance in the presence of a sexually reproducing pathogen, as *C. ribicola* is.

In addition to MGR, two other types of resistance exist; slow rusting, and ontogenetic. Slow rusting resistance (SRR) is expressed by lower infection frequency and pathogen rate of growth, and by bark reactions that abort infections after they establish in stem tissues. Slow rusting may be useful if sufficient genetic diversity can be found within parental lines containing that form of resistance. Ontogenetic resistance (OGR) is genotype specific and confers resistance to some adult trees that were not exposed to the disease when they were young. Ontogenetic resistance may be useful if trees can be kept free or relatively free of the disease until the resistance kicks in; that may be 20 years or more.

A strategy and program for the development of rust-resistant sugar pine has been developed in California. The objective of the program is to identify forms of resistance that will have long-term effectiveness and can be incorporated in a sufficient number of individuals to preserve sugar pine's genetic diversity. The program is expanding to include pine species in addition to sugar pine. The challenge for breeders, conservationists, and silviculturists is how to deploy genes for resistance throughout the natural ranges of affected white pine species. The program critically depends on the availability and identification of resistant trees in all the various seed zones of 5-needle pines in California regions.

References (Additional Reading)

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Jesse Rios: Editor
CDF Forest Pest Management (916) 653-9476