

Forest Pathology for the Last Century: A Retrospective and Directions for the Future

White Pine Blister Rust in North America: Past and Prognosis

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ABSTRACT

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After a full century in North America, the blister rust epidemic has yet to stabilize, continuing to spread into warmer and drier areas previously considered climatically inhospitable. The disease apparently has no environmental limits wherever white pines and *Ribes* spp. cohabit and will eventually become pandemic. Although much timber value has been lost, more severe long-term damage is disruption caused to ecosystems by altered patterns of natural succession. During the last half of the century just past, development of genetic resistance superceded other

direct control measures—mainly *Ribes* spp. eradication and antibiotics—which proved ineffective and/or unfeasible in large areas of the white pine range, especially in the West. Several mechanisms of complete (major gene) and partial resistance are common to at least several white pine species. Although North American populations of rust have low genetic variability overall, rust genotypes with specific virulence to major resistance genes exist in some local demes at high frequencies. The challenge will be to package and deploy resistance genes in ways that will dampen sudden increases in rust races of wide virulence. New introductions of blister rust from its gene center in Asia remain the gravest threat to genetic improvement programs.

North America is endowed with the world's richest diversity of white pines: nine different species occupy most areas of the continent, from sea level to the subalpine zone. All but one species are in the West, but this one, eastern white pine (*Pinus strobus* L.), has the widest distribution. The wood quality and timber value of eastern, western white (*P. monticola* Dougl.), and sugar (*P. lambertiana* Dougl.) pines remain unsurpassed, but in recent decades other values have been increasingly emphasized. In some large geographic provinces, such as higher elevations of the Great Basin, limber pine (*P. flexilis* James) is the only existing tree. All high elevation species provide critical watershed protection and some, such as whitebark (*P. albicaulis* Engelm.) and limber pines, can be key species for wildlife. White pines include the oldest (*P. longaeva* DK Bailey) and some of the most picturesque trees on earth.

All North American white pines are susceptible to blister rust. Most species have been severely damaged in at least parts of their native ranges, as well as where they have been planted as exotics (notably, western Europe, and Japan). As a result of decades of epidemic conditions, there is virtually no seed source of eastern white pine remaining for reforestation at the northern range limit in Ontario and the Maritime Provinces of Canada. In the West, western white pine growing stock has been reduced by 95%. Infection levels of whitebark and limber pines range from 70 to 100% in large parts of their northern range, leaving ghost forests of weather-bleached snags. Sugar pine, the largest of all the pines, has also sustained very heavy damage in many places in the central Cascades and Sierra Nevada. But the most insidious impact of the disease on all white pine species is the destruction of regeneration, because this alters natural succession.

The epidemic. History. The progress of the early epidemic has been thoroughly described by Spaulding (22) for eastern North America, and for the west by Mielke (19) and Smith (21). A brief outline will suffice here.

The first introduction of blister rust was in the East around 1898, followed soon by several others. By 1909 it had established in several states as far West as central Pennsylvania, and by 1919 it had reached north to Ontario and Quebec and west to the natural limit of eastern white pine in Minnesota. In the West, blister rust spread from a single point introduction from a nursery on Vancouver Island, B.C., south through the Cascades, and east into the northern Rocky Mountains on western white, limber, and whitebark pines. Sugar pine was heavily attacked in the central Cascades, but it was thought that the disease might be checked once it reached the warmer, drier climate of the Sierra Nevada. This proved a vain hope; although the rate of spread slowed somewhat and displayed a more patchy distribution, blister rust reached the southern Sierra by 1961. Where established, the disease showed that it could intensify and be just as destructive as anywhere further north. More recently, blister rust was discovered on southwestern white pine (*P. strobiformis* Engelm.) growing on isolated tops of the Sacramento Mountains of New Mexico, over 600 miles from the nearest possible source of inoculum in the Sierra Nevada (8). This epidemic, though localized so far, has developed with particularly savage intensity. Another remote outbreak was reported in South Dakota (4).

So far, blister rust has not been detected on sugar pine in the Transverse or Peninsula Ranges of Southern California and Baja California, or on Mexican white pine (*P. ayacahuite* Ehrenb.) in mainland Mexico, even though alternate host *Ribes* spp. are abundant. Nor has it reached the high elevation stands of bristlecone pine (*P. longaeva* DK Bailey) in the White Mountains of California, even though some of these stands support under stories of pure *R. cereum*. However, the record to date provides no assurance that any North American climate where alternate hosts grow will preclude blister rust from becoming established.

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Dynamic. Blister rust, caused by *Cronartium ribicola* J.C. Fisch., is a cool weather disease, endemic on white pines of eastern Asia that occupy relatively high latitudes and elevations, such as *P. sibirica* Du Tour and *P. pumila* Regel. Its complex life cycle, involving five spore stages and alternating between hosts of white pines and *Ribes* spp. (as well as *Pedicularis* spp. in Asia), has exacting environmental requirements for completion. Foremost of these are prolonged cool, moist conditions of at least 2 days to produce, disseminate, germinate, and establish basidiospores on pines. Because of their vulnerability to desiccation and sunlight, these delicate spores are usually transported only short distances (up to 900 feet has been the accepted dogma). However, under appropriate conditions, basidiospores can be carried by moist air masses much further and over wide areas in a single episode (23). Few involved with this disease for any length of time have not come across cases of considerable pine infection with no *Ribes* spp. in sight. These conditions are variable and only partially understood, but can contribute to the notorious “wave year.” Wave years largely determine the rate and intensity of the epidemic from region to region. The probability of wave year occurrence diminishes as the climate becomes warmer and drier.

A different dynamic operates on the asexual side of the cycle. Thick-walled aeciospores are adapted to resist desiccation, and so are capable of long-distance transport. A new blister rust colony can start from an individual founder spore infecting a *Ribes* bush, which can then propagate clonally by vegetative urediniospores on neighboring bushes. If subsequent environmental conditions favor pine infection, the colony becomes securely established as perennial mycelium in pine tissues; if not, it becomes extinct. Thus, the epidemic does not necessarily progress in a uniform front, but rather in a patchwork pattern over the landscape. Local spread and intensification on white pines progresses more or less concentrically and often relatively slowly, somewhat analogous to spread of root rot from infection foci. An important genetic consequence of this pattern is a fragmented, metapopulation structure driven by drift. Evidence from both virulence and molecular marker loci show that neighboring rust demes often exhibit more genetic distance from each other than those that are more geographically distant (16). This structure is consistent with the life cycle of a pathogen that occasionally establishes colonies from a long distance with single or few founder spores that reproduce clonally and are then followed by sexual spores with limited distribution.

Control. *Ribes* eradication. Benedict (1) has written an engaging and detailed account of early control efforts, of which space here allows only brief summary. These were focused on breaking the disease cycle by alternate host eradication. To be effective, removal had to be thorough, permanent, and over entire landscapes, no matter how remote and steep. With many *Ribes* spp. widely spread over the continent wherever white pines grew, this task amounted to ecological warfare on a biblical scale. Of course, disease suppression was only half of the motivation; thousands of government jobs were created to feed the hungry and unemployed during the Great Depression. In the end, the war was lost to superior enemy fitness—at least it was in the West, where the problem was greatest. The *Ribes* bushes were too numerous, too large, and too well adapted. They sprout from dormant seed in the duff and from roots not thoroughly removed. The operation was premised on the perception, with some empirical evidence to support it, that only *Ribes* spp. growing near white pines posed a hazard; few had anticipated the devastating effect that wave years could have over large areas, even on those that had been treated. The terrain was too steep and the areas requiring treatment were too remote to sustain the enormous logistical effort needed.

In the East, operations moved much more quickly, at much less cost, and with greater success than in the West, so that at the end of the eradication era “...the white pine of the East is no longer endangered by blister rust” (1). This certainly did not apply to large areas of Ontario and the Maritime Provinces of Canada.

Some argued that the control measures only succeeded because the hazard was never that great. Samuel Detwiler, then head of Blister Rust Control, succinctly compared the magnitude of the problem faced west and east of the Great Plains: “...blister rust shows unprecedented severity in spread and damage [in the West]. I have seen nothing like it in the East. Here there are many cankers in each tree that kill all sizes of trees relatively quickly, whereas in the East there are usually but one or two cankers per tree from which trees succumb slowly...” A camp foreman in the West commented, “We miss more *Ribes* per acre than are pulled [on an acre] by the crews of the East.” The target for residual *Ribes* was 5 feet of live stem per acre, but in the West, individual plants sometimes approached 1,000 feet of live stem (1).

Antibiotics. As the realization that eradication was not going to be effective in areas of moderate to high rust hazard (most of the West), hope soared briefly that antibiotics might be the silver bullet. Operational tests were conducted first with actidione as a stem spray from the ground then with phytoactin as an aerial spray over whole watersheds before any research or adequate evaluation of their efficacy had been made. Both failed and were soon abandoned.

Silvicultural techniques. Several silvicultural treatments have long been used against blister rust, including regulation of stand density, *Ribes* eradication (on a local scale), site hazard alignment, and pruning or scribing of potentially lethal cankers. All treatments will continue to have a place when applied by practitioners with intimate knowledge of local conditions. For the most part, though, they can have only mitigating effects.

Genetic resistance. Research and development of genetic resistance in white pines during the last half of the 20th century has not produced a silver bullet either, but does perhaps offer the best long-term prospect for control. Much progress was made in understanding the biological basis of resistance. Application of this knowledge has been mainly to the two commercial species in the West, sugar pine and western white pine. With the experience of chestnut blight, Dutch elm disease, and other catastrophic exotic diseases that were unfolding simultaneously with blister rust, there was little basis for hope that adequate amounts of silviculturally useful resistance could be found in species that had not co-evolved with the pathogen. So, we were surprised and encouraged when a relatively rich array of resistance genes and mechanisms presented themselves (2,10). These included major dominant and recessive genes that confer virtual immunity, as well as mechanisms of partial resistance having more complex inheritance. All genes involved are at low to very low frequencies in natural populations. The problem for breeders and foresters is, first, to concentrate the appropriate genes in synthetic populations, such as seed orchards, and then to strategically deploy them to effect durable resistance.

We already know that some of the major resistance genes are neutralized by corresponding genes for virulence in the rust population (11,18) in a classic gene-for-gene relationship (15). Thus, deployment strategies should focus on how well combinations of different resistance genes are able to buffer synthetic host populations against sudden and destructive increases in virulence gene frequencies in the rust population. Building resistance gene pyramids and/or multilines are traditional means to this end, and are being implemented (12).

The identification of major genes invites the prospect of gene cloning and transformation of susceptible species and genotypes with desired combinations of resistance. However, this is probably still many years in the future. For example, although the Cr1 gene in sugar pine has been mapped to an interval of approximately 2.0 centimorgans, defined by two random amplified polymorphic DNA markers (3), the sheer enormity of the pine genome severely limits ever arriving at the target gene by positional cloning techniques. Another approach, probing with expressed sequence tags of different resistance gene analogues from other species, revealed

a large family of genes of the nbs/lrr type in sugar pine, but none of these has been functionally associated with Cr1. Also, an operational transformation system for pines is not yet in place. Rapid advances in understanding of the molecular structure and function of R genes in annual plants will undoubtedly reveal novel ways to simulate R gene signaling pathways and co-opt alternative natural defense systems common to all plants in a species, including susceptible ones.

Somatic embryogenesis is not yet operational for white pines, but is another potentially powerful tool for the future. Resistant phenotypes are often observed, either as individuals or as members of partially resistant families, without any clue of the genetic basis of their resistance. But basic understanding, while most desirable, it is not prerequisite to practical use; the benefits of being able to deploy multiple copies of diverse resistant genotypes, irrespective of the mechanism or inheritance, are obvious. This is especially true of slow growing, noncommercial subalpine species, for which intensive selection and breeding for resistance are not ever likely to happen. Presently, these species must rely on natural selection, perhaps assisted by silvicultural manipulation (9). One of the major problems of using somatic embryogenesis technology for resistance is to be able to induce embryos from tissues old enough to assess their rust resistance phenotype in the field.

At this time there is an awkward disconnect between science and policy. For example, the U.S. Department of Agriculture Forest Service, responsible for much of the research on the biology of blister rust resistance, is also custodian for most of the land that white pines grow on. Recent deemphasis on planting, in favor of natural regeneration, will relegate development of genetic control of blister rust to natural selection for as long as this policy lasts. Recent deployment of resistant western white pine in British Columbia, on the other hand, is accelerating.

What about virulence? Breeders have a natural anxiety about virulent races arising against deployed resistance genes, but should not overstate the danger. So far, there is hard evidence for only two virulent races against major resistance genes, but much speculation about others. Virtually nothing was known about the population genetic structure of the blister rust pathogen until the last decade, when molecular and virulence markers became available (6,7,16,17). Results of studies using these markers at first seemed paradoxical: although the genetic diversity measured turned out to be very low, population differentiation was relatively great, even over short distances (16). This implied that gene flow is low, in spite of the documented ability of the fungus to make long-distance jumps (21). The small amount of variability present is distributed without pattern over the landscape, characteristic of a metapopulation structure largely driven by frequent founder events and genetic drift. Yet, this lack of pattern is entirely consistent with the life cycle of this rust, which occasionally colonizes *Ribes* from a long distance by single or few spores that reproduce clonally (aeciospores or urediniospores). The new colony will persist as perennial mycelium if and when conditions favor pine infection by basidiospores. These spores are haploid products of meiosis and genetic recombination but are restricted to a much more limited geographic distribution. The colony thus persists with the unique genetic baggage it arrived with. Much of the apparent heterogeneity in allele frequencies should dissipate as different subpopulations begin to coalesce. An implication of limited gene flow is that virulence mutants arising in local demes may not spread rapidly to others. This has been confirmed empirically by data from monitor plantations of sugar pine containing Cr1 genotypes (13; B. B. Kinloch, Jr., unpublished data).

In so far as neutral molecular markers can proxy for virulence, the low amount of variation found is good news. Bad news is the finding that the rust is outcrossing, allowing recombination among virulence genes (5), but even this is mitigated by the fact that at least two virulence loci are cytoplasmically inherited (14; B. B. Kinloch, Jr., unpublished data).

Concern has also been voiced about the potential genetic consequences of different population bottlenecks and refugia resulting from the several different introductions of blister rust to North America from Europe. But the real genetic bottleneck was in Europe itself. After discovery in the Baltic port of Riga in mid-18th century, the disease swept across Europe to Great Britain in less than 4 decades. Spaulding (22) thought it was probably carried to the Baltics on single or a few diseased transplants of eastern white pine from botanical gardens further east in Russia. Such an epidemiological unity would almost inevitably result in corresponding genetic homogeneity, implying that the rust germ plasm exported from several European nurseries on diseased nursery planting stock to various destinations in North America, East and West, was all from the same gene pool. Nothing in the recent molecular data contradicts this, even though some of the measured genetic distances among subpopulations in North America appear relatively large. This may be more of a scale effect, resulting from relative differences within an overall background of low variability. The gravest danger to genetic selection and breeding programs may well be in reintroductions from Asia, where the *C. ribicola* complex is known to exist with different alternate host affinities, including autoecious species (20). It would be prudent to assume that it also contains additional genes of wider virulence.

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