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Genetic Interactions in the White Pine/Blister Rust Pathosystem

Bohun B. Kinloch, Jr.
Institute of Forest Genetics
Pacific Southwest Research Station
Albany CA

The nine white pine species native to North America have very different ecological roles and values, which include high quality timber production, important watershed protection, keystone ecological species, and the oldest and some of the most picturesque trees on earth. All are highly susceptible to white pine blister rust (*Cronartium ribicola*), and some have been damaged severely in parts of their natural range. The epidemic has not yet stabilized, even after a century since introduction of the disease.

Host resistance. Fortunately, if unexpectedly for hosts of exotic diseases, white pines exhibit a number of heritable resistance mechanisms to blister rust. For heuristic purposes, these can be grouped into three main kinds:

- *Major gene resistance* (MGR), is controlled by single genes that condition classic hypersensitive necrosis in needles, the primary infection courts. These genes are usually dominant, but can be modified by suppressor or enhancer genes that affect penetrance and even dominance relationships in some genetic backgrounds.
- *Slow rusting resistance* (SRR), is a single phenotypic expression that integrates different underlying resistance mechanisms. SRR is a rate reducing, partial resistance expressed by lower infection frequencies in different families, and by different kinds of bark reactions that abort infections after they establish in stem tissues. More complexly inherited than MGR, its strongest effects are observed in specific combinations of parents.
- *Ontogenetic resistance* (OGR) is a kind of resistance that develops with age in some adult trees. It is recognized by parent trees that are phenotypically highly resistant compared to cohorts in natural stands or seed orchards, but which produce highly susceptible offspring. It is genotype specific, but appears to be very strong and stable. OGR would be useful in stabilizing a crop in later parts of a rotation, but is the least understood and probably most difficult of all the mechanisms to develop. On the negative side, it masks juvenile susceptibility.

MGR is present in at least three species: sugar pine (*Pinus lambertiana*), western white pine (*P. monticola*), and southwestern white pine (*P. strobiformis*), and possibly others. Although their phenotypic expression is similar, the genes are not the same, because they interact differentially with different races of rust. The loci are designated *Cr1*, *Cr2*, and *Cr3*, respectively. Two virulence alleles in the rust have been identified corresponding to *Cr1* and *Cr2*, designated *vcr1* and *vcr2*. These loci interact in a way typical of gene-for-gene systems, such that *vcr1* neutralizes *Cr1* but not *Cr2*, and vice versa for *vcr2*. *Cr3* conditions hypersensitivity to both *vcr1* and *vcr2* (as well as the wild type race), but no race with *vcr3* has been found yet. This specificity among major genes for resistance and virulence is remarkable, considering that this pathosystem has not coevolved. Frequencies of *Cr* alleles are rare to low in natural white pine populations, yet are much higher than mutation rates. The origin and persistence of these alleles in natural populations, in the absence of any obvious selective agent (prior to

blister rust) is as mysterious as it is intriguing. It suggests some kind of genetic memory of similar encounters, perhaps with native pathogens, persists in these species from past geological epochs.

Additionally, two recessive genes for resistance have been hypothesized in western white pine in Idaho populations, but corresponding racial specificities with the rust are not known. SRR mechanisms are not specifically vulnerable to *vcr* alleles in either sugar or western white pines.

Pathogen variation. Blister rust spread in western North America from a single introduction, and in the east from several, all from infected seedlings imported from European nurseries. Similarly, the epidemic in Europe was also from a single introduction that spread very rapidly. This epidemiological unity strongly suggests that all North American introductions represent the same gene pool. Population parameter estimates of *C. ribicola* in western North America indicate that overall variability is low (only 8% polymorphic loci, 2.5% expected heterozygosity), with a genetically fragmented, metapopulation structure (Gst 0.21). Gene flow is low, in spite of high outcrossing. Variation in virulence also appears limited; none has been found on different highly resistant *Ribes* cultivars in extensive trials in Europe and North America, and only the two races that neutralize major resistance genes in sugar pine and western white pine are confirmed. Both races appear to have limited distributions. Virulence to MGR in sugar pine is evidently inherited cytoplasmically, an unusual phenomenon among pathogen virulence genes. Although other races may exist in North American rust populations, a greater threat to genetically improved white pines may lie in new introductions from Asia, the ancestral gene center of the pathogen.

Genetic management. The challenge to breeders, conservationists, and silviculturists alike is to develop *durable* resistance. The key here is to prevent sudden and drastic increases in frequency of pathogen races of wider virulence. This might be effected by concentrating and deploying different kinds of resistance genes that are mutually buffered against different virulences into synthetic or naturally selected populations. Basic strategies include building gene pyramids and developing multilines, or a combination of the two. These are discussed in the context of an ongoing developmental program with sugar pine.