

GENETIC INTERACTIONS OF WHITE PINES AND BLISTER RUST IN WESTERN NORTH AMERICA

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Background and objectives

A century since its introduction to North America from Europe, white pine blister rust has come to be recognized as one of the catastrophic plant disease epidemics in history. It has yet to stabilize, continuing to spread and intensify. The nine native white pine hosts comprise major timber producers, important watershed protectors, keystone ecological species, and the oldest trees on earth. All are highly susceptible and some have been damaged severely in parts of their native range, as well as where they have been planted as exotics. Resistance, the most promising approach to control, requires understanding of genetic interactions between hosts and pathogen, a quest that has been ongoing for half a century.

Results and conclusions

Unlike hosts of other spectacular exotic diseases that have not co-evolved with their hosts, such as chestnut blight and Dutch elm disease, white pines exhibit a number of resistance mechanisms to blister rust, even if at only low frequencies [1]. Major genes that condition hypersensitive necrosis in needles, the primary infection courts, occur in at least three species. Epistatic interactions that affect penetrance and even dominance relationships of Mendelian genes exist in some genetic backgrounds. Partial resistance, more complexly inherited, is expressed by lower infection frequency and by different kinds of bark reactions that abort infections after they establish in stem tissues. Ontogenetic resistance of adult trees is very strong, but is genotype-specific and least understood. The problem for breeders, conservationists and silviculturists is how to concentrate and deploy appropriate genes into synthetic or naturally selected populations buffered against racial change in the pathogen.

Estimates of population parameters of *Cronartium ribicola* in western North America indicate that overall variability is low: only 8% of marker loci were polymorphic, with expected heterozygosities (H_e) of 2.5%. Yet, population differentiation was high ($G_{st}=0.21$), in spite of high outcrossing. This apparent paradox may be explained by a combination of genetic drift due to founder effects of single or few aeciospores establishing new infection centres after long-distance dispersal from source populations, and low gene flow in spermatia, the gametic spore stage, which has limited mobility [2]. The epidemiological unity that characterized the spread of blister rust across Europe and then to both coasts of North America implies a corresponding genetic unity. No private alleles were found in any of the western or eastern North American populations sampled, and all may share the same gene pool as the European populations from which they derive. Variation in virulence appears limited; none was found to resistant *Ribes* cultivars after extensive trials in Europe and North America. On pines, only two races are confirmed. These specifically neutralize major gene resistance (MGR) in sugar pine and western white pine. Virulence to MGR in sugar pine is evidently conditioned by a single plasmagene. Both races appear to have limited distributions. Other virulent races may exist in North American populations, but a greater threat may lie in new introductions from Asia, the ancestral gene centre of the pathogen.

The specificity that exists in this non-co-evolved pathosystem among major genes is intriguing. For example, MGR in sugar pine is not affected by the presumed gene for virulence to MGR in western white pine, and *vice versa*. This simple complementary structure may represent a nascent gene-for-gene system.

References

1. Kinloch BB, Davis D, 1996. In Proceedings of a symposium presented by the California Sugar Pine Management Committee. University of California, Division of Agriculture and Natural Resources, Davis, pp.125-132.
2. Kinloch BB *et al.*, 1998. Canadian Journal of Botany (in press).

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