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The first conference of the IUFRO Rusts of Forest Trees Working Party (7.02.05), formerly the Rusts of Pines Working Party, was held on August 2–7, 1998, in Saariselkä, Lapland, Finland, with participants from 12 countries. The proceedings consists of 41 papers or abstracts, including those from the oral and poster presentations made at the conference and a few papers submitted by scientists who were unable to attend the conference. The articles cover various aspects of the taxonomy, epidemiology, disease resistance, control, genetics, molecular biology, distribution, ecology, and physiology of specific rusts of forest trees in Asia, Europe, North America, and South Africa. One paper provides a review of tree rusts in Hungary and another of tree rusts in Finland. A general paper discusses the systematics of rust fungi in the family Melampsoraceae and another deals with the taxonomy of autoecious pine stem rusts. Other papers concern one or more of the following rusts: *Melampsora* on *Populus* or *Salix* leaves, *Melampsora pinitorqua* on *Pinus* shoots, *Melampsoridium* on *Alnus* or *Betula* leaves, *Chrysomyxa* and *Thekopsora* on *Picea* needles or cones, autoecious stem rusts of *Pinus* (*Peridermium pini*, *Endocronartium* (*Peridermium*) *harknessii*), *Peridermium kurilense*), and heteroecious stem rusts of *Pinus* (*Cronartium flaccidum*, *Cronartium ribicola*, *Cronartium comandrae*, *Cronartium quercuum*).

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Long-term differential expression of blister rust resistance in western white pine

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Summary

Between 1950 and 1970, western white pines (*Pinus monticola*) resistant to the blister rust fungus (*Cronartium ribicola*) were bred for the northern Rocky Mountains of the western United States. F_1 and F_2 seedlings were selected using a program of full-sib crossing with intense artificial inoculation. Since selected resistance was hypothesized to be vertical, long-term exposure to racially stable rust was predicted to yield 33% canker-free trees for F_1 and 66% for F_2. Epidemics observed in an experimental plantation established in 1970 were compared to test this hypothesis. Poor early survival, unevenly distributed throughout the plantation, negated the original complete block design. Subsequently, data were stratified into two site-types, one characterized by a hot site-preparation burn (brush) and the other by a normal burn (forest). Rust incidence (proportion cankered) was measured on cohorts of about 100 trees at exposure times of 2, 4, 6, 12, and 26 years. The nonlinear monomolecular model of disease progress was used to analyze rust incidence and absolute infection rate. Rust incidence in epidemics of control stock and one resistant stock attained unity, indicating that K (incidence asymptote parameter) should equal one. Incidence data were bootstrapped 20x to generate confidence limits. Of six epidemics (two sites x three stocks), only F_1-forest and F_2-brush mean infection rates were not significantly different. The finding that brush to forest infection-rate ratios were nearly constant over stocks indicated that environmental factors other than sporidial density influenced the infection rate for all stocks. Finally, all combinations of resistant stock and site exceeded expected incidence thresholds after 26 years. Was racial variation of the rust responsible, or did selection focus on horizontal resistance?

Key words: *Cronartium ribicola*, *Pinus monticola*

1 Introduction

The blister rust (BR) fungus (*Cronartium ribicola* J.C. Fisch.) was introduced into western North America in 1910 and promptly caused widespread mortality. Particularly large losses occurred in extensive stands of commercially valuable western white pine (WWP; *Pinus monticola* Dougl.) growing in eastern Washington, northern Idaho, and western Montana, a region known locally as the Inland Empire. The pathogen reached the Inland Empire about 1925, where the initial response was to eradicate alternate

hosts, species of the genus *Ribes*. Sufficient eradication was impossible, given that five million acres of mountainous terrain needed attention. Soon, the need for alternatives became evident.

Resistant phenotypes of WWP were well known by 1947, and breeding for resistance began in 1950 (Bingham 1983). Reproductively mature and canker-free trees were selected from stands exposed to severe rust hazard. Ultimately, 400 candidate trees were located, crossed in a partial-diallel mating scheme, exposed to artificial inoculations, and screened for canker-free seedlings four years after inoculation (Bingham 1972). Large progeny tests were analyzed for evidence of resistance mechanisms based on signs, symptoms, and disease cycle. Two mechanisms located in needle tissue, premature-needle-shed and reduced-needle-lesion frequency, were found (McDonald and Hoff 1970, Hoff and McDonald 1980b). Fungicidal-short-shoot and canker elimination (Hoff and McDonald 1971, 1972a) were located in stem tissue. General combining ability (GCA) for resistance based on these mechanisms was found in about 100 candidates. These GCA-selected individuals produced increased levels of canker-free progenies in all crosses (Bingham *et al.* 1969).

GCA-selected trees were crossed to yield an F_1 that produced 25% spots-only (combined premature-needle-shed and fungicidal-short-shoot) and 8% canker elimination resistance after exposure to intense artificial inoculation. Thus, 33% of trees known to have needle infections were expected to remain free of cankers (Hoff *et al.* 1973). Canker-free trees selected from the artificially inoculated F_1 population became the parents of the second generation (F_2) of resistant material. When exposed to artificial inoculation, F_2 produced 59% spots-only and 7% canker elimination resistance, or a 66% canker-free expectation (Hoff *et al.* 1973). Simple inheritance (McDonald and Hoff 1971) and assumed blockage of further rust development (Hoff and McDonald 1980a) in plants with these resistance mechanisms formed the basis of a vertical resistance hypothesis. Comparative analysis of epidemics can provide a critical test.

Disease progress curves allow comparisons of epidemics based on a strong theoretical foundation (van der Plank 1963, 1975, Zadoks and Schein 1979, Madden and Campbell 1990). BR epidemics are known to follow the nonlinear monomolecular model (Kinloch and Byler 1981, McDonald and Hoff 1982). Madden and Campbell's (1990) equation,

$$y = K(1 - e^{-rt}), \qquad (1)$$

is appropriate, where y = incidence at time t, K = maximum incidence, e = base of natural logarithm, r = absolute infection rate in incidence/year, and t = age of the epidemic in years. The model assumes r is constant (unbiased variation) over t and that disease occurrence and pathogen spread are random processes. Rate of incidence increase is assumed proportional to the amount of healthy tissue remaining (Campbell 1986) or the number of healthy plants (Waggoner 1981). When initial amount of disease is zero, as in WWP plantations, the infection rate r is

$$r = \ln(K/(K - y))/t, \qquad (2)$$

(McDonald and Hoff 1982), where r, K, y, and t are as above and ln = natural logarithm.

Infection is an ecological process, and resistance mechanisms can be either resistance to infection or resistance to colonization (Nelson 1978). Mechanisms preventing infection are vertical, hypersensitive, race-specific, or major-gene resistance. These mechanisms in BR, along with environmental factors, control the magnitude of the spores-to-spots ratio. At least one classic vertical mechanism is known in the BR pathosystem (Kinloch and Comstock 1981). Nelson (1978) defined mechanisms that impede colonization of the invader as horizontal, generalized, minor-gene, or race-nonspecific resistance. In BR, these mechanisms and environmental factors control the magnitude of the spots-to-canker ratio. Finally, additional horizontal mechanisms in the BR pathosystem could influence canker size and shape, spore-production rates, and growth rates of cankers (Hoff 1984).

Resistance definitions stated in terms of the monomolecular model provide quantitative points useful for epidemic comparison. If resistance is vertical in a portion of a population, the ratio K_S/K_R (susceptible/resistant) is greater than one. Three factors that depress K are clumped inoculum distribution, heterogeneous infection microclimate, and incomplete vertical resistance (McDonald *et al.* 1991). K determined by actual observation is needed to use equation (1) (Neher and Campbell 1992). When K_S measured on-site is equal to one, the assumption of random distribution of disease is validated. Then, K_R can be used to compare resistant material. In epidemics involving vertical resistance, the infection-rate ratio, r_R/r_S, will equal one (van der Plank 1975). Here, the hypothesized canker-free WWP proportions of 0.33 (F_1) and 0.66 (F_2) become K = 0.67 and 0.34, respectively. When vertical resistance is complete, K_R equals zero and K_S/K_R is undefined. However, racially-caused breakdown of resistance would move K_S/K_R toward one. Alternatively, if resistance is horizontal, K_S/K_R equals one and all action shifts to the r parameter. Epidemics in different WWP stocks would have unequal r and equal K_S/K_R. Ratios of r parameters associated with differing levels of resistance would be stable over time and not equal to one. Thus, variation of infection rate distinguishes populations expressing horizontal resistance (Nelson 1978). This paper reports a preliminary test of the hypothesis that BR resistance in Inland Empire WWP is vertical.

2 Material and methods

Bingham *et al.* (1973) presented the layout and experimental design of the 14.6 ha Merry Creek plantation located in northern Idaho, USA. After the second growing season, seedlings were selected from control, F_1, and F_2 stock. A metal tag was applied to 25 trees from each of nine 0.4 ha plots planted to each stock. Seedlings were inspected each fall at 2, 4, 6, 12, and 26 years after planting, except that the 26-year inspection required an entire field season. Cankers/tree, tree height, and tree condition (living or dead) were recorded to provide disease severity (cankers/tree) and disease incidence (proportion of population infected) assessments. Cankers were counted while inspectors were standing at the base of crowns. All 26-year-old trees designated canker-free from the ground inspection were climbed to verify their status.

Uneven mortality, not caused by *C. ribicola*, negated original design objectives and forced creation of a new design. A boundary delineated by

density of a brush species afforded an opportunity to divide the plantation into two site-types. Density of evergreen ceanothus (*Ceanothus velutinus* Dougl.) is directly related to intensity of the heat pulse generated by a forest fire or site-preparation burn (Noste and Bushey 1987). Twenty-eight years after burning, a boundary seen as a change from low to high brush density was mapped using GPS technology. Then, location of individual sample trees was determined by an offset technique (McDonald *et al.* 1998). Next, plot trees were assigned to brush and forest site-types based on their mapped location. Nutrient and biophysical verification that brush and forest site-types were different was planned but not completed for this preliminary study.

Random distribution of infections is required for comparative analysis of epidemics (Madden and Campbell 1990). The three components of infection distribution are distribution of spores, infection microclimate, and resistance genes in the host population (McDonald *et al.* 1991). Distribution of sporidial concentrations was not measured at Merry Creek. However, much is known about sporidial distribution. Pine infection is caused by windborne sporidia (5 µm in diameter) produced on the alternate host. Five spore-stages function in clearly defined annual cycles on two woody perennials to produce long-lived epidemics. Annual sporidia concentrations can vary 130x (McDonald *et al.* 1991). At Merry Creek, 3000 sticky currant (*Ribes viscosissimum* Pursh.) per hectare (unpubl.) should produce about 150 spores cm^{-2} in a "good" weather season (based on extrapolation of Figure 3 in McDonald *et al.* 1991). At such densities, average distance from spore source to target is 2 m (McDonald *et al.* 1981). An empirical disease-distance curve constructed for BR (Buchanan and Kimmey 1938) shows that about 90% of spores produced by a bush will be deposited within 2 m of that bush. The relationship assumed that the ratio of needle infections to canker appearance was not distant-dependent. We assumed that high *Ribes* bush densities evenly distributed across brush and forest site-types would provide random horizontal distribution of sporidia.

Growth of host tissue may cause problems with application of disease progress models (Madden and Campbell 1990). These problems mostly pertain to polycyclic diseases where inoculum is being produced on the growing hosts. Available host tissue can increase faster than available inoculum. In the case of BR, inoculum production is not related to amount of needle tissue except to the extent that WWP canopies shade *Ribes* bushes. Such shading can result in significant reductions of inoculum load (Hagle *et al.* 1989), thereby reducing infection rate as trees grow. In addition, high population densities of *Ribes* produce such immense volumes of sporidia that trees can reach a large size before available infection sites exceed the number of available spores. For example, a 25-year-old WWP growing on a good site has a projected crown area of about 49×10^3 cm^{-2} (calculated from data in McDonald *et al.* 1981). Thus, 7.36×10^6 sporidia (49×10^3 cm^{-2} x 150 sporidia cm^{-2}) should be available in the 25th season to infect 1.1×10^6 cm^2 of infective needle surface (calculated from data in McDonald *et al.* 1981). On this theoretical basis, we assumed that host tissue had not saturated inoculum availability by the 26th year of the epidemics.

Heterogeneous infection microclimate could be another major cause of clumped infection. Microclimate data were not collected at Merry Creek, but microclimate could be modeled for the 26-year duration of the experiment.

However, in the Inland Empire, conditions for pine to become infected appear to be uniform within infection cycles (season) because most pine infections develop during the passage of stable slow-moving cool and wet air masses (McDonald *et al.* 1981). Thus, the assumption was made that infection microclimate remained uniform across brush and forest sites within an infection season.

Rust incidence was determined three times for control populations planted on brush (91 trees) and forest (80 trees) site-types. Incidence was determined five times on F_1-brush (54 trees), F_1-forest (91 trees), F_2-brush (36 trees), and F_2-forest (68). To supply estimates of confidence limits, these samples were bootstrapped 20 times using a sampling with replacement algorithm in Systat 7.0 (Wilkinson and Engelman 1997). Using the bootstrapped samples, r was computed by equation (2), where t = 2, 4, and 6 years for controls and 2, 4, 6, 12, and 26 years for the remaining populations. Thus, 20 estimates of r became available for each combination of measurement-year, stock, and site-type. Means and confidence limits (95%) of rust incidence were computed from the bootstrapped data for each inspection year x site-type x stock combination. These observed values were compared to expected incidence (y in equation (1) where r = mean infection rate, and K = 1).

3 Results

Attainment of 100% rust incidence by control stocks at Merry Creek validated the assumption of random distribution of disease (Table 1). The BR epidemic in the F_1-brush pine population progressed to unity as well. The remaining epidemics, F_1-forest, F_2-brush, and F_2-forest nearly achieved unity after 26 years of exposure (Table 1). On this basis, we tentatively concluded that K = 1 in all Merry Creek epidemics and continued with the analysis.

The ranking of epidemics with respect to observed rust incidence was mostly consistent (Table 1). Two exceptions were evident in F_1-forest and F_2-brush after six and 12 years (Table 1). Agreement between observed and expected rust incidence within individual epidemics and across years appeared to be unbiased for all epidemics (Table 2). This finding means that infection rates could be pooled over years for each stock and site combination. Four of the combinations differed significantly from mean infection rate and two, F_1-forest and F_2-brush, did not differ (Table 3).

Table 1. Observed incidence of blister rust (number of cankered trees/total number of trees) in six populations of western white pine grown at the Merry Creek plantation located in northern Idaho, USA.

	Years of exposure				
Epidemic	2	4	6	12	26
C-Brush	0.83	0.89	1	–	–
C-Forest	0.71	0.84	0.94	1	–
F_1-Brush	0.45	0.58	0.71	0.81	1
F_1-Forest	0.37	0.45	0.58	0.67	0.96
F_2-Brush	0.29	0.41	0.63	0.72	0.97
F_2-Forest	0.20	0.23	0.42	0.50	0.89

Table 2. Residual of observed blister rust incidence minus predicted incidence[1] in six populations of western white pine grown at the Merry Creek plantation located in northern Idaho, USA.

	Years of exposure = *t*				
Epidemic	2	4	6	12	26
C-Brush	+0.05	–0.06	0.0	–	–
C-Forest	+0.08	–0.02	0.01	0.0	–
F_1-Brush	+0.01	+0.01	0.01	–0.11	0.0
F_1-Forest	+0.12	–0.02	–0.04	–0.18	–0.02
F_2-Brush	+0.04	–0.03	+0.05	–0.10	0.0
F_2-Forest	+0.05	–0.06	+0.02	–0.14	0.0

[1] predicted incidence = $(1-e^{-rt})$ where r = mean rate in Table 3

Table 3. Means of absolute rates of blister rust infection calculated from bootstrapped (20x) data and the monomolecular model of disease progress as observed in six populations of western white pine grown at the Mary Creek plantation located in northern Idaho, USA.

	Mean	95% confidence limit		Brush to	C/F_1 & C/F_2	C/F_1 & C/F_2
Epidemic	Rate	Lower	Upper	Forest ratio	Ratios (brush)	Ratios (forest)
C-Brush	0.753	0.707	0.799			
C-Forest	0.504	0.48	0.528	1.49		
F_1-Brush	0.212	0.199	0.224		3.7	
F_1-Forest	0.162	0.147[1]	0.177	1.31		3.1
F_2-Brush	0.144	0.135	0.153[1]		5.2	
F_2-Forest	0.084	0.079	0.089	1.71		6.0

[1] confidence limits overlap, therefore F1-Forest and F2-Brush are not significantly different

The various ratios of infection rates provide clues about relationships between resistance and environmental factors. A nearly constant brush-to-forest ratio of 1.5 over the three stocks (Table 3) indicated that the brush site was more suitable for expression of cankers than the forest site. Control to F_1 ratio was larger on the brush site than on the forest site, as expected. Control to F_2 ratios diverged unexpectedly, where the brush ratio was smaller than the forest ratio (Table 3).

4 Discussion

The primary objective of this paper was to ascertain if long-term performance of Inland Empire blister rust (BR) resistant western white pine (WWP) followed the model of vertical resistance. If resistance in the F_1 and F_2 populations was vertical, maximum rust incidences would be 0.67 and 0.34, respectively. Also, according to theory, the susceptible portions of these two populations should exhibit equal infection rates and that rate should be equal to the susceptible population. Results showed that rust incidence for one of the four possible combinations of resistant populations and sites had already achieved 100% infection. Two of the remaining three exceeded 95% infection and the best combination (F_2-forest) was 90% infected. Since this experiment was not replicated, chance cannot be ruled out as an explanation. However, F_2 stock has been planted in many locations in the Inland Empire and critical

determination of rust incidence has shown several cases where K exceeds 0.34 (unpubl.). One noteworthy case of rust incidence above 34% in F_2 stock was attributed to mislabeled planting stock (McDonald *et al.* 1994); now this analysis should be revisited. One could argue that the 5% and 11% proportion canker-free trees at this time represent K values of 0.95 and 0.89. The following discussion about rate of infection will add evidence with which to judge this question.

Another important aspect of horizontal resistance is its relative nature (Shaner and Finney 1977). Horizontal resistance to a leaf pathogen of wheat could only be measured in relation to a susceptible cultivar. As conditions for disease improved (more soil nitrogen or better weather), the relative performance of the resistant cultivar improved (Shaner and Finney 1977). At Merry Creek, four of six combinations of stocks and sites exhibited significantly different infection rates. More importantly, the ratios of infection rates from brush site-type to those from forest sites-types of all stocks were about 1.5. Thus, the brush site provided the highest relative infection rate in both controls and resistant stock. As expected from the wheat experience, relative F_1 performance was better on the brush site-type than on the forest site-type. This trend did not carry through to the F_2, where the forest site gave the best relative performance. Overall, analysis of rates of infection also suggests that resistance in Inland Empire WWP is horizontal.

Since a thorough understanding of infection rate is crucial, this area should receive future emphasis. One potentially productive approach would be to study soil biophysical differences between brush and forest sites at Merry Creek to verify they differ. On the other hand, the difference between brush and forest may have been caused by a consistent difference in sporidial concentration that was assumed to be absent. A careful analysis of canker frequency on a large sample of surviving trees at Merry Creek may provide some answers about relative hazard. Additional evidence about the Merry Creek epidemics might be obtained by applying the needle-trace method of assaying needle retention in pines (Jalkanen *et al.* 1994). If signatures of expression of needle-spots-only and/or canker elimination resistance remain in WWP needle traces, then the frequency of such host reactions could be measured on living and dead white pine. In addition, this method has potential to track amounts of needle tissue over time to validate pine target models.

In a thorough review of horizontal resistance in crop plants, Simmonds (1991) listed seven characteristics of horizontal resistance. These are: polygenic inheritance, continuous distributions of infection rates, resistance rather than immunity, pathotype non-specificity, durability over time, and resistance composed of several components. Finally, genetic evidence is necessary for critical demonstration of horizontal resistance. The population of rust-resistant WWP selected by the Inland Empire program fits most of the seven characteristics.

Infection rate was continuously distributed over several levels of selection where early experimental plantations contained a spectrum of stock from susceptible to F_1 (Goddard *et al.* 1985). Inland Empire WWP has expressed resistance rather than immunity. Pathotype non-specificity in the BR pathosystem was critically investigated once (McDonald *et al.* 1984). Materials from a selection program in Oregon were used to delineate the

Champion-Mine (CM) pathotype. Selected and non-selected WWP were compared after infection with wild type and CM inoculum. The CM pathotype was equally more aggressive than the wild type toward reduced-needle-spot-frequency and needle-spots-only mechanisms on both selected and non-selected families (McDonald *et al.* 1984). This finding is consistent with the horizontal resistance hypothesis because differences were relative. Durability over time can be currently assessed only by looking at the correspondence of mechanisms and their durability in related five-needle pine growing in native associations with *C. ribicola.* There is some evidence that BR-resistant Inland Empire white pine has these durable mechanisms (Hoff and McDonald 1972b, Hoff *et al.* 1980). The single hypothesis constant with results reported in this paper and the existing literature is that the Inland Empire BR breeding program selected for classic horizontal resistance instead of vertical resistance.

Regarding critical genetic evidence, many of the tests suggested by Simmonds (1991) could be applied. Foremost among these are generation means and variance analysis, analysis of specific and general combining ability, offspring-parent regressions, and response to selection. The last test has been applied in that response from susceptible to F_1 was 0.33 and from F_1 to F_2 was another 0.33. Horizontal resistance typically yields a linear response to selection (Simmonds 1991). These critical tests, as well as other avenues, should be explored, because breeding and integrated management programs alike depend on determination of the true nature of resistance in Inland Empire WWP. These investigations will provide an improved foundation for breeding and blister rust management of all five-needle pines.

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