Relative Effectiveness and Stability of Different Resistance Mechanisms to White Pine Blister Rust in Sugar Pine

Bohun B. Kinloch, Jr. and James W. Byler

Geneticist, Pacific Southwest Forest and Range Experiment Station, Forest Service, U. S. Department of Agriculture, P. O. Box 245, Berkeley, CA 94701; and plant pathologist, Region 1, Forest Service, U. S. Department of Agriculture, P. O. Box 7669, Missoula, MT 59807.

Accepted for publication 2 September 1980

ABSTRACT

Kinloch, B. B., Jr., and Byler, J. W. 1981. Relative effectiveness and stability of different resistance mechanisms to white pine blister rust in sugar pine. Phytopathology. 71:386-391.

In progeny and clonal tests, several heritable types of resistance to *Cronartium ribicola* were found in a selected population of sugar pine parents. Major gene resistance (MGR), controlled by a single dominant gene, was found in 16% of selected candidates and was highly effective. Although vulnerable to a virulent race that had been previously detected at low frequency in the rust population, MGR was highly stable for up to 14 yr. Sudden erosion of resistance was observed following 1 yr of unusually favorable conditions for inoculum production and infection. It was uncertain whether this represented only an ephemeral breakdown in resistance due to an unusually high inoculum density, or a change in frequency of the virulent race. Slow rusting, presumably under polygenic control, was indicated by differential infection rates of families from different parents. The degree of resistance exhibited for this trait was low, but relatively stable and amenable to considerable improvement in early breeding generations. Another kind of apparently quantitative resistance was expressed by large differences in infection among grafted clones. Since this variation contrasted greatly to the generally high levels of infection of seedling progenies derived from the same clones, the existence of ontogenetic factors of resistance was strongly suggested.

Additional key words: Pinus lambertiana, hypersensitivity.

Since its introduction to western North America early in the century, white pine blister rust (*Cronartium ribicola* J. C. Fischer ex Rabenh.) has spread epidemically on white pines over vast areas. Sugar pine (*Pinus lambertiana* Dougl.), a major commercial species in California and Oregon, is one of the most susceptible, and its regeneration, both natural and artificial, has been severely curtailed in areas of high disease hazard.

Although North American white pines have not coevolved with the blister rust pathogen, a surprising number of resistance mechanisms have been reported (3), and considerable progress has been made in breeding for resistance (4). Single genes for resistance have been indentified in both sugar pine (10) and western white pine (*P. monticola* Dougl.) (14). Several other mechanisms thought to be under either monogenic, oligogenic, or polygenic control have

been suggested (3,6,7).

Because major genes in cereal and other crops have proven to be so vulnerable to races of rust with complementary genes for virulence, emphasis in recent years has shifted to polygenic sources of resistance that have less dramatic effects but are thought to be more stable. The effects of polygenic resistance are more difficult to measure and their benefits take longer to achieve (23). The underlying mechanisms, usually more varied and complex than major gene resistance, are only now becoming clarified (5,20,24).

One phenotypic expression that may integrate several underlying components of polygenic resistance is slow rusting (12), by which differential amounts or rates of infection are evident among susceptible families. Another type of resistance recognized by plant breeders is adult plant resistance, which effectively confers a high degree of resistance to plants at maturity, but not in juvenile stages. Slow rusting and adult plant resistance usually are assumed to be under polygenic control (19), although both simple (1) and complex (11) modes of inheritance are known.

Few studies have compared the stability and effectiveness of major gene, polygenic, and adult plant resistance in wild populations under natural conditions. The purpose of this paper is to show that all of these types of resistance exist in sugar pine and to assess and compare their relative values in a long-range breeding program.

MATERIALS AND METHODS

Selection of putatively rust-resistant parent trees began in 1957 in the Klamath and Siskiyou mountains in northern California, where blister rust first entered the state and has been chronic or severe since about 1936 (16). Selected trees were free or nearly free of rust in stands where infection had been intense for 15 yr or longer and alternate host *Ribes* spp. were abundant. The ideal candidate tree was one whose branches were interlaced with neighboring sugar pines with multiple infections. The approximate numbers of blister rust cankers on several trees surrounding each candidate tree were determined by inspection with field glasses. Scions of selected trees were grafted onto potted rootstocks and later (1960–1966) planted in a clone bank on a site where disease hazard was high. Ramets (four to six per clone) in any given year were randomized and planted on 3-m centers. In 1966, *Ribes* bushes were interplanted at every third row and line. Subsequently, numbers of cankers on each ramet were recorded periodically.

Meanwhile, sexually fertile trees were intercrossed in the wild to produce full-sib families for progeny testing. Because of limited manpower, rugged terrain, and considerable distances between candidate trees, no set mating plan was used, although an attempt was made to cross each putatively resistant candidate with several others. Often, this meant pollinating the relatively few candidate trees that produced ovulate strobili consistently and abundantly.

Progeny test outplantings were made each year beginning in 1962. The progeny test (and clone bank) site is on the east fork of Indian Creek in the Siskiyou Mountains near Happy Camp, CA, at 800 m elevation. The climate is characterized by hot, dry summers and wet winters, with extended periods of fog in the autumn. Alternate host *Ribes* spp. and blister rust are plentiful in the area.

Seedlings were grown for 1 yr in tar-paper containers, outplanted in late winter the following year, and watered for the first 2 yr on the site. Survival was generally high.

Seedlings were planted in a randomized complete block design with three to five replications of each family, depending on the amount of seed available, in row plots of 10 trees on 34-cm centers. Test families were planted in units of six, including a control derived from known susceptible parents, or from bulk seed lots, presumed to be susceptible, from squirrel caches. Each unit was surrounded by bushes of *R. sanguineum* Pursh. with occasional bushes of *R. roezlii* var. *cruentum* Jepson; both species are naturally abundant in the area.

Infection of *Ribes* by blister rust took place naturally and usually abundantly throughout the growing season. Teliospore production and sustained periods of cool, moist weather conducive to basidiospore dispersal and infection of pine usually did not take place until fall. In occasional dry years, *Ribes* shed their leaves before conditions favorable for infection occurred. Unusually moist and cool conditions were recorded throughout the summer of 1976 (17).

Seedlings were observed for rust symptoms and signs each year from 1964 to 1978. Data recorded were presence or absence of bark infection on bole or branches, mortality due to rust infection, and pathogen sporulation. Since two patterns of variation in susceptibility were evident among different families—one discontinuous and the other continuous—different analytical approaches were used. Data for families that segregated into qualitative classes (infected and noninfected) were pooled over replications and analyzed for Mendelian ratios by chi-square tests. The remaining families varied continuously in several traits, including percentage of seedlings infected, mortality rate, number of aborted infections, and rate of infection over time. Variability in infections per seedling was evident also, but could not be accurately documented because of the small size of most seedlings; multiple infections often merged and became individually indistinguishable.

The most useful parameter was infection rate, estimated by the formula

$$r = \frac{1}{t_2 - t_1}\left[\log_e \frac{1}{1 - X_2} - \log_e \frac{1}{1 - X_1}\right]$$

in which r is the apparent infection rate, t_1 and t_2 are dates (years), and X_1 and X_2 are the proportions of seedlings infected at t_1 and t_2 (25). This equation is applicable to "simple interest" disease increase, in which all inoculum is generated apart from host plants and is not self-multiplying. It is appropriate for the portion of the blister rust disease cycle on pines, which become infected during one relatively brief period in the autumn. The interval t_2–t_1 was usually 5 yr—from the second to the seventh year of record. Since a full year after inoculation usually is required before bark symptoms (the criterion of infection in this study) can be detected in the field, infections occurring between the first and sixth seasons of exposure were thus considered. When individual families reached 100% of seedlings infected earlier than the seventh year, the next previous record year was used for t_2 and X_2; otherwise a zero would appear in the denominator of the infection-rate equation.

In order to provide an adequate sample of half-sibs from which the relative breeding value of a given parent could be estimated, infection rates were analyzed only for parents represented by at least five crosses. Infection rates were computed for each family and averaged over families common to the same parent. Only five plantations (those established from 1964 to 1968) were used in this analysis; those before 1964 had too few parents and those after 1968 consisted largely of open-pollinated families. To estimate standard errors of infection rates for each parent, an entire family in a given year was treated as a plot and its half-sib relatives as replicates, irrespective of the planting year in which these replicates were established; thus, replicates and planting years inevitably were confounded.

RESULTS

The amount of infection varied considerably from year to year but was particularly severe in the earlier (1962–1966) and later (1975–1976) years. For example, infection among different control lots after equivalent durations of exposure (4 yr) averaged 97% (range, 87–100%) in seven plantations established between 1962–1966 and between 1973–1974, but only 59% (range, 37–79%) in six plantations established between 1967–1972. By 1978, when these evaluations were completed, over 99% of control trees in all plantations were infected and 97% were dead. Under these conditions, we consider the likelihood of disease escapes to be small, and that any plants that remained either uninfected or infected but alive were expressing resistance.

Major gene resistance. Major gene resistance (MGR) in sugar pine is conditioned by a dominant gene that causes a hypersensitive reaction to the pathogen in needles (10), which are the main

infection courts, and thus prevents bark symptoms from developing. Although a direct examination of needle symptoms is not feasible in the field, MGR can be inferred from segregation ratios of progenies for presence or absence of bark symptoms. Thus, with an inoculum potential adequate to preclude disease escapes, heterozygous carriers of MGR mated to homozygous recessives should average 50% rustfree offspring. By 1976, susceptible controls in all plantations were over 90% infected, except those in the three plantations established from 1972–1974, which only ranged 34–69% infection. To adjust for disease escapes in these three plantations, parents were regarded as MGR carriers if the incidence of bark infection in their progeny was ≤50% that of the

TABLE 1. Mean cumulative blister rust infection on sugar pine families from selected parents, as a percentage of infection of susceptible control seedlings, in 13 plantations (1962–1974)

Parents	Families[a] (no.)	Years planted (no.)	Blister rust infection (% of controls[c]) 1976	1978
Heterozygotes[b]				
K 10	4	3	54.1	79.4
K 17	14	7	54.3	71.7
K 19	5	3	51.3	77.9
K 50	2	2	47.3	77.9
K 65	2	2	56.0	89.6
K 73	17	10	47.4	79.7
P 10	2	2	45.6	51.2
P 11	2	1	56.7	66.7
P 20	3	3	47.7	54.7
BLM 4	5	2	55.7	72.0
Others (10)[d]	10	6	51.9	68.4
Totals 20	66	...		
Means			51.3	73.4
Homozygotes[b]				
E 7-2	1	1	2.4	26.2
K 70	11	7	2.2	45.6
Totals 2	12	...		
Means			2.2	44.0
Remaining candidates (115)	240	...	93.0	97.5

[a] Average number of seedlings per family was 39, and for control lots was 232.
[b] For major gene resistance.
[c] Mean infection of controls (over all plantations) was 87.4% as of 1976, and 99.8% as of 1978.
[d] Ten parents in a single cross of each.

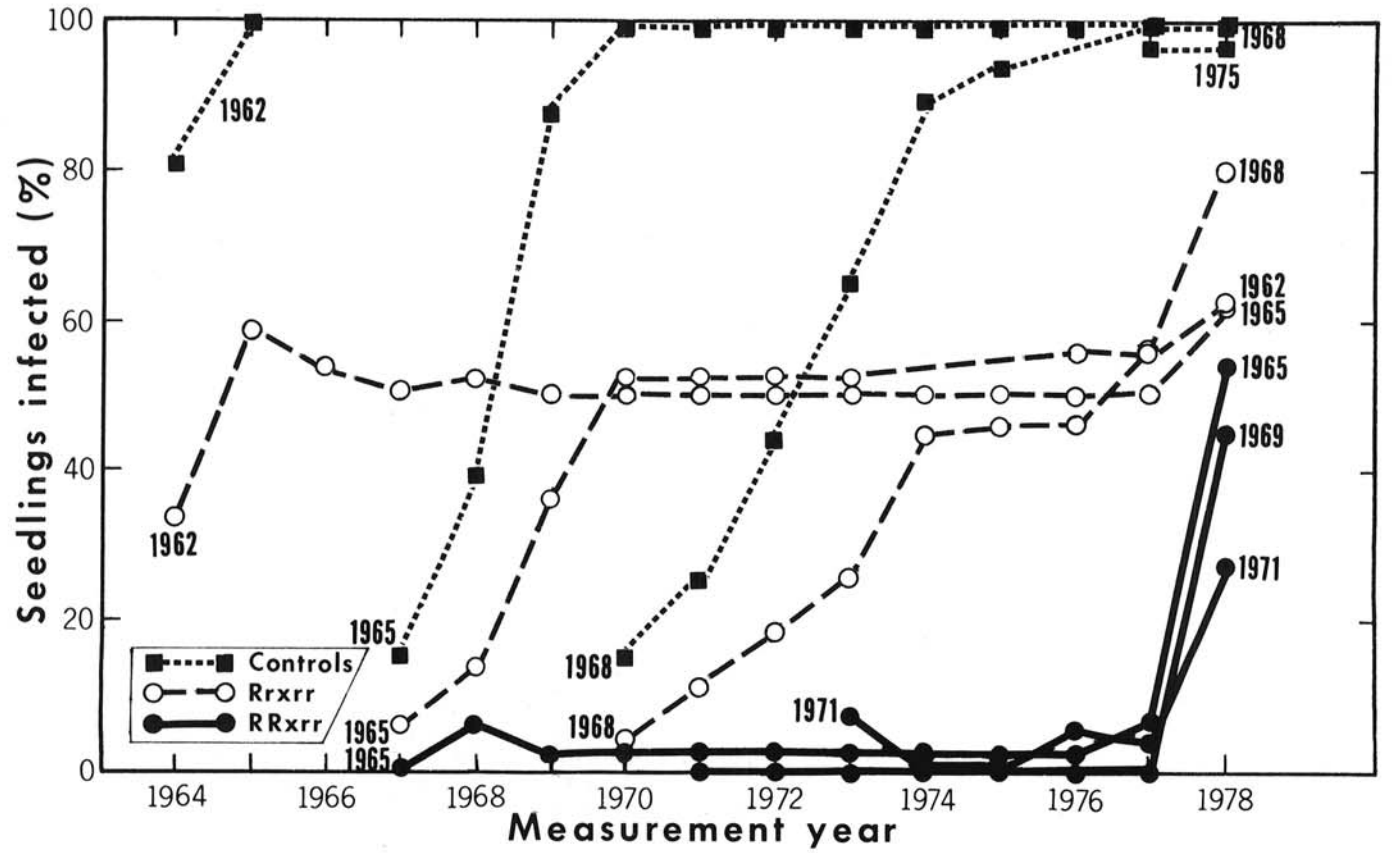

Fig. 1. Progress of blister rust infection on susceptible and resistant sugar pine in typical plantings at the Happy Camp test site for representative planting years. R and r represent dominant and recessive alleles for resistance and susceptibility, respectively.

controls.

Of 137 phenotypically resistant parent trees evaluated in all plantations, 22 (16%) were identified as carriers of MGR; two were homozygotes and the rest heterozygotes (Table 1). None of the chi-square values computed for any of the families of any of the segregating parents was significant up to the 1976 measurement year. However, nine of a total of 342 seedlings from the two putatively homozygous parents did become infected during this period; four of these died and five subsequently recovered.

Between 1976 and 1978, infection suddenly and dramatically increased on seedlings previously classified as resistant in both segregating and nonsegregating families with MGR. Until 1977, disease progress curves on these families had stabilized (some for as long as 14 yr, in the case of the 1962 plantation; Fig. 1). By 1978, however, infection had reached 73% of controls in 66 segregating families and 44% of controls in 12 nonsegregating families for MGR (Table 1).

Most of the new infection observed in 1978 took place in 1976. This increase of infection observed on seedlings of known MGR genotype was associated with environmental conditions unusually favorable for early disease development on both alternate hosts during the late summer of 1976. Ordinarily, summers in the Klamath Mountain region are hot and dry; weather sufficiently cool and moist to promote production, dispersal, and germination of basidiospores usually does not arrive until late September or October. In 1976 the mean temperature in July and August was, respectively, 0.8 and 2.6 C degrees cooler than average (23.2 and 22.3 C) and precipitation was 11.8 and 66.0 mm greater than average (10.9 and 8.9 mm), respectively, at the Happy Camp Ranger Station, 10.3 km south of the test site (17). During these 2 mo precipitation exceeded 2.5 mm on 9 days. Although we had no direct measure of inoculum potential, abundant production of telia was noted on *Ribes* bushes as early as late July. With the cool, moist weather that followed in August, it is likely that the inoculum potential in this year was much higher than usual, as evidenced by the extremely rapid infection of control seedlings planted in 1974 and 1975. Cumulative infection on the 1974 controls increased from 34.1% in the 1976 measurement year to 100% in 1978, and that on the 1975 controls reached 96.1% in 1977.

Slow rusting. Families lacking MGR were highly but not uniformly susceptible. Relative resistance, as measured by mean infection rates (r), varied continuously. Among families of 18 parents (including controls), representing five or more crosses of each parent, r values ranged from 0.257 to 0.883, and had greater resolving power than cumulative infection, which only ranged from 80 to 98% (Table 2). Rigorous statistical analysis of these data was not possible because the several parents were confounded with planting years; there were unequal numbers of crosses among parents, and common testers were not used for the parents evaluated.

Nevertheless, we consider the observed differences in infection rates to be real. Within any given year, there was a fourfold to fivefold difference in mean infection rates among parent trees, consistent with the more than threefold difference combined over all years and parents. Standard errors of infection rate for the different parents were relatively small and uniform, and overlapped adjacent means only slightly. The four parents (K71, K86, K16, and K36) and the controls common to the five planting years evaluated (1964–1968) and representing relatively low, intermediate, and high infection rates, consistently maintained rank with respect to each other. Coefficients of rank correlation between any two comparison years ranged from 0.6 to 1.0, the mean of all possible comparisons being 0.8. Parents K71 and K86, with 20 and 15 crosses, respectively, ranked in the least susceptible quartile in all 5 yr, whereas controls ranked in the most susceptible quartile in 3 of 5 yr.

Potential gains in resistance from selective breeding were indicated by the relative performance of families from crosses among slow × slow, slow × fast, and fast × fast-rusting parents. The mean infection rate (0.272) of eight families from the six top-ranked parents for slow rusting in Table 2 was only one quarter of that for 13 families from the six lowest ranked parents (1.026), while 15 families from slow × fast matings were intermediate (0.562). Individual families of crosses among relatively slow-rusting parents occasionally had infection rates and cumulative infection far below the average for their parents. For example, a selfed family of K71 had an r value of only 0.085, compared to 1.026 for controls of the same year and 0.257 for all K71 families over all years, and a cumulative infection of only 52% after 13 yr. It is noteworthy that infection did not increase from this level on this family following the severe epidemic of 1976.

Mature tree resistance. Patterns of infection observed in the clone bank and progeny tests suggested the existence of different levels of resistance that are unrelated to MGR, and also possible differences in resistance between juvenile and mature stages of growth. To test these hypotheses, we compared rust infection between candidate trees and their cohorts in the wild, between candidates and their ramets in the clone bank, and between these ramets and seedling progeny immediately adjacent to them. These

TABLE 2. Infection rates and cumulative infection of seedling offspring and grafted clones of putatively resistant sugar pine parents by white pine blister rust after 6 yr of exposure in a disease garden

Parent	Number of families	Planting years[a]	Infection rate (r)			Cumulative infection after 6 yr (%)	
			Mean	± Std. error	Range among families	Families[b]	Clones[c]
K44	6	1	0.257	0.084	0.098–0.386	81	14
K71	20	5	.356	.041	.078– .995	80	0
K86	15	5	.356	.045	.134– .704	84	0
K39	5	1	.475	.099	.193– .709	87	33
ST10	5	2	.520	.071	.395– .636	92	33
K16	9	5	.529	.068	.317–1.275	90	50
K46	10	3	.536	.052	.267–1.136	94	75
K78	10	4	.545	.060	.173–2.190	90	0
K33	5	2	.572	.106	.168–1.339	89	0
K14	8	2	.588	.073	.346– .897	97	0
ST11	5	2	.595	.076	.315– .828	94	33
K62	23	4	.650	.064	.160–2.043	94	71
K9	5	2	.690	.101	.499–1.301	98	88
K5	10	3	.711	.055	.358–1.793	98	80
K36	26	5	.766	.042	.206–1.950	98	0
K41	10	4	.778	.066	.277–1.851	96	17
K60	21	4	.812	.054	.194–2.043	98	50
		Grand mean	.533				
Controls	5	5	.883	.107	.536–2.497	98	...

[a] Number of different planting years in which parent is represented by one or more families in the 1964-1968 plantations.
[b] Mean cumulative infection of all families with indicated parent in common.
[c] Mean cumulative infection of all ramets of indicated parent.

comparisons were flawed by several confounding effects: clone bank establishment was spread over a 6-yr period and *Ribes* were interplanted only upon completion, and at lower density than in adjacent progeny tests. By this time, ramets ranged in height from 1 to 4 m, and may have presented a different microclimate to impacting rust spores than did the small seedlings. Other microenvironmental variability within the clone bank was reflected by variation in severity of infection among ramets of the same clone. For example, one clone ranged from 0 to 26 rust cankers per ramet. Nevertheless, the pattern of infection on clones was far from random. Data for 57 candidate trees (excluding MGR carriers), their cohorts, and their ramets are summarized in Table 3, and parent-offspring relationships (of candidates with at least five families each) are given in the last two columns of Table 2.

Selected candidate trees, by definition, had far less rust than their cohorts of comparable age and size. Twenty-six percent of the candidates had from one to four positive or suspected cankers. By comparison, 253 cohorts were 98% infected with 1 to >100 cankers per infected tree (Table 3). Evidently, there are large differences in the relative susceptibility of mature trees in the wild population. This was corroborated by variation in the relative severity of infection among candidate clones. Over half of the clones were free of rust, while variation in susceptibility among the rest ranged from 14 to 100% of ramets infected and from 1 to 9.5 cankers per infected ramet (Table 3). Although differences among clones were clear, not all of the relationships between candidate trees (ortets) and their ramets were consistent. No ortets or ramets of selected parents known to be carriers of MGR were infected, but 30% of ortets free of rust in the wild had ramets ranging from 17 to 100% infected, and five ortets known or suspected to be infected in the wild had no rust on ramets in the clone bank.

The degree of infection on clones was less than a third of that on seedling progeny of the same parents, and the variation in susceptibility among different clones bore little relationship to the performance of their offspring (Table 2). Although this disparity might be partially explained by the confounding differences in treatment and history, the most obvious variable associated with differences in infection between parent and offspring populations was the difference in physiological age.

DISCUSSION

Major gene resistance. The effect of MGR on white pine blister rust in sugar pine is dramatic, but its value depends on its stability. Relatively little is known about variation in virulence in *C. ribicola*. McDonald and Hoff (15) and McDonald (13) hypothesized two races with genes complementary to recessive resistance alleles in western white pine, identifiable by the color (yellow or red) of the lesions they induce on needles. These lesion color types also occur on sugar pine, but neither putative race is virulent to MGR (10).

Our data indicate the existence of one or more *C. ribicola* races virulent to sugar pine with MGR. The question remains, however, whether the new infections observed on previously resistant trees indicate a selective increase of an old race, long resident in the rust population at low frequency, or only an ephemeral flare-up of this race. A third possibility, which we consider less likely, is the appearance of a new race.

On one hand, the sudden increase of infection on resistant progeny strongly suggested an abrupt increase in virulence in the spore population. From 23 to 44% of seedlings previously classified as resistant, some of which had remained rustfree for as long as 14 yr under conditions of chronically high disease hazard, became infected between 1976 and 1978 (Table 1). Many of these sustained multiple, sporulating infections no different from those in susceptible genotypes.

On the other hand, it is evident that rust biotypes virulent to MGR have been present at low and apparently stable frequency in the area of this test site for many years. From 2 to 8% of progeny from parents homozygous for MGR in eight different planting years became infected prior to 1976 (Fig. 1). The sharp increase in infection on resistant genotypes observed subsequently was associated with unusually early and prolonged climatic conditions favorable for inoculum production, dissemination, and infection during the summer of 1976. Inoculum potential could have been far above usual levels under these conditions, as indicated by the nearly complete infection within 2 yr of control trees planted in 1975 (Fig. 1). Thus, a large increase in absolute numbers of virulent spore genotypes could have the same effect as an increase in the relative frequency of those genotypes.

By analogy with the notorious lability of major resistance genes to virulent races in cereal-rust pathosystems, it has been argued that the use of major genes in forest trees would be futile (6,27). The analogy cannot be ignored, but evidence is lacking. Little consideration has been given to the great disparities that exist between domesticated annuals and wild perennials in life history, genetic structure, ecological diversity, and crop management, that may profoundly affect the racial composition and epidemiology of their pathogens. Vanderplank (25,26) introduced the concept of stabilizing selection in plant pathogens, which he said occurs as a consequence of reduced fitness of unnecessary genes for virulence in complex races on host plants lacking major resistance genes. Person et al (22) formulated the dynamics of stabilizing and directional selection in mixed host genotype populations, based on assumed values in reduced fitness of virulent alleles on plants lacking MGR and the proportion of susceptible interactions that occurred in the total pathosystem. Since sugar pines will be used in mixed species plantings established in mosaics among larger blocks of wild populations and in very diverse environments, stabilizing selection that would mitigate rapid replacement of avirulent with virulent races is a distinct possibility. It is still controversial whether reduced fitness of more complex races on susceptible hosts (and therefore stabilizing selection) occurs as a general phenomenon in plant pathogen populations (18). The sugar pine-blister rust pathosystem provides an unusually good opportunity for testing this hypothesis in a wild population, because new assay techniques permit direct and rapid estimation of changes in frequency of the virulent allele (8,9).

Slow rusting. The vulnerability of MGR emphasizes the need for utilizing other resistance mechanisms. The measurement of relative levels of resistance among different genetic units as a function of their rate of infection over time was proposed by Vanderplank (25,26). In sugar pine, average infection rates of progenies of individual parents ranged from 0.247 to 0.812, with specific families as low as .078 (Table 2).

Families of all selected trees had lower average infection rates than the controls, but without further improvement most of them would still be too susceptible for use in commercial silviculture under moderate to high disease hazard. The quantitative pattern of variation, however, indicates that continued selection and breeding for slow rusting should be effective. When selection of the top six of the 17 parents evaluated in this study was simulated, the estimated average infection rate was reduced by two thirds, compared with controls (0.272 vs 0.883). Individual trees from slow rusting parents that survive to sexual maturity should have more genes for resistance than either of their parents. Although these empirically derived conclusions require proof by independent tests, the possibility of rapid improvement in early breeding generations is strongly suggested.

Mature tree resistance. The presumption of inherent variability in resistance among mature trees was implicit in the selection of phenotypically resistant candidates in heavily infected stands. This

TABLE 3. White pine blister rust infection on selected sugar pine parents, cohorts, and clones

	Infection (%)	Range of cankers per infected tree (no.)
Parent trees (ortets)[a]	26	1–4
Cohorts	98	1–100 +
Clones	47[b]	1–9.5[c]

[a] Fifty-seven parent trees, with 3–10 cohorts each, of similar age and size.
[b] Percent of clones with one or more ramets infected. Among clones, the proportion of ramets infected ranged from 0 to 100%.
[c] Averaged over all infected ramets for each clone.

was confirmed by the wide range of infection (0–100%) among different clones (Tables 2 and 3). The mechanisms and inheritance of mature tree resistance are not known but appear to be both strong and relatively stable. Apparently they are unrelated to both MGR (since the progeny of many resistant clones were highly susceptible) and to slow rusting resistance, since parent-offspring correlations were low. Interesting in this respect were rustfree or lightly infected clones, such as K41, K36, and K14, that produced some of the most susceptible offspring (Table 2). Very similar patterns of differences between select trees and cohorts, and between grafted ramets and seedlings, were found in western white pine (2). Patton (21) also showed a consistent and progressive decrease in susceptibility of eastern white pine ramets as the age of the ortet increased. Results of all these studies, taken together, suggest that ontogenetic factors for resistance to blister rust are pervasive in white pines, but that their strength and expression varies greatly with genotype.

Although the mode of inheritance will be difficult to determine, mature tree resistance has a potentially valuable role in stabilizing the overall resistance of a crop throughout its rotation. We consider it particularly significant that none of the ramets known to have MGR was infected following the 1976 epidemic.

Prospects for genetically controlling white pine blister rust in sugar pine are good. MGR is highly effective, and its deployment into planting stock could be rapidly achieved. Though its durability might be limited by an increase in frequency of virulent races, we do not consider this outcome inevitable, or the risk great enough to preclude its use, at least for the near term. Meanwhile, the traits of slow rusting and mature tree resistance can be developed independently by selection and breeding of survivors in progeny and clonal tests. Ultimately, the integration of all three types of resistance should be feasible and result in populations with genetically well-buffered, highly effective, and enduring resistance.

LITERATURE CITED

1. Bartos, P., Dyck, P. L., and Samborski, D. J. 1969. Adult-plant leaf rust resistance in Thatcher and Marquis wheat: a genetic analysis of the host-parasite interaction. Can. J. Bot. 47:267-269.
2. Bingham, R. T. 1966. Breeding blister rust resistant western white pine. III. Comparative performance of clonal and seedling lines from rust-free selections. Silv. Genet. 15:160-164.
3. Bingham, R. T., Hoff, R. J., and McDonald, G. I. 1971. Disease resistance in forest trees. Annu. Rev. Phytopathol. 9:433-452.
4. Bingham, R. T., Hoff, R. J., and McDonald, G. I. 1973. Breeding blister rust resistant western white pine. VI. First results from field testing of resistant planting stock. U.S. Dep. Agric., For. Serv. Res. Note INT-179. Odgen, UT. 12 pp.
5. Browning, J. A. 1974. Relevance of knowledge about natural ecosystems to development of pest management programs for agro-ecosystems. Proc. Am. Phytopathol. Soc. 1:191-199.
6. Hoff, R. J., and McDonald, G. I. 1972. Stem rust of conifers and the balance of nature. Pages 525–535 in: Bingham et al, eds. Biology of Rust Resistance in Forest Trees. U.S. Dep. Agric., For. Serv., Misc. Publ. 1221. Washington, DC. 681 pp.
7. Hoff, R. J., McDonald, G. I., and Bingham, R. T. 1976. Mass selection for blister rust resistance: a method for natural regeneration of western white pine. U.S. Dep. Agric., For. Serv Res. Note INT-202. 11 p.
8. Kinloch, B. B., Jr., and Comstock, M. 1980. Cotyledon test for major gene resistance to white pine blister rust in sugar pine. Can. J. Bot. 58: 1912-1914.
9. Kinloch, B. B., Jr., and Comstock, M. 1981. Virulent race of *Cronartium ribicola* to major gene resistance in sugar pine confirmed. Plant Dis. 65:(In press).
10. Kinloch, B. B., Jr., and Littlefield, J. L. 1977. White pine blister rust: hypersensitive resistance in sugar pine. Can. J. Bot. 55:1148-1155.
11. Lupton, F. G. H., and Johnson, R. 1970. Breeding for mature plant resistance to yellow rust in wheat. Ann. Appl. Biol. 66:137-143.
12. MacKenzie, D. R. 1976. Application of two epidemiological models for the identification of slow stem rusting in wheat. Phytopathology 66:55-59.
13. McDonald, G. I. 1978. Segregation of "red" and "yellow" needle lesion types among monoaeciospore lines of *Cronartium ribicola*. Can. J. Genet. Cytol. 20:313-324.
14. McDonald, G. I., and Hoff, R. J. 1971. Resistance of *Cronartium ribicola* in *Pinus monticola:* genetic control of needle-spots-only resistance factors. Can. J. For. Res. 1:197-202.
15. McDonald, G. I., and Hoff, R. J. 1975. Resistance to *Cronartium ribicola* in *Pinus monticola*: an analysis of needle spot types and frequencies. Can. J. Bot. 53:2497-2505.
16. Miekle, J. L. 1938. Spread of blister rust to sugar pine in Oregon and California. J. For. 36:695-701.
17. National Oceanic and Atmospheric Administration. 1976. Climatological Data: California. Annual Summary. Vol. 80, No. 13. National Climatic Center, Asheville, NC. 33 pp.
18. Nelson, R. R. 1972. Stabilizing racial populations of plant pathogens by use of resistance genes. J. Environ. Quality 1:220-227.
19. Nelson, R. R. 1978. Genetics of horizontal resistance to plant diseases. Annu. Rev. Phytopathol. 16:358-378.
20. Parlevliet, J. E. 1978. Components of resistance that reduce the rate of epidemic development. Annu. Rev. Phytopathol. 17:203-222.
21. Patton, R. F. 1961. The effect of age upon susceptibility of eastern white pine to infection by *Cronartium ribicola.* Phytopathology 51:429-434.
22. Person, C., Groth, J. V., and Mylyk, O. M. 1976. Genetic change in host-parasite populations. Annu. Rev. Phytopathol. 14:177-188.
23. Simons, M. D. 1972. Polygenic resistance to plant disease and its use in breeding resistant cultivars. J. Environ. Qual. 1:232-240.
24. Sztejnberg, A., and Wahl, I. 1976. Mechanisms and stability of slow stem rusting resistance in *Avena sterilis.* Phytopathology 66:74-80.
25. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
26. Vanderplank, J. E. 1975. Principles of Plant Infection. Academic Press, New York. 216 pp.
27. Wood, F. A. 1966. The current status of basic knowledge of forest tree disease resistance research. Pages 293–300 in: H. D. Gerhold et al, eds. Breeding Pest Resistant Trees. Pergamon Press, Oxford. 505 pp.