United States
Department of
Agriculture

Forest Service

**Pacific Southwest
Research Station**

Research Paper

PSW–204

White Pine Blister Rust at Mountain Home Demonstration State Forest: A Case Study of the Epidemic and Prospects for Genetic Control

Bohun B. Kinloch, Jr. **David Dulitz**

Kinloch, Bohun B., Jr.; Dulitz, David. 1990. **White pine blister rust at Mountain Home Demonstration State Forest: a case study of the epidemic and prospects for genetic control.** Res. Paper PSW-204. Berkeley, CA: Pacific Southwest Research Station, Forest Service, U.S. Department of Agriculture; 7 p.

The behavior of white pine blister rust at Mountain Home State Demonstration Forest and surrounding areas in the southern Sierra Nevada of California indicates that the epidemic has not yet stabilized and that the most likely prognosis is a pandemic on white pines in this region within the next few decades. The impact on sugar pines, from young regeneration to old growth, already has been severe in some areas, and silvicultural control measures have been largely ineffective. An operational genetic program, based on selection of seed parents carrying a major resistance gene, is described that is simple and effective. This program will be useful in maintaining sugar pine as a crop species in artificial regeneration for at least one rotation, and in preserving the genetic integrity of sugar pine populations in jeopardy from the rust.

Retrieval Terms: Pinus lambertiana, sugar pine, *Cronartium ribicola*, genetic resistance

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Acknowledgments:

The research reported herein was supported in part by a competitive grant awarded by the California Department of Forestry and Fire Protection.

Cover: Parent-offspring relationships of sugar pine to white pine blister rust infection: uninfected seed parents in heavily infected stands (like the one at top left) often transmit their resistance to seedling offspring *(lower left)*. Infected parents *(top right)* consistently produce susceptible offspring *(lower right)*.

Publisher:

Pacific Southwest Research Station
P.O. Box 245, Berkeley, California 94701
November 1990

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This paper describes a case study of the impact and prognosis of the white pine blister rust epidemic on sugar pine in a typical mid-elevation, mixed-conifer forest in the southern Sierra Nevada of California, and implementation of a simple, practical program for selecting genetically resistant parents for reforestation.

The test area was Mountain Home Demonstration State Forest (MHDSF), a second-growth, mixed-conifer forest with 38 percent of its volume in sugar pine (estimate excludes remaining old-growth giant sequoias). Blister rust reached the forest in 1961, but did not become epidemic until the 1970's. In 1985, 25 percent of the sugar pines were either dead or lethally infected, by conservative estimate. Rust-associated mortality included many pole-sized or larger trees weakened by a combination of rust and drought, then killed by bark beetles. Previous attempts at control by eradicating alternate hosts or pruning branch infections from pines were ineffective. The pattern of behavior of the rust at MHDSF and elsewhere in the Sierra Nevada in recent years clearly shows that the epidemic has not stabilized in this region, as had been thought, and can be expected to spread in the future.

Seedlings from 170 selected rust-free trees were screened by artificial inoculation to identify seed parents carrying a major gene for resistance (MGR) to the rust, and to estimate the frequency of the gene in the Forest. With a calculated frequency for MGR of 0.076 in the sample seedling population, the expected proportion of resistant parent genotypes was 15 percent. The realized proportion was 23 percent, the difference being attributed to selecting against any seed parent with visible rust infection.

Small demonstration plantings were established in 1983 and 1984 to test the efficacy of genetic selection for rust resistance under conditions prevailing on the Forest. Although the 1984 planting has experienced only light infection (6 percent), 80 percent of susceptible controls in the 1983 planting became infected, as a result of unusually favorable conditions for rust spread throughout large parts of the Sierra Nevada in that year. None of the seedlings known to carry MGR in either test became infected.

Because of its simplicity and efficacy, this kind of program is feasible for different kinds and sizes of land ownerships interested in commercial production of sugar pine, as well as for conservation efforts aimed at preserving the genetic integrity of sugar pine populations.

INTRODUCTION

A decade ago, the prospect of managing sugar pine under the threat of white pine blister rust in California was optimistic. Compared with the Cascades and northern Rocky Mountains, the relatively hostile environment for the disease in the Sierra Nevada, it was thought, would constrain the epidemic to erratic outbreaks that could be coped with largely by silvicultural prescriptions (Byler and Parmeter 1979; MacGregor 1969). Infection would be intense in some places, absent in others, and overall at a moderate level, perhaps somewhat less than 20 percent. Most infection on sugar pine (*Pinus lambertiana* Dougl.) would occur in regeneration, or low in the crown of larger trees; trees escaping infection until they became 8 inches or greater dbh would probably survive to harvest. Effective management could rely on a judicious combination of site hazard rating to decide on areas for concentrated management of sugar pine, and removal or pruning of "lethally" infected trees—those with infections coming within two feet of the bole (Byler and Parmeter 1979).

None of these perceptions has held. The survey data on which they were based were taken in the late 1960's and early 1970's; but in 1976 and again in 1983, unusually favorable conditions produced "wave" years over large areas of California, spreading the disease into many new areas of the central Sierra Nevada and intensifying infection in older centers. The explosive epidemic that developed on the Mountain Home Demonstration State Forest and surrounding areas since the mid-1970's showed that even in the relatively warm, dry southern Sierra Nevada infection can occur more frequently and extensively than previously thought; at least occasionally, climatic conditions there can be very favorable for spread of blister rust. In places, infection is so intense that it occurs high in the crowns of mature trees, killing pole-size and larger trees directly, or indirectly by predisposing them to bark beetle attack.

This paper illustrates some of these phenomena in a case study that may anticipate what lies ahead for middle elevation stands throughout the Sierra Nevada. It also describes an operational plan, based on genetic principles and proven mechanisms of resistance in sugar pine, that has been tested and can be used to effectively regulate the amount of disease for at least the next rotation of improved planting stock. This plan is simple in concept and execution, and could be readily implemented by relatively small landholders.

The Disease

White pine blister rust is caused by *Cronartium ribicola* Fisch., a fungus inadvertently introduced into western North America at Vancouver Island, British Columbia, in 1910 on a shipment of infected eastern white pine seedlings from a nursery in France. Like many other rust fungi, its life cycle is complex, with five different spore stages on two completely unrelated hosts: pines in the subgenus Strobus (white pines), and gooseberries and currants in the genus *Ribes*. These shrubs abound in the understories of western coniferous forests, with over a dozen species within the range of sugar pine. The spore stage infecting pine is produced in late summer or fall on the undersurface of leaves under cool, very moist conditions, which must be sustained until the spore has been transported by wind to pine foliage and has a chance to germinate and penetrate through a stomatal opening. The mycelium of the fungus then grows down the needle until it reaches the bark of the branch or bole, where it does most of its damage. Bark tissues become swollen and eventually produce two other spore stages. After a few years, this tissue may collapse and becomes a necrotic canker, girdling and killing the shoot; or, it may attract *Dioryctria* spp. (Lepidoptera: Pyralidae), whose larvae tunnel inside the bark as they feed, often vectoring secondary fungal pathogens. While large, older trees can tolerate the loss of many branches in this manner, infected seedlings or saplings almost inevitably die.

The Forest

Mountain Home Demonstration State Forest (MHDSF) occupies 4,600 acres on the western slope of the southern Sierra Nevada in Tulare County. It lies between the north and middle forks of the Tule River at middle elevations, between 4,500 and 7,500 feet, on gentle to steep slopes. Precipitation averages 42 inches per year, all concentrated in the winter months. Site quality is generally high, with 91 percent of the area Dunning site II or better.

Most of the forest is typical second-growth mixed conifer (SAF Type 243; Eyre 1980), with approximately half of the area in the giant sequoia (*Sequoiadendron giganteum* [Lindl.] Buchholz) phase of that type. More than 5,000 giant sequoias on the forest are greater than 40 inches dbh (half of these exceed 84 inches), and have a total volume of about 80 MMbf. Other conifers total about 111 MMbf; their proportionate volumes are white fir (*Abies concolor* [Gord. & Glend.] Lindl. ex Hildebr.) 41 percent; sugar pine 38 percent; incense-cedar (*Calocedrus decurrens* Torr.) 9 percent; and ponderosa pine (*Pinus ponderosa* Dougl. ex Laws.) 7 percent. Young-growth giant sequoia, considered separately from old growth, makes up the final 5 percent.

The most valuable stumpage is in sugar pine. About 284,000 sugar pines grow on the forest, comprising 23 percent of the basal area (excluding old-growth giant sequoias) and 9.2 Mbf per acre. Pole-size and larger (>12 inches dbh) trees make up 13 percent of the sugar pine component and advanced reproduction (<2 inches dbh) nearly half (49 percent).

Alternate host *Ribes* are abundant on the forest. The most prevalent species are the Sierra gooseberry (*R. roezlii* Regel.) on drier, upland sites, and the Sierra currant (*R. nevadense* Kell.)

near streams and more mesic sites. Both are highly susceptible to the rust, but not greatly damaged by it.

The Epidemic

Blister rust was discovered on sugar pine at MHDSF in 1968, from initial infections dated to 1961. More infection occurred in 1964, 1967, and 1969, but even as late as 1972 most of it was confined to 700 acres on the Bear Creek drainage and a total of about 90 acres on three other nearby stream sites. The pattern of infection in the early epidemic clearly indicated spread from locally produced inoculum that resulted from the original infections. Relatively little rust was found on upland sites after the first 12 years of exposure, and most infections were non-lethal, on low-lying branches.

In the 1970's the amount of infection started to increase dramatically, occurring higher in the crowns of the trees and spreading rapidly to other parts of the forest and onto adjacent private lands. To quantify the impact of the disease, a systematic survey was initiated in 1980, to recur at five-year intervals. The amount of infection on both sugar pine and *Ribes* was recorded on 119 0.2-acre, fixed, Continuous Forest Inventory (CFI) plots on a 20-by-20 chain grid covering the entire forest. Current and estimated future damage by the disease on individual sugar pines was evaluated according to the proximity of infections to the bole (Byler and Parmeter 1979); infections on or within 24 inches of the bole are considered lethal.

Results of the 1980 survey showed that 78 percent of the plots had *Ribes* growing on them, of which 89 percent were infected. Sugar pine was present on 89 percent of the plots. Rust was on 28 percent of sugar pines in 1980, and increased to 36 percent 1985 *(fig. 1)*, with 25 percent of sugar pines either dead or lethally infected. Intensity of infection varied greatly: on 15 percent of the plots all sugar pines were infected, while on 19 percent of the plots none were infected *(fig. 2)*. The greatest increase in infection (from 21 to 31 percent) took place in diameter classes 12 inches dbh and above.

Grim as they are, these statistics do not convey the full impact the disease is having on the sugar pine population in this forest. They are conservative, because they do not reflect the many thousands of infected sugar pines removed before the surveys—in some areas up to 80 percent. And, contrary to earlier surveys elsewhere in the Sierra Nevada (MacGregor 1969; Byler and Parmeter 1979), where infection was largely confined to young seedlings and saplings, or low-lying branches of larger trees, in recent years at MHDSF susceptible old-growth sugar pines are being attacked throughout their crowns, up to 180 feet high. Incidence of bole cankers and top-kill has increased greatly. Pole-size trees up to 20 inches dbh are being killed outright, and more than half of the crowns of some large veterans have been killed by multiple branch infections. An ominous development we have frequently observed is bark beetles attacking trees weakened and dying of rust, then subsequently attacking neighboring (potentially resistant) trees having no infection.

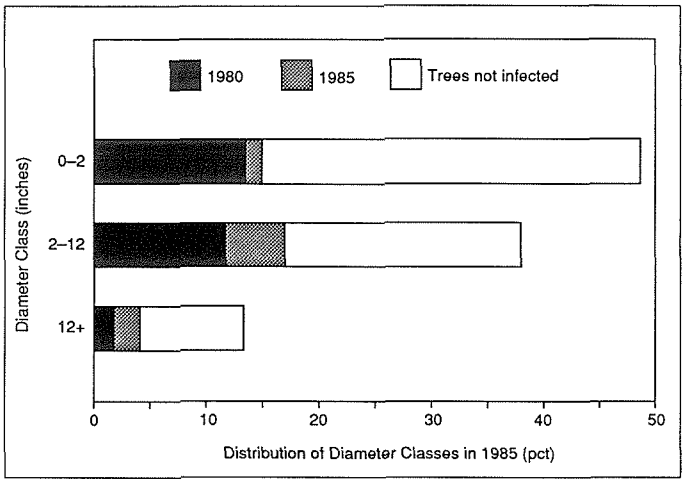

Figure 1—Distribution of diameter classes of sugar pines on Mountain Home Demonstration State Forest in 1985, and percentage of trees infected in each in 1980 and 1985.

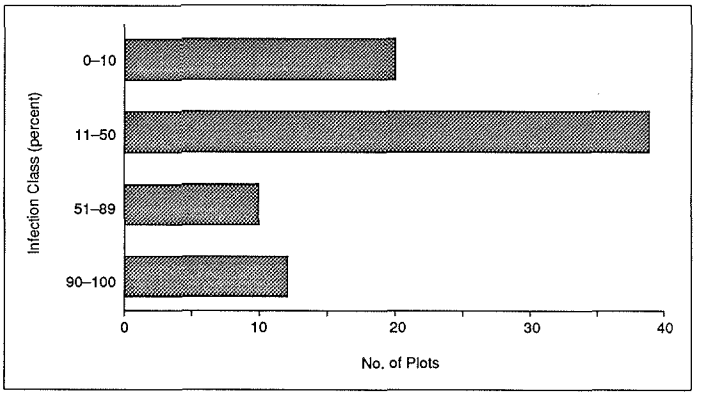

Figure 2—Range of infection of white pine blister rust on sugar pines in 81 sample plots on Mountain Home Demonstration State Forest.

Previous Attempts at Control

In the early stages of the epidemic in parts of the western United States, breaking the life cycle of the blister rust fungus by eradicating alternate host *Ribes* spp. was the indicated control strategy. In anticipation of the rust's eventual arrival, this practice was followed at MHDSF on selected sugar pine management areas for almost two decades before the disease was actually detected. Then, from 1968 through 1978, more direct control measures were attempted: the limits of infection centers were demarcated, and all sugar pines with bole infections (or branch infections within 12 inches of the bole) within their boundaries were removed. Crop trees were selected on the basis of size, spacing, and absence of rust, then pruned to a height of 18 feet. Most of the smaller trees (less than 36 feet tall) were also removed. None of these measures was very effective, however, and the epidemic continued unabated. Data from the 1985 survey showed that the pruned trees actually had slightly more rust infection than untreated trees.

Yet, many sugar pines, old and young, remained uninfected year after year. Since these were often surrounded by heavily

Figure 3—Blister rust flags high in the crowns of old-growth sugar pines at Mountain Home Demonstration State Forest.

Figure 4—Severe crown damage from blister rust infection on mature sugar pine.

infected cohorts, a genetic basis for their continued resistance seemed likely.

THE GENETIC OPTION

The prospect that rust could be controlled genetically was greatly enhanced by the discovery that strong, simply inherited resistance exists in some sugar pines (Kinloch and others 1970). Because the single gene conditioning resistance is dominant, at least half of the offspring of any resistant parent carrying the gene will also be resistant, irrespective of the other parent. Such carrier parents are easily identified by segregation of open-pollinated seedlings into Mendelian ratios (50 percent resistant:50 percent susceptible) after natural infection in the field, or artificial inoculation in the greenhouse. Resistance is expressed within a few weeks after inoculation by the kind of symptom produced on leaves, the normal infection courts of the pathogen. Susceptible seedlings develop bright yellow spots that continue to enlarge, whereas on resistant seedlings the spots remain small and soon turn brown, because of dead and dying host cells in and around the spot reacting hypersensitively to invasion by the fungus (Kinloch and Comstock 1980).

Parent Selection and Progeny Testing

Selection of candidate seed parents on MHDSF was begun in 1981 and continued over several years. Criteria for selection were that the candidate be free of rust (as determined by careful binocular inspection), bear cones, and have average or better growth and form.

From two to four ripened cones were shot from trees with a .22 caliber rifle in late August. Seed was extracted at the California Department of Forestry nursery at Davis, cold stratified for three to four months at the USDA Forest Service's Institute of Forest Genetics, Placerville, and sown, two to a container, into Leach Super Cells.[1] Seed parent identities were maintained throughout. From 28 to 98 seeds of each seed parent were sown.

Seedlings were inoculated with blister rust about two months after germination, using procedures already described (Kinloch

[1]The use of trade or firm names in this publication is for reader information and does not imply endorsement by the U.S. Department of Agriculture of any product or service.

Table 1—*Identification of resistant sugar pine seed parents and frequency of a major gene (R) for resistance to white pine blister rust in their wind-pollinated progenies*

Seedlings				Freq. of R	Mendelian ratio tested[1]	Inferred genotype of parent[2]
Parent	Resis.	Suscep.	Total			
7	26	22	48	0.541	1:1	Rr
61	12	16	28	0.428	1:1	Rr
200	1	21	22	0.045		rr
201	3	70	73	0.041		rr
202	2	57	59	0.033		rr
203	4	52	56	0.071		rr
204	1	37	38	0.026		rr
205	6	37	43	0.139		rr
206	43	39	82	0.524	1:1	Rr
208	23	13	36	0.638	1:1	Rr
209	3	10	13	0.230		rr
211	19	17	36	0.527	1:1	Rr
213	3	0	3	1.000	1:0	(RR)[3]
214	13	45	58	0.224	1:1	Rr
215	36	31	67	0.537	1:1	Rr
216	0	26	26	0.000		rr
217	34	26	60	0.566	1:1	Rr
218	20	25	45	0.444	1:1	Rr
219	5	34	39	0.128	1:1	Rr
220	23	18	41	0.560	1:1	Rr
221	6	36	42	0.142		rr
222	2	54	56	0.035		rr
223	17	24	41	0.414		rr
224	60	0	60	1.000	1:0	RR
226	6	78	84	0.071		rr
227	9	78	87	0.103		rr
228	0	12	12	0.000		rr
229	2	38	40	0.050		rr
230	1	9	10	0.100		rr
231	9	28	37	0.243	1:1**	rr
232	2	27	29	0.068		rr
233	1	38	39	0.025		rr
234	1	26	27	0.037		rr
235	1	6	7	0.142		rr
236	41	31	72	0.569	1:1	Rr
237	1	33	34	0.029		rr
238	19	38	57	0.333	1:1*	rr
240	2	87	89	0.022		rr
241	2	4	6	0.333	1:1	Rr
242	2	76	78	0.025		rr
243	0	15	15	0.000		rr
244	9	8	17	0.529		rr
245	5	79	84	0.059		rr
246	7	64	71	0.098		rr
247	0	17	17	0.000		rr
248	5	82	87	0.057		rr
249	18	21	39	0.461	1:1	Rr
250	15	18	33	0.454	1:1	Rr
251	5	2	7	0.714	1:1	Rr
252	33	26	59	0.559	1:1	Rr
254	5	5	10	0.500	1:1	Rr

[1]1:1 or 1:0 ratios (resistant:susceptible) tested against chi-square values with 1 d.f.; * and ** indicate departure from critical values at $P \leq .05$ and .01, respectively. Parents not tested (blank spaces) are assumed to be susceptible (rr) genotypes, but derive some resistant offspring from wind pollination.

[2]R and r are alternate alleles for resistance and susceptibility, respectively.

[3]Insufficient sample size for valid test.

and Comstock 1980). Briefly, leaves of the European black currant (*Ribes nigrum* L.) infected with the telial stage of blister rust were suspended on wire trays over test seedlings in a moist chamber for 48 hours. Basidiospores, the spore stage infective on pine, were cast naturally and abundantly onto seedlings from germinating teliospores, at spore densities ranging typically from 100 to 300 per mm^2. Few seedlings escaped infection under these conditions.

Foliage symptoms began to appear two to three weeks after inoculation and were diagnostic for each of the two phenotypic classes (resistant and susceptible) by eight weeks. The total number of seedlings in each class was tallied for each progeny and tested for segregation into Mendelian ratios for single gene inheritance by chi-square analysis.

The wind-pollinated families fell into three groups: those with mostly all susceptible seedlings (the largest group); next, those segregating in an approximate 1:1 ratio of susceptible to resistant; and last, a very few that were all resistant. An example is given in the data for the 1986 inoculation *(table 1)*. The seed parents of the two resistant progeny groups were interpreted as having the dominant allele for resistance in either single copy (heterozygous, Rr; half of the offspring resistant) or double copy (homozygous, RR; all offspring resistant). Progenies with no resistant seedlings were from seed parents homozygous for the recessive alleles (rr) conditioning susceptibility. Those with predominantly susceptible offspring, but with a few resistant seedlings mixed in, were also from homozygous recessive seed parents, but derived their resistant siblings from wind pollination from an unknown pollen donor carrying the resistant allele. In a few progenies with low germination but relatively high numbers of resistant offspring, it was difficult to determine whether the seed parent had received above-average amounts of resistant pollen (perhaps from a near neighbor) or was actually a resistant genotype itself. These cases were resolved by chi-square analysis (5 percent probability level), or by retesting the seedlot the following year with greater numbers of offspring.

Knowing which parents were homozygous recessive genotypes (rr) enabled an easy and direct estimate of the frequency of the resistance allele (R) in the ambient pollen cloud. Since a genetically susceptible seed parent can produce only a single kind of egg gamete (r), any resistant seedlings coming from such a parent *must* derive their resistance from the pollen parent. The relative frequencies of r and R pollen gametes are derived by simply counting resistant and susceptible offspring in each progeny. The data provide two estimates of the frequency of R: the gametic frequency from pollen donors, and the zygotic frequency from the seed parents themselves. In a completely random sample of seed parents, the two estimates should be equivalent.

Results are shown in *table 2*. Of 170 seed parents tested over the years, 39 were resistant genotypes: 36 heterozygotes and three homozygotes. The data show an overall and consistent gametic frequency of about .07-.08 for R over the three seed years sampled. The zygotic frequency (weighted average) was considerably higher (.12). Although not nearly as consistent among test years, sample sizes were much smaller for seed trees (zygotes) than for their progeny (the measure of gametic gene

Table 2—*Zygotic and gametic frequencies of the R allele for resistance to white pine blister rust in sugar pine populations on the Mountain Home Demonstration State Forest*

Seed year	Seed parents: Tested	Genotypes recovered[1] RR	Rr	rr	R freq. (zygotic)	Seedlings[2] Tested	Resis.[3]	R freq. (gametic)
	No.					*No.*		
1981	33	2	6	25	.152	595	43	.072
1982	86	0	11	75	.064	938	75	.080
1985	51	1	19	31	.196	1372	103	.075
Total/mean:	170	3	36	131	.115[3]	2905	221	.076[4]

[1]R and r are alternate alleles for resistance and susceptibility, respectively.
[2]From susceptible (rr) parents.
[3]Resistance (R allele) donated from unknown pollen parent.
[4]Weighted average.

frequency). We interpret the higher zygotic frequency as an effect of the phenotypic selection imposed against susceptible seed parents.

Field Tests

To demonstrate the efficacy of genetic resistance under natural conditions at MHDSF, we planted some wind-pollinated progenies of tested resistant seed parents in replicated field tests. In 1983, one-year-old seedlings of two families from homozygous (RR) resistant seed parents were planted on a flat, open site in a natural depression; the next year the same progenies plus four others from heterozygous (Rr) parents were planted in a different area of the forest on a 30 percent south-facing slope. Susceptible controls were planted in both tests.

Survival was poor on both sites, and infection in the larger test on the dry, south-facing slope has been light—only 6 percent overall. But in the smaller test, infection was much heavier (undoubtedly, the result of epidemic conditions in 1983, not duplicated since), with 80 percent of controls showing positive stem infections by 1987. None of the seedlings from homozygous resistant parents was infected in either test.

DISCUSSION

The Epidemic: Evaluation and Prognosis

MHDSF was the first area in the southern Sierra Nevada to be hit by the rust, over 30 years after its entry into California from Oregon, and almost 20 years after reaching Dodge Ridge on the Stanislaus National Forest, only 150 miles to the north. Blister rust is predominantly a cool-weather disease, and conditions for its spread to pines become less frequent and of shorter duration from north to south (Kimmey and Wagener 1961). Evidently, the gradient of increasing temperature and aridity from north to south has retarded the spread of the disease in the Sierra Nevada, in comparison with its relatively rapid pace in the cooler, more moist climates of the Cascade and Coast Ranges of British Columbia, Washington and Oregon.

Infection is still patchy throughout much of the Sierra—intense in some places, light or absent in others. Although the dynamics of the epidemic are still incompletely understood, the following scenario is consistent with evidence presented here, evidence of recent surveys on nearby National Forests (Kliejunas 1982, 1984), and observations elsewhere.

New infection centers arise by long-distance spread of aeciospores from pine to *Ribes* in the spring. These thick-walled spores, adapted to resist desiccation, have been documented to cause new infection on *Ribes* from as far as 300 miles away (Mielke 1943). The rust must recycle and persist on *Ribes* throughout the long, dry summer until cool, moist conditions return in autumn. But a balance must prevail; infection causes stress to the plants and, if too severe, leaves may shed prematurely. An early frost, or a prolonged dry autumn, can have the same effect. In either case, the disease cycle is broken, with little jeopardy to pine unless and until inoculum from distant infected pines reenters the area another spring.

Ironically, the odds of pine infection in any given year or site are low. The basidiospores that infect pine are delicate, requiring cool temperatures for their production on *Ribes*, high relative humidities for dissemination on air currents, and free moisture for germination and penetration of pine needles. Because these conditions are both exacting and transient, pine infection usually occurs only in the autumn, if at all, and almost always from nearby sources of inoculum.

The coincidence of favorable weather with adequate amounts of inoculum to infect pine becomes more likely to occur as the

availability of inoculum in an area increases generally. From initial point foci, infection of pines tends to spread more or less concentrically until separate infection centers eventually coalesce. Meanwhile, inoculum is building up locally, so that some new infection usually recurs every year. Then, in especially favorable "wave" years, the stage is set for such new infection to become widespread and devastating. Even so, intensity can vary greatly from site to site within areas as small as MHDSF, as we have documented above (*fig. 2*).

Risk to sugar pine in plantations is much greater than to natural stands, and attempts to classify site hazard based on general ecological relationships prevailing in natural stands have not worked well. In a survey of 29 plantations in the northern and central Sierra Nevada, average infection of sugar pine was 69 percent, with no clear relationship between infection intensity and any major environmental feature, such as aspect, slope, proximity to water, or amount of *Ribes* in or near the plantation (DeNitto 1987). Although experimental evidence is lacking, this extreme vulnerability of plantations is undoubtedly due in large part to the known drastic changes in microclimate caused by clearcutting. Proliferation of *Ribes* plants, which are specially adapted to sprout from roots or to germinate from dormant seed following severe site disturbance, may increase the inoculum potential by orders of magnitude. Exposure of the site to open sky by removal of the canopy is very conducive to dew formation on foliage of seedlings low to the ground, which in turn enhances spore germination and host penetration.

Results of recent surveys in natural stands (Kliejunas 1982, 1984), as well as our own experience at MHDSF, dampen optimism expressed in the previous two decades (MacGregor 1969; Byler and Parmeter 1979) that the epidemic in the Sierra Nevada has become relatively stabilized, and that practical control can be effected by sanitation of lethally infected trees and judicious pruning of branch infections before they become lethal. A more likely prognosis is that the disease will become pandemic on sugar and western white pine (*Pinus monticola* Dougl.) populations within the next few decades. In any case, the prospects of regeneration, natural or artificial, are extremely bleak without resistance.

Genetic Control: The Outlook

In our program, we were able to identify 39 genetically resistant seed parents out of a total of 170 selected and tested *(table 2)*, a yield of 23 percent. In stands with little or no rust infection, seed parents must be selected more or less randomly with respect to their rust phenotype. In these cases, the potential yield of resistant parents is simply a function of the frequency of R in the stand. Knowing the frequency, one can predict the proportion of selected parents that will carry the gene, or determine the number of parents that must be screened to obtain a given number of resistant genotypes. At low frequencies (between 0.0 and 0.1) the number of resistant genotypes (R-) will be approximately double the gene frequency (because each seed parent tested carries two copies of every gene locus). In our case, for example, with a gene frequency of almost 0.08 we would expect about 15 percent of randomly selected parents to have major gene resistance (MGR). Because we were able to identify and reject many of the highly susceptible trees, under the epidemic conditions prevailing at MHDSF, the efficiency of our phenotypic selection was increased by 53 percent over random selection.

However, even in stands with high and uniform infection, most trees with phenotypic resistance will not necessarily have MGR. Other kinds of resistance exist that are more complexly inherited. One of these is a resistance that develops with age, but is not expressed in young seedlings (Kinloch and Byler 1981). An undesirable effect of this kind of resistance is to mask juvenile susceptibility.

Because of the simplicity and efficiency of the screening test, yield of resistant seed parents will usually justify the cost of selection and testing, even when frequencies of R are much lower than at MHDSF and even when no rust disease is present in a stand to aid in eliminating susceptible genotypes. In either case, selection for other economic traits (growth, form, fecundity, etc.) can be done simultaneously. Even though resistance and phenotypic superiority in other traits are unlikely to coincide frequently, both can be had by selection of resistant individual seedlings from superior (but susceptible) seed parents. The seed parent will contribute half of its additive genetic variance for superiority, while resistance will be captured from an unknown pollen donor.

Although the tests we have described are not feasible for most landholders to conduct, the USDA Forest Service's test facility on the Eldorado National Forest is currently providing this service for the National Forests as well as other public and industrial landholders. Once identified, resistant seed parents can be put to use immediately for reforestation. Assuming average cone harvests of from 10 to 50 cones per tree and 150 seed per cone (Critchfield and Kinloch 1986), annual seed yields would range between 1500 and 7500 per tree, half of which would have MGR.

How many MGR seed parents are needed for a given area? For MHDSF, we set a target of 50. This somewhat arbitrary figure approximates one parent for every 100 acres, which we judged a reasonable compromise between cost, seed supply with ample reserves, and the preservation of genetic diversity on the forest. For reforestation, we will use bulk mixtures of seed from our resistant parents. We will further enhance long-range conservation of genetic diversity by preserving all seedlings from susceptible seed parents that have captured MGR from unknown pollen donors in archive plantings maintained by the USDA Forest Service. This option enables a virtually unlimited amount of the extant genetic variability of sugar pine on this (or any other) forest to be sampled and preserved, if exercised before the epidemic takes a much greater toll.

Although MGR is very strong, it is vulnerable to genetic variation in the pathogen. A race capable of completely overcoming MGR was discovered in a test plantation with resistant progenies in the Klamath National Forest (Kinloch and Comstock 1980). However, this race has not been found elsewhere, and has not spread out from the progeny test plantation, even though *Ribes* and sugar pine abound in the area (Kinloch and Dupper

1987). Although eventual recurrence and spread of this and similar races are probably inevitable, neither is likely to happen very rapidly, or simultaneously over wide areas. In other words, it took over 30 years for the common race to get from northern California to the southern Sierra Nevada; there is no reason to believe that the virulent race will spread any faster.

Meanwhile, other mechanisms of resistance not vulnerable to the race of rust virulent to MGR are being identified (Kinloch and Byler 1981). A realistic but long-range goal is the combination of these mechanisms with MGR in the same individual. Such integration of independent mechanisms will have a much greater probability of achieving effective, well buffered, and durable resistance. In the meantime, use of MGR by itself can continue to provide a large measure of protection from blister rust.

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