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# Breakdown of major gene resistance to white pine blister rust in sugar pine at Mountain Home Demonstration State Forest: what are the implications?

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A virulent race of blister rust capable of neutralizing major gene resistance (MGR) in sugar pine made its first appearance nearly two decades ago at a test plantation of resistant sugar pines near Happy Camp, in northern California. Until this year (1996), it had not been found outside the very close neighborhood of this site. Its discovery last summer at Mountain Home Demonstration State Forest (see Adams, preceding paper) was thus alarming, because of the implications this held for federal, State, and industry selection programs based on deploying MGR in commercial plantations. Mountain Home is a strategic test site for assessing the efficacy of genetic resistance to control blister rust. The forest is well inventoried; the impact of blister rust has been closely observed and documented over the years; and selection and deployment of MGR has been implemented in a model program (Dulitz et al. 1996; Kinloch and Dulitz 1990). Was this an entirely new race? Or had the race at Happy Camp migrated over 500 miles south in one leap? Or had it also spread to other places along the way, as yet undetected?

We have studied the behavior and epidemiology of the Happy Camp race for almost two decades. Much of this has been described previously (Kinloch and Comstock 1981; Kinloch and Dupper 1987; Kinloch et al. 1996). Here, I will try to put the discouraging recent news into the perspective of this experience.

## Biology of the virulent race

We all have an intuitive and general understanding that a disease is "virulent" on its host, in the sense that if it causes infection it does harm. But in plant pathology virulence has a more special and technical meaning: the capacity of a pathogen to overcome a specific kind of resistance mechanism in its host, a mechanism usually controlled by a single gene. This capacity is enabled by mutation at a single gene locus in the pathogen (called a virulence allele) which is specific to the host resistance allele in question. This is the basis of gene-for-gene interactions in host-parasite relationships.

In the present case, a specific race, strain, or genotype of rust overcomes a single dominant gene (R) for resistance in sugar pine. Presumably, the rust's ability to do this is also conditioned by a single gene (vR), although formal proof is lacking. Other resistance mechanisms exist in sugar pine, but their genetic control is not known at this time (Kinloch and Davis 1996). The specificity of gene-for-gene interactions cannot be overemphasized. R in sugar pine is only vulnerable to basidiospore genotypes in the rust that have this mutant allele—vR; it is resistant to all other "wild types" in the rust population. Conversely, vR is only specifically virulent to R; it does not affect other resistant mechanisms in sugar pine any more than wild type does. Sugar pines with no resistance mechanisms are attacked by virulent and wild type races alike. The only way to differentiate one rust race from another is by its effect on a particular host resistance gene. Thus, the functional ability of a spore or inoculum source to infect sugar pines with MGR defines the virulent race. By this criterion, the rust attacking MGR at both Happy Camp and Mountain Home are the same race, because they both have vR. They may differ at other gene loci, however.

We can detect vR anytime the breakdown of function in R is perceived after being chal-

lenged, either by natural or artificial inoculum. Natural challenge is effected by strategically locating monitor plantations of appropriate genotypes. Susceptible (rr) sugar pine genotypes will detect whether (and when) challenge has occurred, while infection of known R-genotypes will detect whether vR is present in the inoculum. The precise frequency of vR in any area can then be determined by direct assay of inoculum—basidiospores cast from *Ribes* leaves bearing telia onto young sugar pine seedlings with known resistance. Since basidiospores are haploid, the phenotype of the lesion (needle spot) on seedlings denotes the genotype of the spore that induced it. On seedlings with R, wild type spores cause hypersensitive, necrotic reactions, while spores with vR cause bright yellow spots typical of susceptible interactions. By counting the two types of spots, the frequency of vR in any given inoculum can be determined.

## History of vR

vR was first detected in 1978 in a test plantation near Happy Camp (Kinloch and Comstock 1981). The infections on MGR sugar pines were dated to 1976, a wave year throughout California, in which inoculum levels were very high and blister rust spread into new areas. vR was not found elsewhere in extensive inoculum surveys in Washington, Oregon, and California—or even within a quarter mile of the test site. Monitor plantings on the Klamath, Plumas, and El Dorado National Forests also showed no evidence of vR through 1985 (Kinloch and Dupper 1987). In 1987, infection was observed on a known MGR seed parent on Thompson Ridge, downwind about two miles east and 1000 feet higher in elevation than Happy Camp (Kinloch et al. 1996). Subsequently, a few branch infections were found on two other nearby MGR selections on Thompson Ridge, all within a half mile of each other.

Meanwhile, several additional monitor plantations were established in 1983 in the Klamath Mountains and in the northern and central Sierra Nevada, and at Mountain Home State Demonstration Forest in the southern Sierra. Presently, there are 12 plantations (Fig. 1) that have had at least three challenges of heavy inoculum in different wave years. One of these, Poker Flat, is on a ridge in a line of sight only eight air miles west of Happy Camp. Trees on this site have been severely challenged; on susceptible controls, nearly every internode of every branch has been infected. At Cole Creek, an older plantation even closer to Happy Camp, all susceptible trees have long since died from rust associated mortality, yet MGR trees on both sites still remain free of rust. Nor has rust been found on MGR trees at any other monitor site, until 1996 at Mountain Home. Here, new infections were found on approximately half of the MGR trees in two of three monitor plantations established in 1983-1984, all within a mile of each other (Fig. 2). The great majority of these infections were dated to 1992, with a few from 1989. The third plantation had no infections on MGR trees.

Inoculum also was assayed annually for the presence and frequency of vR at the Happy Camp test site and near vicinity, and in occasional years at other sites, including Mountain Home. vR remained at very high and near constant frequency at Happy Camp, but has so far never been detected at nearby Poker Flat. On Thompson Ridge, the frequency of vR at the three microsites surrounding the infected trees has fluctuated greatly over the years, ranging from near zero to 89% (Fig. 3). Differences among the sites, all within about a half a mile of each other, in any given year also are striking. vR was never detected at Mountain Home before this year, when assays were intensified on different parts of the forest. Presently, it appears to be concentrated near the southern end of the forest at variable frequencies. Among

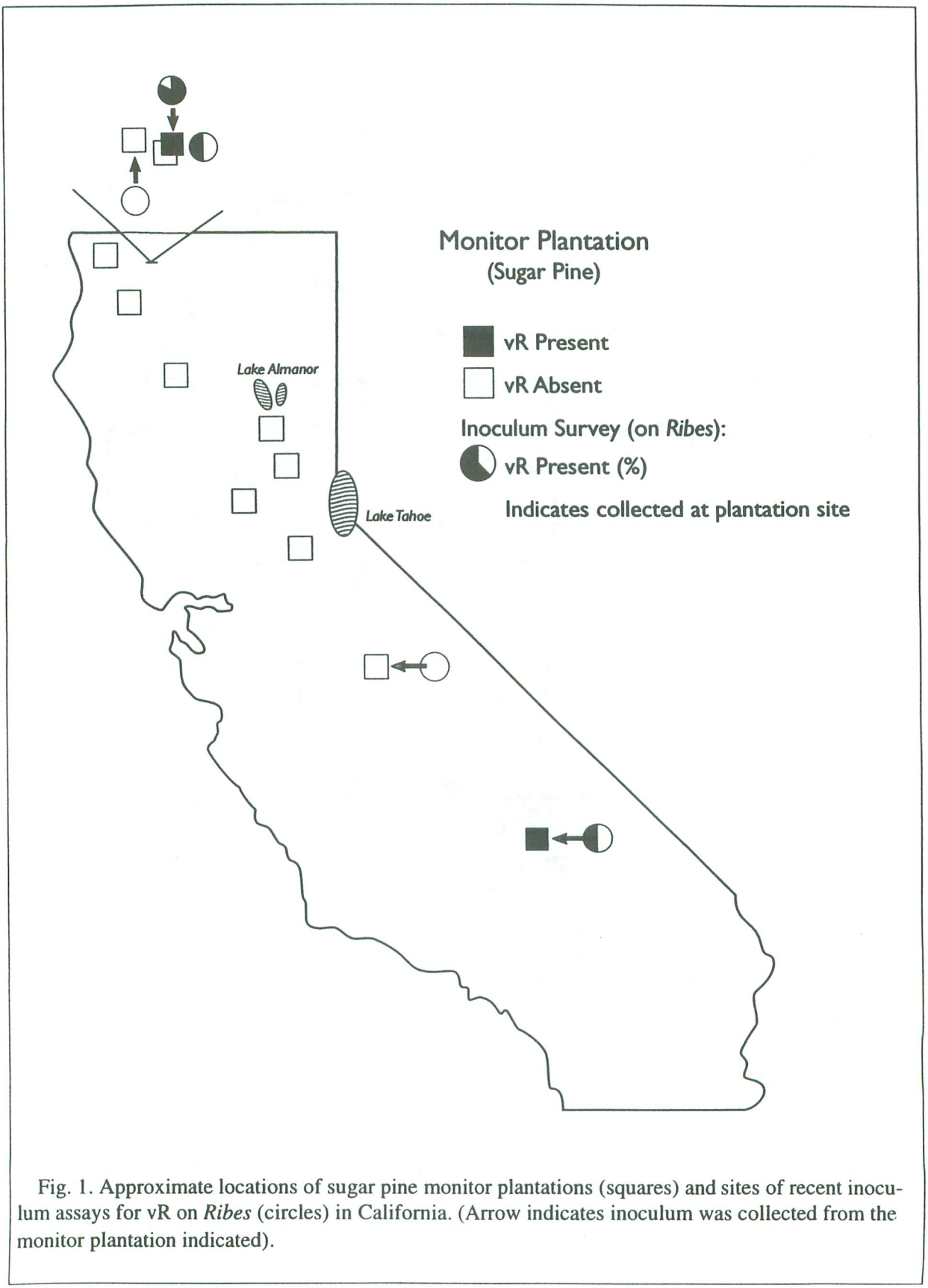


Fig. 1. Approximate locations of sugar pine monitor plantations (squares) and sites of recent inoculum assays for vR on *Ribes* (circles) in California. (Arrow indicates inoculum was collected from the monitor plantation indicated).

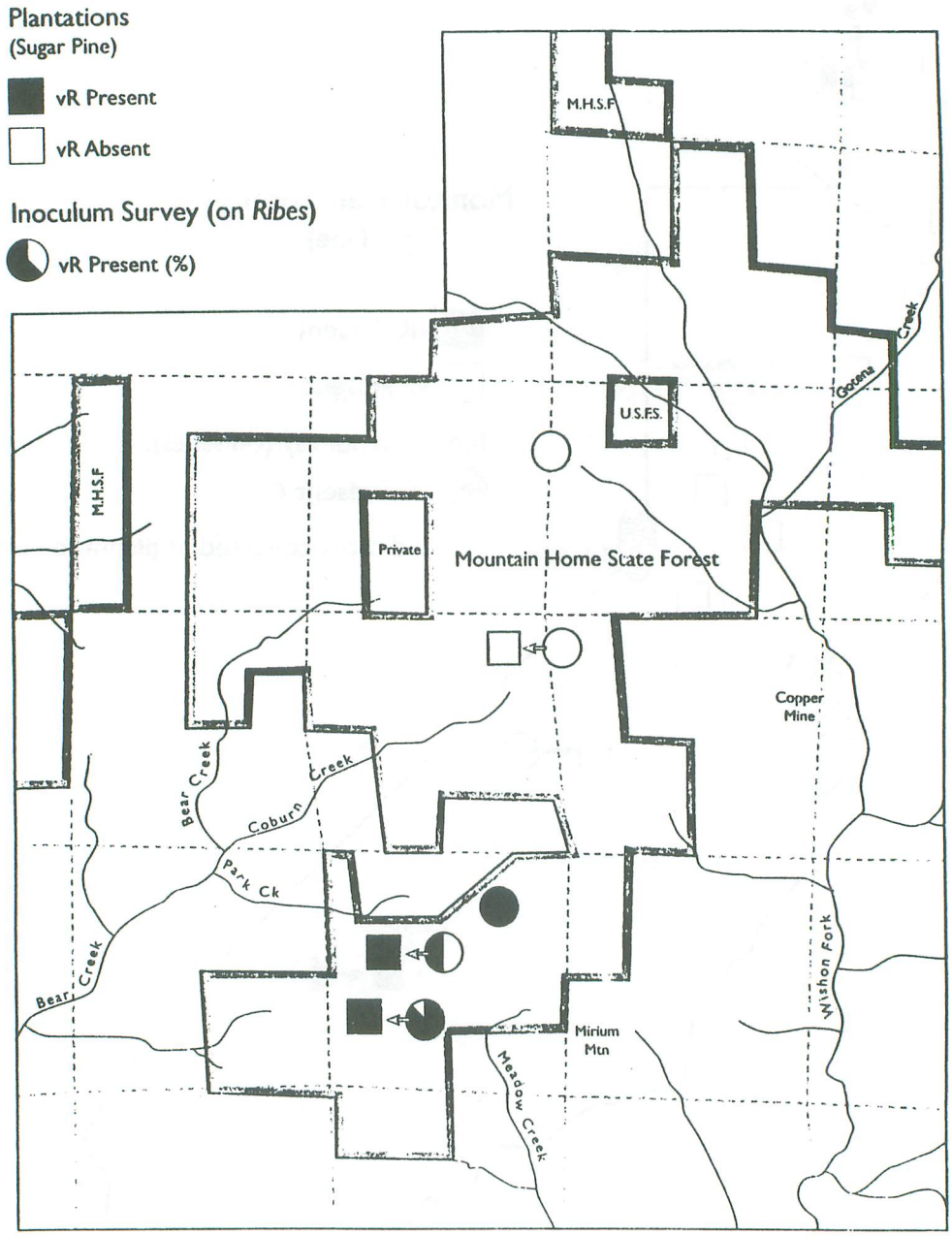


Fig. 2. Locations of sugar pine monitor plantations (squares) and sites of inoculum assays for vR on *Ribes* (circles) on Mountain Home Demonstration State Forest. (Arrow indicates inoculum was collected from the monitor plantation indicated).

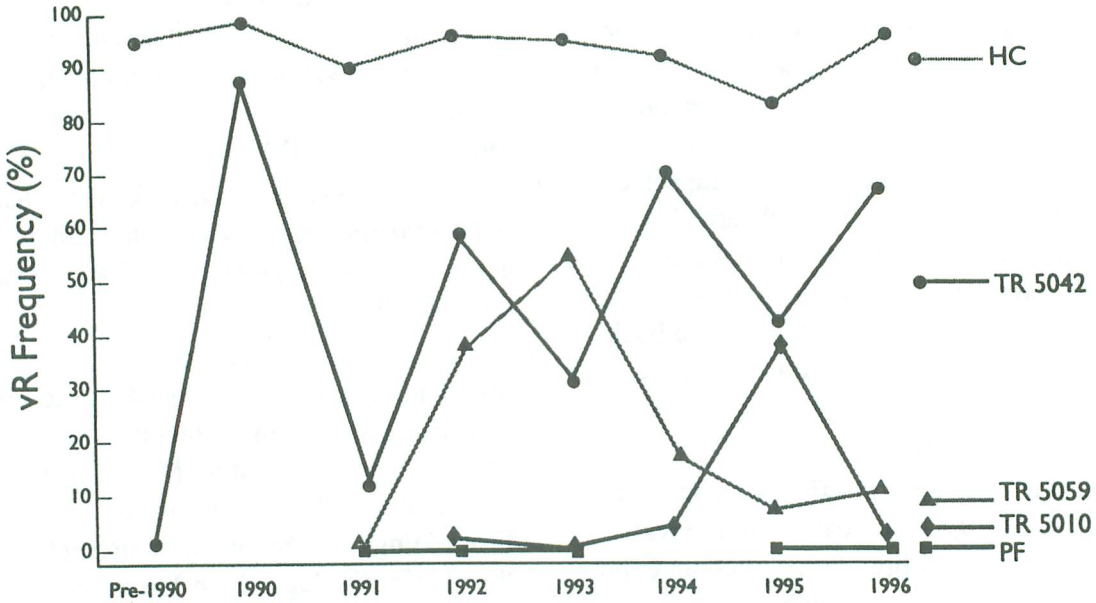


Fig. 3. Frequency of vR in the basidiospore population over years at Happy Camp and nearby sites. HCOS, Happy Camp outplanting site; PF, Poker Flat; TR, Thompson Ridge, near MGR seed parent indicated.

five sites sampled, vR frequency ranged from 0 to almost 100% (Fig. 2).

## Evaluation and Prognosis

As this history shows, it would be risky to attempt to forecast the future behavior of vR. Nevertheless, we can describe past behavior that at least suggests patterns to anticipate.

vR does not spread very fast. Nine years after establishment at Happy Camp in 1976, it was not detected in extensive inocula surveys anywhere else (Kinloch and Dupper 1987). Since that time, it has migrated only about 2 miles east to Thompson Ridge, where it has a very tenuous foothold. Fluctuating frequencies at the three sites in that area over the years (Fig. 2) suggest that extinction of the founder population is a real possibility.

vR does not recur very often. It took 14 years to appear at Happy Camp, where inoculum potential was deliberately and artificially increased in test plantations by interplanting *Ribes* among pines, and about 28 years after rust first established at Mountain Home. Presently, it exists at 2 of 12 monitor plantations (Fig. 1).

Yet, recur it certainly will. The origin of vR at Happy Camp was attributed to mutation (Kinloch and Comstock 1981), and there is no reason to suspect otherwise at Mountain Home; migration in one leap from the Siskiyou Mountains to the southern Sierra Nevada is extremely unlikely. Mutations occur at regular and predictable rates. Although these rates may vary, all are rare, and would seldom be detected in a wild population unless increased a few orders of magnitude by selection. Three factors that seem to be critical in establishment of vR are: high inoculum density, to increase the probability of mutant spores arriving at infection courts; a high concentration of R- genotypes, to selectively increase vR inoculum; and chance. Each of these conditions were fulfilled at both Happy Camp and Mountain Home. Wave years of unusually high inoculum density occurred in 1976, the year vR appeared at

Happy Camp, and 1989, when we think it made initial establishment at Mountain Home. Chance apparently failed at other known monitoring sites in both of these wave years (and others, such as 1983). Although vR infects R- and rr genotypes alike, R- has absolute selectivity: only basidiospores with vR are able to infect. Therefore, all subsequent inoculum produced by the perennial infections on R- genotypes will carry this virulence gene back out to neighboring *Ribes*. At this point, the mutation rate becomes academic, because vR is increasing exponentially.

Long range strategy for effecting control of blister rust through genetically durable resistance is premised on preventing such an exponential increase of vR, or any other race of wider virulence that may arise. Although MGR in sugar pine remains the only effective genetic tool for the short term, its useful life might be extended in areas of relatively low rust hazard by suppressing or eradicating *Ribes* inside and surrounding high value plantations containing MGR trees, thus reducing inoculum loads and selection pressure on MGR. However, this may not be effective in stands where rust hazard is chronically high and/or vR has already established in naturally occurring MGR trees, as is the case at Mountain Home. Eventually, we anticipate reinforcing MGR with other known resistance mechanisms in every tree planted (Kinloch and Davis 1996; Samman and Kitzmiller 1996). Because these mechanisms are not specifically vulnerable to vR, they will have the same result of reducing the effective inoculum load challenging MGR.

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