PROGRESS IN BREEDING BLISTER RUST RESISTANT WESTERN WHITE PINE

by

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Nowadays, we foresters can expect increasingly frequent "mandates" from the public concerning the preservation of wildlands we manage. We know that this trend of increasing public awareness and involvement is healthy--even though such active interest can temporarily upset established practices or delay ongoing programs.

In our attempt to prevent further deterioration of sensitive forest ecosystems (especially by pesticide accumulation), we foresters are placing renewed emphasis on biological, rather than chemical, control of forest pests. This paper explains the mechanics of one of these biological control methods, improvement of genetic resistance in western white pine to blister rust disease.

Blister rust disease in an incubation (hidden) stage on white pine nursery stock was introduced into western North America in 1910. By 1923, it had spread from Vancouver, B. C., into northern Idaho, and by 1941, the blister rust epidemic had spread to most of the Inland Empire's 3-million acres of western white pine.

Conventional rust control measures, e.g., mechanical and chemical eradication of alternate host plants (Ribes spp.) and other chemical (antibiotic) controls applied to the primary pine host, proved to be inadequate. Consequently, aside from research, attempts to control the disease in this region were abandoned in 1967.

We are now engaged in a massive salvage operation, seeking to harvest severely damaged, salable trees before they are lost. At present, our best hope of restoring western white pine to Inland Empire lands lies in development of genetic resistance, a slow but sure means.

Recognizing the high intrinsic and aesthetic values of western white pine, the Forest Service has become the leader and primary investor in research and development of blister rust resistant western white

pine. We have refused to abandon a highly valuable and tractable species or to add it to what is becoming a dangerously long list of pest-threatened species. Implied in the genetic control we seek is the stabilizing influence of resistant types on sensitive forest environments, and the avoidance of environmental pollution.

The clear history of the rust's introduction from Europe and the severity of resulting epidemics in western white pine led to the assumption that this pine was undergoing its first exposure to blister rust. Host populations appeared to be uniformly and highly susceptible. Soon, however,--unlike American chestnut trees, which appear to be consistently susceptible to chestnut blight--rare white pines were found to be disease-free, despite long exposure in heavily rusted stands. And, when such rust-free selections were mated, some really remarkable gross levels of resistance were noted in the first generation seedling progenies.

Resistance reactions were found to occur at a succession of sites in needles and bark, as follows:

1. In the needles, resistance reaction(s):

   a. Limited penetration or establishment of the fungus and resulted in either a decrease or an absence of needle lesions; or

   b. Slowed or prevented extension of the rust mycelium down the needles and into the bark, which resulted in fewer bark cankers per given number of needle lesions.

2. In the bark, where the same or other resistance reaction(s) resulted in:

   a. The bark rapidly becoming necrotic in the immediate area of very young bark cankers at needle-bundle bases; or

   b. Lengthier "corking-out" syndromes involving well-established bark cankers.

From this series of reactions, we hypothesized that four or more resistance genes might be involved in the survival of test seedlings.

The sun total effect of these resistance reactions was a range of from 0 to 50 per cent survival of F1, or first-generation, progenies. These survival levels seemed to follow normal distribution. This and the evidence on sites of reactions led to suggestions that (1) resistance may be controlled by several to many genes, and (2) survival level might be inherited quantitatively, as reported for many cereal rusts. Quantitative genetical analyses were undertaken.

Narrow-sense heritability (h_2) analyses of progeny survival percentages indicated that a high proportion (60 per cent) of the phenotypic variance, or total genetic and environmental variance in the system, was of the additive-genetic type. About one in four of the parents exhibited general combining ability for transmission of blister rust resistance (i.e., a number of F_1 test crosses were consistently high in resistance). When both parents exhibited a general combining ability, their F_1 crosses averaged 30 + per cent survival. Subsequent genetic gain analyses then showed that by breeding a second generation from the survivors of such crosses the survival level might reach 50 + per cent. With about one in four of some 400 selected, rust-free parents exhibiting general combining ability, a genetic base of 100 + trees was available for practical breeding work.

Survival level, the gross effect of all resistance reactions, was considered a useful interim concept. However, studies were obviously needed to define individual resistance genes, pathogenic races (if any) of the fungus, and interactions of these that produce visible resistance reactions. Such research was planned, hut not manned, until the late 1960's.

In our work, we observed levels of survival in F1 progenies and predicted levels of survival in the F2 generations were accepted as adequate to permit the use of F_2 stocks for reforestation of select white pine lands. In 1957, we began a "first-stage" developmental work program, designed to produce about 10-million F_9 seeds and semi-resistant seedlings per year from 40 acres of F1 -seedling orchards. This program is underway and will have continued for 28 years before initial production of F2 seed in 1985. It is estimated that by that time, the program will have cost about $1-million. In the 20 years following 1985, 600,000 acres of the best white pine lands will be restored to that species.

In undertaking this program, the Forest Service accepted two known risks. We realized that:

1. The resistance expressed by parental trees long exposed to the rust might be unrelated to that observed in their juvenile test progenies in the rust nursery, and vice versa.

2. That effects of most resistance genes would be short-lived, because they might have been or soon might be bypassed by various pathogenic races of the rust. fungus.

The wheat breeder has been fighting leaf **and** other rusts of wheat for 50 years. It seems that no sooner doss he produce an immune variety than the rust makes an end run and genetically altered races of the rust negate his hard-fought gain. This see-saw battle is economically sound in annual crops, but it is obvious that pines cannot be bred rapidly or economically enough to solve the rust race problem this way.

Agronomists are finding, however, that certain forms of genetic resistance (called uniform or horizontal) defy breakdown by genetically labile disease fungi. Corn breeders have been especially successful in accumulating and using uniform resistance as a basis for lasting resistance. In fact, the United States had never had a major, cataclysmic epidemic of corn leaf rust. This is what we want to accomplish in blister rust resistance research and development work.

Over the past 2 years, results of anatomical and genetical analyses based on resistance reactions have disclosed the existence of five discrete blister rust resistance genes in watery_ white pine, and at least two pathogenic races of the blister rust in western white pine foliage, through which the rust enters the pine. We expect equally important findings from analyses of disease reactions that occur after the rust enters the bark. Surprisingly, some of the resistance reactions we have noted in western white pine foliage and bark are also characteristic of reactions known to occur in Himalayan or Armand pines, two white pines that, in effect, have grown up with the rust and are little affected by its attack. It may be that certain single resistance genes identified in western white pine are much more stable than the single immunity-imparting genes that led to the downfall of the wheat breeder.

Only more research will provide answers to these questions. When we have the answers, we will be ready to take another large step, this time toward permanent resistance. We are now planning research in preparation for second-stage improvement of western white pine. We feel that with new knowledge from the agronomists and from the Cronartium resistance breeders, the development of new western white pine varieties with higher and more stable resistance to blister rust, plus improved growth, is only a matter of time.