Blister Rust in North America: What We Have <u>Not</u> Learned in the Past 100 Years

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ABSTRACT

Introduction of *Cronartium ribicola* (white pine blister rust) greatly motivated development of tree disease control and research in America. Although foresters and pathologists have learned much in the past 100 years, more remains to learn. The most important lesson is that fear of blister rust has reduced pine regeneration more than the disease itself. Based on six decades of study, I share what I've learned on five topics of personal interest—the evolution of pines and rust, history of blister rust, effectiveness of eradication, influences of climate and Ribes, and importance of sustaining research.

The rust fungi first arose on primitive plants and later evolved to alternate between angiosperms and gymnosperms. Early stems rusts were widely distributed on *Pityostrobus* before the modern pine subgenera of *Strobus* and *Pinus* emerged during the Triassic and Jurassic Periods. In the Cretaceous Period, blister rust fungi of the genus *Cronartium* became widespread on Laurasian pines. During the warm Paleocene and Eocene Epochs, pines retreated to cold refugia as angiosperm forests expanded. In North America, pine refugia were too cold for infection of Ribes; so, unchallenged by the rust, surviving pines lost their resistance. In Eurasia, a variety of environments allowed hosts and pathogens to coevolve. Pleistocene glaciations removed white pines from most of Europe; the pathogen and resistant white pines survived in Asia.

Pinus strobus was introduced from North America into Europe in 1553 but not widely planted until the 1700s. Before that, white pine blister rust was restricted to Asian white pines associated with *Ribes nigrum*.

When European foresters and gardeners brought together highly susceptible *P. strobus* and *R. nigrum*, they set off a super-epidemic. Between 1907 and 1909, millions of white pines from European nurseries were imported to eastern North America. Infected seedlings went undetected, and another epidemic took off. Quarantine, inspection, and eradication of infected white pine failed. Control shifted to eradicating Ribes, especially *R. nigrum*.

Mortality early in the North American epidemic reduced white pine stocks by alarming rates. In the Northeast, blister rust was successfully controlled primarily by eradicating *R. nigrum* in well supported state programs. The long-term effectiveness of Ribes eradication in Maine was tested by comparing the percent incidence of infected trees in areas never treated with areas treated repeatedly over 70 years. Eradication produced a reduction in blister rust from 9.1 percent incidence without treatment to 3.8 percent with treatment. Since 91 percent of unprotected trees were not infected, did the amount of rust after the first wave and elimination of *R. nigrum* justify a continued program?

Differences in rust distribution across the Lake States suggested that climatic factors were important. Data for temperature and moisture requirements of rust development enabled me to draw regional hazard zones. In the lowest zone, infection was unlikely except in locally cool-moist sites. In the highest zone, locally cold climates in forest openings prevented Ribes infection but pine infection could occur in sites even with no Ribes because of long-distance dispersal.

On the cold, dry Yellowstone Plateau, Ribes were seldom infected; the few pine infections which did occur resulted from Ribes at lower elevations. A variety of conditions were found in the Sacramento Mountains of southern New Mexico. There are high-elevation sites too cold for Ribes infection, warm-dry low-elevation sites where susceptible hosts are sparse and blister rust is rare, and mid-elevation sites "just-right" for lots of rust.

Control and research on tree diseases requires an understanding of local conditions and a crucial, long-term commitment. Eastern ideas on Ribes control, spore dispersal, and climate factors had to be adjusted for the West. Unjustified embracement of antibiotics, followed by disappointment, lead to abandonment of the therapy strategy. Loss of white pine's commercial value disrupted research still needed to know where to grow white pine for wildlife and diversity.

INTRODUCTION

Foresters, pathologists, and administrators have learned a great deal in hundred years of infestation by white pine blister rust in North America. Introduction of *Cronartium ribicola* J.C. Fisch. in Rabh., the pathogen of white pine blister rust, was a great motivator for development of tree disease control and research. But, there remain many things we have not yet learned or have repeatedly failed to apply. The most important lesson is that unjustified fear of blister rust has reduced pine regeneration more than damage from disease itself. White pines given a chance can produce enough seedlings to overcome destruction by blister rust. Blister rust is wiping out white pines on some sites, but there are also places where white pines are thriving. We need to learn from a better understanding of the rust where we can grow white pines.

A principal interest of my professional and volunteer work over six decades has been how climate and Ribes affect rust dispersal and infection. Other topics of personal and special interest have been the evolution of pines and rust, history of blister rust, effectiveness of Ribes eradication, and the importance of a commitment to research and white pine silviculture.

Origins of the Rust
The first rust fungi probably arose on mosses, ferns, and other primitive vascular plants during the warm-moist carboniferous periods of the Paleozoic Era (Millar and Kinloch 1991). The early rusts were autoecious fungi with simple spores. The modern pine subgenera *Strobus* (white pines) and *Pinus* (yellow pines) diverged from Pityostrobus in the Triassic and Jurassic Periods (early Mesozoic) and diversified during the Cretaceous Period. Early Cronartium rusts evolved with pines during the middle Mesozoic Era after Laurasia and Gowanda separated from

Pangaea. *Cronartium ribicola* adopted a heteroecious lifecycle of alternating between Strobus pines as aecial hosts and first Ribes and later Pedicularis and Castilleja as telial hosts.

In the early Cretaceous, pines and Ribes were distributed across Laurasia. Blister rust spread in waves every few years on a species of Ribes ancestral to *R. nigrum* and *R. hudsonianum*. *Cronartium ribicola* became a Ribes rust that overwintered on pines tolerant or partially resistant to infection by frequent exposure. As Laurasia separated into North America and Eurasia, different populations of pines, Ribes, other telial hosts, and rusts coevolved on each continent

During the Eocene and Paleocene Epochs of the Tertiary Period, the earth was intermittently hot or cool (mostly hot) for 27 million years. Angiosperm boreotropical plants flourished up to 70° north; pines retreated to cool refugia at high elevations or latitudes (Basinger et al. 1994; Baez 2006). In North America, the white pines *P. monticola* and *P. lambertiana* crowded into the 5500 m Rocky Mountains; *P. ayacahuite*, *P. strobiformis*, and *P. flexilis* into the 5500 m Sierra Madre Oriental; and *P. strobus* along the Arctic shore (Millar 1993). Because these areas were too cold for *C. ribicola* to infect Ribes, surviving white pines were not exposed to infection and lost their resistance. In East Asia, a major refuge was along the Tethys Sea. Mountain ranges formed diverse environments as India pushed under Asia. Because both *R. nigrum* and *C. ribicola* persisted, selection for resistance remained in the Asian white pines (*P. koraiensis*, *P. pumila*, *P. sibirica*, and *P. wallichiana*).

The Pleistocene Epoch encompassed a series of glacial and interglacial periods. *Cronartium ribicola* resided on *P. pumila*, *P. sibirica*, and *R. nigrum* and tracked across Eurasia during the ice ages or persisted in unglaciated Siberia. Because of continuous association, Eurasian white pines retained rust resistance. In the Eem Interglacial, a pine forest extended across Eurasia and included pine infected by Cronartium (Flint 1971; Mirov 1967). This forest was largely wiped out during the Weichsell Glaciation when most of Europe was tundra. At the end of the Pleistocene, the only pines in Europe were *P. sylvestris* (a yellow pine, non-host for *C. ribicola*) and white pine species restricted to high-elevation sites— *P. cembra* in the Alps and Carpathian Mountains and *P. peuce* in the mountains of the Balkan Peninsula (Holzer 1972).

Introduction of the Rust

Pinus strobus was introduced into Europe in 1553 but not widely planted in gardens until 1705 or forest plantations until 1750 (Spaulding 1929; Moir 1924). Although resistant *P. wallichiana* from Asia were planted, the only white pine throughout most of Europe was the susceptible *P. strobus* from North America (Holzer 1972). Long before 1750, *R. nigrum* was widespread across northern and mountainous Eurasia as a wild native (Spaulding 1929; Kakishima et al. 1995) and nearly ubiquitous in gardens as the European black current (Spaulding 1929). Tubeuf (1917) and Spaulding (1929) believed the *C. ribicola* which caused the European super-epidemic had originated in northern Asia. That rust first became widespread in Russia after *P. strobus* was introduced to gardens there and then spread to Europe in several waves on *R. nigrum*. Blister rust was discovered on Ribes in 1830 in Austria, 1846 in Crimea and 1854 in Estonia (Unger 1836, Spaulding 1929; Peterson1973). The close association of susceptible *P. strobus* and *R. nigrum* in European gardens and nurseries allowed development of an epidemic Tubeuf (1927) suggested was more widespread across Europe than shown by available data.

White pine reforestation accelerated in the eastern States from 1900 to 1910. Initially, American nursery stock of *P. strobus* was too expensive and European stock was burdened with a high tariff. After the tariff was removed, millions of small trees—many infected with *C. ribicola* —were brought from Europe (principally Germany). From 1907 to 1909, these trees were established in plantations widely scattered across the northeastern States, Lake States and eastern Canada (Boyce 1961). Early introductions were at Kittery Point, Maine in 1897 on *R. nigrum* from Nottingham, England (Posey and Ford 1924) and Geneva, New York about 1900 on *P. strobus* (Stewart 1906). Separate and serial introductions in New England, Ontario, and Quebec resulted in widespread establishment of the rust on both white pines and Ribes (Detwiler 1918a, 1918b, 1920). Spaulding (1922) mapped changes in rust distribution 1909 to 1919.

Quarantine, inspection, and eradication of infected white pines proved ineffective. Tubeuf (1897) warned infected nursery stock was distributed throughout Germany. Although the J. Heinz nursery maintained that its stock was disease-free, Tubeuf (as cited in Spaulding 1929) responded that disease absence could not be assured because symptoms were invisible for years after infection. Spaulding (1909) inspected trees from the J. Heinz Nursery and planted in New York. He confirmed symptoms could escape detection for the first three years and concluded nursery inspection could not detect every infected tree (Spaulding 1913). Infected Ribes and pine at the Geneva, New York introduction site were destroyed in 1906, but two 15-year-old pines sporulating in 1913 were found too late to prevent permanent establishment (Spaulding 1914).

Blister rust intensified and spread from northern and eastern nurseries (including Ontario) but did not spread from southern and western nurseries (Indiana, Illinois, and southern Pennsylvania). This difference was first attributed to eradication but later recognized as a result of a warm-dry climate unsuited for rust dissemination. I reported (Van Arsdel 1954) that rust did not spread where the mean July temperature was > 21°C. In addition to a warm climate, spread was barred by distance from wild Ribes (e.g., Gibson County, Indiana, 120 km, Van Arsdel 1949). Although infected trees shipped in 1908 from the J. Heinz nursery to an Illinois nursery failed to spread the rust in Illinois, re-movement of trees to Wisconsin, Minnesota, and western Ontario established infestations (Haddow 1969; Kroeber 1948; Pickler and Pierce 1919; Sampson 1918). Early efforts were made to control blister rust by destroying symptomatic pines and removing all hosts within an immediate introduction area, but the rust kept spreading. By 1919, the control strategy was changed to eradicating all Ribes within an infecting distance of pine stands (Kroeber 1948).

Mielke (1943) asserted the introduction of *C. ribicola* into western North America was the result of importation in 1910 of infected *P. strobus* from Ussy, France to Point Grey, Vancouver, British Columbia. Hunt (2003) and Geils et al. (2010) questioned whether this was the first and only western introduction. Other importations of *P. strobus* at the time into Cascade nurseries were from nurseries in Illinois and Ohio that were too warm for rust survival, so these imported trees could not have been infected. The early infestation on Mt. Hood, Oregon can be attributed to long-distance dispersal of aeciospores from infested sites to the northwest.

Early Loss and Control

Soon after establishment in the East, there was a great deal of blister rust on both Ribes and pines; and it was spreading rapidly (Detwiler 1918b). Reports documented destruction of 90 percent of the best trees (Snell 1931) and loss of nearly 50 percent of the crop volume (Rusden 1952). Such losses lead to near panic. *Ribes nigrum* was banned and eradicated. Patriotic appeals were made, "The war against blister rust was just as important as the war against the Germans" (Detwiler 1918a).

In New England, early control was successful because *R. nigrum* was practically the only inoculum source; red currants (*R. rubrum*) and wild Ribes were not important (Snell 1941). Ostrofsky et al. (1988) evaluated the effectiveness of Ribes eradication in Maine by comparing disease incidence in areas never treated with areas where Ribes were eradicated for 70 years. On treated areas, 3.8 percent of trees were infected; on areas never treated, 9.1 percent were infected. These results showed an average, statewide reduction in rust coincident with eradication. But, did the small incidence in untreated areas—91 percent were not infected—justify eradiation?

Success of the eradication program in the Northeast whereby *R. nigrum* was eliminated in the proximity of most pine stands means that blister rust is no longer a major disease there. Now, claims are made that blister rust never was a problem and *R. nigrum* can be grown again. Blister rust was a problem and would be again if *R. nigrum* were re-introduced. Two questions not resolved from work in the Northeast are: 1) the role of wild Ribes (including their susceptibility and distance to pine) and 2) how climate affects where and when blister rust would be a threat.

Blister rust control laws and programs were also established in the Lake States of Wisconsin, Minnesota, and Michigan. Under direction of Dr. E. E. Honey, a series of plots were established to determine the effects of Ribes eradication on pine infection, relative susceptibility of *Ribes* species, distance of rust spread, and Ribes reproduction. Information on these investigations was contained in unpublished reports by Honey and others from 1933 to 1947. I reviewed this work in Van Arsdel (1972) and briefly summarize various observations below.

On the Lake States plots, rust infection before eradication in 29 stands ranged from 0 to 118 cankers per 100 trees per year. Since Ribes were abundant in all these stands, this variation is attributed to environmental differences. After eradication, infection on all plots was reduced to 5 to 20 cankers per 100 trees per year. Had Ribes not been removed, only several plots would have been severely damaged and most plots only slightly damaged (Van Arsdel 1968). The problem was to know beforehand which would benefit from treatment.

One environmental difference among the study plots was the mixture and behavior of Ribes species. *Ribes hirtellum* was susceptible enough to build an epidemic by itself. Although *R. cynosbati* was very susceptible, it defoliated before rust spread to pines; *R. americanum* was slightly susceptible. Where these latter two species were alone, few pines were infected. Where both species were present, infection went from pine to *R. cynosbati* to *R. americanum* to pine. Even with both Ribes present, 89 percent of trees on one plot remained infection-free. On another plot where Ribes were not removed, abundance, susceptibility, and per-leaf inoculum potential differed independently among the five species present and 70 percent of trees remained not infected.

Boyce et al. (1934) said that without control, *P. strobus* could not be perpetuated except where Ribes were few. Certainly, the loss of seedlings and damage to plantations had been great. But infection rates appeared to decline after the first waves—perhaps because there was less inoculum after blister rust defoliated the most susceptible Ribes. Where there is abundant natural reproduction, the regenerative power of white pines can mitigate the damage caused by the rust.

Rust Hazard in the Lake States

My University of Wisconsin and Forest Service Research showed that frequent blister rust infection on pine required a favorable climate at either a regional level or in a locally cooled microclimate. Where the general climate was too warm for pine infection, the rust was limited to sites cooler and wetter than average. These included sites cooled by nocturnal, down-slope winds such as at the base of a slope or in a narrow valley and small forest openings cooled by net radiation heat loss. Using this information (and a wide pen), I mapped four rust hazard zones in the Lake States (Van Arsdel 1961, 1972; Van Arsdel et al. 2006). These are climatic hazard zones. Hazard at any particular site is further modified by the distribution and susceptibility of the *Ribes* species present and the landscape pattern of nocturnal winds transporting spores.

Climatic Hazard Zones

Zone 0 is the southern 80 percent of the Midwestern states of Iowa, Illinois, Indiana, and Ohio. With a mean July temperature > 23°C, the area is so warm that rust infection or persistence on white pine is unlikely.

Zone 1 covers the northern 20 percent of the Midwestern states and lower southern portions of the Lake States. The general climate is too warm and dry for blister rust spread except in favorable sites where a combination of factors create a locally cool (and therefore mesic) microclimate. A favorable site would be where nocturnal air drains into a constantly shaded forest opening.

Zone 2 includes elevated regions across the lower half of the Lake States. Only a single microclimatic modification is required to cool a generally warm climate sufficiently for rust infection. A favorable site would be where a row of high trees shades an opening from morning sun long enough to prolong the dew period sufficiently to allow infection.

Zone 3 extends from northwestern Minnesota into a narrow band across central Wisconsin and Michigan. The general climate is cool and moist enough without local modification for pine infection to occur every few years. Cankers are scattered among many pines but are usually restricted to that portion of the crown within 2 m of the ground where dew and fog linger.

Zone 4 stretches across northeastern Minnesota, northern Wisconsin and Michigan. The general climate is so cool and wet that infection is possible on all sites and can extend to the tops of trees. The widespread distribution of infection and presence of cankers high in the crown indicate long distance spread from infected Ribes.

A Case History in a High Hazard Zone

The experience of Tom Nicholls demonstrates that the outlook expressed by Boyce et al. (1934) was overly pessimistic. Blister rust has not been as serious in the Lake States as was initially feared. Tom manages a tree farm in Fifield, Wisconsin (Price County), a glaciated region now covered in a patchy mosaic of vegetation types. Logging the virgin white pine peaked in 1892 and ended in 1920 when the site was converted to dairy pasture. After dairy farming ended in the 1950s, a few remaining mature white pine seeded the pasture to a thick stand of saplings. The farm is now managed for timber and wildlife.

Typical of hazard zone 4, blister rust is scattered throughout the new stand. Although blister rust had killed many seedlings, enough regeneration remained to require additional thinning. Blister rust killed a few larger trees, but these snags are valuable wildlife habitat. Silviculture and pest control includes thinning, pruning, excising cankers, and periodic harvests. *Ribes cynosbati* is spread by birds along nearby fence rows and woods. Rather than eradicating the Ribes that attract birds, Tom minimizes pine infection by early pruning of the lower crown. Snowshoe hares assist with this pruning. To establish the next crop, Tom knows he will have to minimize damage from deer and white pine weevil. He protects white pines from weevils by shading young trees beneath overstory red pines. Tom has learned that even in northern Wisconsin blister rust is unlikely to wipe out the white pine. With good stewardship, he produces white pine timber and wildlife habitat on a site with a climate favorable for blister rust and susceptible Ribes nearby.

Extending the Rust Hazard Concept to the Interior West

My observations in Wyoming and New Mexico have identified another climatic factor limiting blister rust infection. Because some high-elevation sites are too cold for Ribes infection, pine infections on these sites are dependent on spread by Ribes growing at lower, warmer elevations.

Yellowstone

Yellowstone National Park is a high caldera (2450 m) surrounded by peaks ranging from 3000 to 3450 m. Vegetation includes *P. albicaulis* (whitebark pine, alpine-subalpine), *P. flexilis* (limber pine, subalpine and montane), and numerous *Ribes* species with their own distinct habitats (alpine, riparian, or forest edge). Because blister rust was considered a serious threat, early trials of Ribes eradication were established in the Park. Removal of Ribes on control plots, however, failed to reduce pine infection significantly from that on non-treated plots (Berg et al. 1975).

In an unpublished service report, Hendrickson (1970) discussed climatic escape to explain the low rate of pine infection and ineffectiveness of Ribes control. He reported temperature and humidity from weather stations representing Park elevations from a low at Mammoth (1950 m) to a high at Eagle Peak Summit (3450 m). My examination of these data confirmed that temperatures at night and during extended wet

periods were well below that required for aeciospore or urediniospore germination and therefore Ribes infection (Van Arsdel et al. 1956). For the most part, pine infections were not the result of spores from nearby Ribes (regardless of abundance and proximity) because it was too cold for them to be infected. Those few pine infections were from Ribes growing at much lower elevations located many kilometers distant.

New Mexico

The idea that temperatures can be too cold for Ribes infection is demonstrated again in the Sacramento Mountains of New Mexico (Van Arsdel et al. 1998). *Pinus strobiformis* is common in montane, mid-elevation forests on a broad dissected plateau above extensive woodlands and below a preeminent volcanic peak (Sierra Blanco). The distribution of *R. pinetorum*, the most important telial host of *C. ribicola* in the region, is only a little smaller than that of the white pine. *Ribes pinetorum* abundance varies from site to site depending on disturbance history and canopy opening; it is rarely infected above 2750 m (too cold). White pines are rarely infected above 3000 m (as result of long-distance spread) or below 2450 m (where susceptible hosts are scarce). Between these limits, blister rust is common wherever susceptible hosts occur together.

Commitment

Geils et al. (2010) presented a brief history of blister rust control and research in North America. Rather than repeat that story or expand with more details, I will only relate several important lessons to researchers, administrators, and foresters.

Ribes eradication methods and expectations were first developed in the Northeast. They did not translate well to the very different conditions in the West. There were more Ribes, bigger Ribes, more species of Ribes. Many of the species were more susceptible than most of the Ribes in the East. The climate was much more favorable for spread of the rust. Access to forest Ribes was extremely difficult; in these remote areas, eradication camps were established at high cost. One foreman observed, "We miss more Ribes per acre than are pulled by the crews of the East."

In spite of the difficulties, Western programs greatly reduced Ribes populations and lowered rust incidence to a very few trees within control zones. Stillinger (1944) thought the rust on the remaining infected trees was from Ribes outside the control zone. Swanson

and Walters (1953) thought these infections were from missed Ribes inside the control zone. Program administrators in Washington DC (Detwiler, Martin, and others) were sure that spread was limited to 300 m as it appeared to be in the East. They dismissed observations from British Columbia that spread could reach kilometers and that a single Ribes could produce much infection (Pennington 1925; Buchanan and Kimmey 1938). Both Stillinger and Swanson had a good understanding of rust epidemiology—Stillinger cited dilution formulae and Swanson recognized the importance of microclimate. My early work on microclimatic influences on rust spread in Wisconsin provided little insight to the extremely complex and highly favorable conditions in the Northwest. A very wet site, low diurnal temperature range, and sea breeze backflow allowed more spread and infection on some sites than considered at the time to be possible.

My work in Wisconsin and later studies in the West have helped to clarify matters of spread and climatic hazard. Spores dispersed from Ribes to pines are transported by diffusion and by air currents (Van Arsdel 1958, 1960, 1965, 1967; Van Arsdel et al. 2006). Ideas of sub-continental spread of aeciospores (Van Arsdel et al. 1998) were fostered by work on peanut rust (Van Arsdel 1973) and applied by Frank et al. (2008). The concept of blister rust hazard zones I developed for Wisconsin (Van Arsdel 1954) has been adopted for numerous regions by many other researchers. It has been revised to fit landscapes in Nevada (Van Arsdel and Krebill 1995) and New Mexico (Geils et al. 1999). Reports by others finding no relation between Ribes and pine infection are clearly wrong; they fail to understand the infection process. More work is still needed on many details of aerial dispersal, temperature control of Ribes infection, and the ultimate question of which Ribes bushes infect which white pine trees.

Chemical control of blister rust included not only use of herbicides to kill Ribes but also attempts to find an antibiotic that would act as a selective, systemic fungicide, killing the pathogen without damaging the pine host. Virgil Moss tested numerous candidates, including Actidione (cycloheximide produced by Upjohn). Although Actidione was not successful on cone rust, results of direct application to basal cankers were promising. But spraying each canker was little improvement over excising it, so a chemical cure remained more a dream than a practical reality.

In 1958, Phytoactin showed promise as a foliar spray delivered from aircraft. Encouraged by limited, short-term, field observations, the Forest Service quickly adopted Phytoactin as an operational tool (Benedict 1981) despite my urging, "Wait, let's test it first." I had noted in a greenhouse test that cankers treated with an antibiotic could resume growth and phytotoxic effects to pine could be severe (Van Arsdel 1962). Research field trials were started, but other testing reported systemic transport of the antibiotic throughout the tree, persistence for two years, and satisfactory performance (Moss 1961). Victory over blister rust was acclaimed. Then, research results from numerous regions and studies came in (Phelps and Weber 1968). Antibiotics had simply masked disease symptoms or temporally reduced spore production. The fuel oil carrier had an equal effect; treatment was not very practical since 87 percent of trees still died. What had gone wrong? The great desire for a chemical control and wishful thinking lead to large tests with more trees than could be carefully observed for a sufficient period. Alternative factors such as secondary fungi and natural inactivation were not considered (Benedict 1981). Although research disproved management's great hope, a good and solid background of information was developed. Rather than continue this work, however, the Forest Service decided to give up on blister rust control and research, and even on planting white pine (Ketcham et al. 1968).

Tom Nicholls showed that white pine in Wisconsin were able to produce abundant regeneration. With escape, old-age resistance, and silviculture, white pines can be grown there. Geneticists have found some white pines in western North America still carry resistance genes; breeders have produced lots of planting stock with these genes. We should have learned from Ribes eradication that disease results from an interaction of pathogen, host, and environment. We should have learned from antibiotic programs the importance of research before deployment. Nursery stock will be expensive; the fitness of resistance and other traits will vary with the environment. Genetic solutions do not relieve us the necessity of understanding epidemiology. I would like to see more research to answer the fundamental question: where can we grow white pines that escape or resist blister rust?

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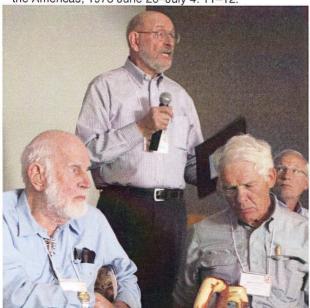
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