Genetic Resistance in Port-Orford-Cedar to the Non-native Root Rot Pathogen *Phytophthora lateralis*—2003 Update

Richard A. Sniezko

**Abstract**—Genetic resistance to *Phytophthora lateralis* in Port-Orford-cedar (POC) provides a management tool for restoration and reforestation in areas heavily impacted by this non-native pathogen. Since it began in 1997, an operational program to develop resistant populations of POC has made rapid progress in some breeding zones. Seed is now available, but additional selections from natural stands are needed for other breeding zones. In greenhouse tests, the best resistant seedling families have 50 to 100 percent survival versus no survival for the most susceptible families. Families also vary in time to mortality in greenhouse testing. In young field validation trials, resistant families are showing good potential, but more time is needed for assessing durability of resistance. A few field selections from 1989 and 1990 confirmed as resistant in greenhouse testing have been revisited and continue to thrive. Investigations into the nature of the resistance mechanisms and their inheritance are underway. Future needs of the program are discussed.

**Introduction**

Since 1952, a non-native, invasive pathogen, *Phytophthora lateralis*, has been spreading throughout the native range of Port-Orford-cedar (*Chamaecyparis lawsoniana*). This root rot kills seedlings as well as large trees, particularly in riparian areas (Casavan and others 2003; USDI-BLM and USDA-FS 2004). Some management tools are available for slowing the spread of *P. lateralis* (Goheen and others 2000; Goheen and others 2003a; USDI-BLM and USDA-FS 2004). Use of genetic resistance may be key in efforts to restore heavily impacted areas.

The first indication in Port-Orford-cedar (POC) of genetic resistance to *P. lateralis* was noted in the 1980s (Hansen and others 1989; Sniezko and others 2003c). Since then, greenhouse and field studies have confirmed genetic variation in susceptibility to *P. lateralis*, the low frequency of resistance in natural POC populations, and some understanding of the inheritance of resistance (Sniezko and Hansen 2003; Sniezko and others 1996; Sniezko and others 2000; Sniezko and others 2003a; Sniezko and others 2003b; Sniezko and others 2003c; Sniezko and others this proceedings). An examination of some of the underlying mechanisms of resistance is also underway (Eun-Sung Oh, personal communication)

This paper briefly summarizes my presentation from the genetic resistance panel at this conference (with some updates). It covers aspects of the operational breeding program, what is known about the resistance, and questions for the future. Further information is available from cited papers and at www.fs.fed.us/r6/dorena.

**Resistance Program Overview**

In 1997, the USDA Forest Service (FS) and USDI Bureau of Land Management (BLM), in cooperation with Oregon State University (OSU), began an operational program to develop resistant populations of POC for use by land managers. Breeding zones have been established based upon common garden tests (Kitzmiller and Sniezko 2000; Kitzmiller and others 2003; Jim Hamlin and Jay Kitzmiller, personal communications) (see figure 1 for breeding blocks). Elevation bands within breeding blocks have been used to define breeding zones.

The first stage of the operational resistance program involved selecting thousands of candidate trees to test for resistance. Most of the earliest selections were made in Oregon in high disease areas. Parents confirmed as resistant are used to establish seed orchards to provide genetically diverse seedling populations with resistance to *P. lateralis*. A major emphasis of the program is to strive to maintain broad genetic diversity in restoration populations; this differs fundamentally from many horticulture and crop breeding endeavors (in which crop uniformity is often paramount). It is unlikely there is sufficient resistant POC in the highest hazard areas to provide large trees for the future without restricting the genetic variation. The resistance program brings together naturally occurring resistant trees from the same breeding zones.

Since 1997, over 10,000 field selections have been tested using the stem dip method (see Bower and others 2000 for summary of the first 7000 selections). The top ten percent of these candidates (approximately) undergo a second phase of testing using rooted cuttings (root dip test). Approximately 500 of the parents have been through this second phase of testing, and about 100 of these appear to be resistant (100 percent survival); an additional 160 some parents show about 50 to 90 percent survival. Further investigation of these parents is underway. Most of the early selections have been in the most northwestern breeding block (BB1 in figure 1).
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Containerized seed orchards have been started for several breeding zones, and the first seed was produced in Fall 2002. Seed has already been sown for restoration and reforestation activities on federal and private lands. Control pollinations are being used to generate full-sib families for evaluation using the root dip test.

The number of resistance mechanisms and their inheritance is under investigation. At this preliminary stage, two groups of resistant families appear evident from greenhouse tests—1) families that show high survival and 2) families that show longer time to mortality (‘slow dying’). In greenhouse testing, some parents show very high survival (50 to 100 percent) as rooted cuttings or as full-sib families versus no survival for many of the most susceptible parents (table 1; and Sniezko and others, this proceedings). In greenhouse testing, time-to-mortality varies by 50 percent or more among families, with a very few families showing much slower mortality. Further confirmation is underway (figure 2, also Sniezko and others, unpublished data). Traits such as ‘high survival’ may have immediate utility in field plantings while more information is needed on the ‘slow dying’ response (and further breeding may be needed).

The underlying nature of resistance is currently under investigation. Preliminary results from a study at OSU showed that 24 hours after root inoculation with zoospores, a susceptible family had more cysts than the resistant family tested (Eun-Sung Oh, personal communication). The hyphal growth in the susceptible family was faster than in the resistant family following stem inoculations (mycelium was used to inoculate, and the stem was examined four weeks later). Fewer hyphae in the resistant family suggested that there may be a defense response in resistant families against P. lateralis (Eun-Sung Oh, personal communication).

Table 1—Percent mortality in seedling families at ten months in the greenhouse root dip trial, results from 2000 test

<table>
<thead>
<tr>
<th>Female Parent</th>
<th>Male Parent</th>
<th>CF1</th>
<th>CF2</th>
<th>510049</th>
<th>510008</th>
<th>118569</th>
<th>117344</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>118573</td>
<td>CF1</td>
<td>58</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>33</td>
</tr>
<tr>
<td>117490</td>
<td>CF2</td>
<td>67</td>
<td>71a</td>
<td>100a</td>
<td>63a</td>
<td>79</td>
<td>92</td>
<td></td>
</tr>
<tr>
<td>510042</td>
<td></td>
<td>50</td>
<td>6a</td>
<td>42</td>
<td>89a</td>
<td>54</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>118562</td>
<td>510042</td>
<td>67</td>
<td>25</td>
<td>42</td>
<td>89a</td>
<td>56</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>510044</td>
<td>510041</td>
<td>42</td>
<td>61</td>
<td>83a</td>
<td>94a</td>
<td>61a</td>
<td>68</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OF</td>
<td>51</td>
<td>36</td>
<td>53</td>
<td>56</td>
<td>94</td>
<td>46</td>
<td>56</td>
</tr>
</tbody>
</table>

* Reciprocal cross

Field tests have been established (primarily since 2000) to examine field resistance and its durability. In general, the survival in field tests correlates well with greenhouse tests (for example, see figure 3; Sniezko and others 2000). There are exceptions and further investigation is needed. The numerous phenotypic field selections made since the program began (over 10,000, many now confirmed as resistant or susceptible) provide an opportunity for monitoring in the field. Several candidate trees (such as 510015, 510005, and 117490—selected in high disease hazard areas prior to 1991 and shown to be resistant in greenhouse testing) have been revisited and found to be alive and healthy (Chuck Frank and Leslie Elliott, personal communication).
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Further investigation of the status of confirmed resistant parents is planned. How different field environments (for example, soil temperature and sustained flooding of low areas) influence effectiveness of resistance is currently unknown.

**Future Needs**

1. Additional selections to increase the genetic base of resistance in some breeding zones. Some breeding zones require evaluation of more than 1000 additional candidates.

2. Completion of second phase (root dip inoculation and assessment) of resistance testing for remaining candidates.

3. Establishment of seed orchards for all breeding zones in which resistant seed is needed. Production of resistant seed with a broad genetic base for each zone.


5. Further evaluation of field plantings. This will be key to discerning whether resistance is durable. Are the resistance mechanisms exhibited in the greenhouse trials equally effective in the field? The resistant material consistently has higher survival than the susceptible material. However, survival of the resistant material is sometimes less than in controlled greenhouse testing. It should be noted that these sites are often visited only annually, and it can be difficult to definitively discern the cause of mortality on young seedlings. Within sites currently classified as high hazard, there may be differences in mortality. Some sites have standing water for sustained periods, and these may be the highest hazard especially to young seedlings. Is foliage on very small seedlings also an infection court, thus bypassing root defense mechanisms?

6. Examination of the susceptibility of very young resistant seedlings in the field. Small resistant seedlings or rooted cuttings may be more susceptible (first few years after planting) than larger resistant trees.

7. Monitoring the durability of resistance. Field trials will eventually give good data on this, but the 1000’s of parent trees selected can also serve a monitoring function. Some of the oldest confirmed resistant trees have recently been revisited and are still alive. In the future, additional selections will be visited.

8. Further summary of the resistance data to refine estimates of the frequency of natural resistance and geographic trends in resistance.

9. More knowledge about *P. lateralis* and its genetic variation and the geographic origin of this pathogen.
Maintaining genetic diversity is a major focus of the resistance breeding program. A proposal is in preparation to investigate the levels of genetic variation in the resistance populations (Kolpak and Sniezko, personal communication).

10. Planting strategies for using resistant Port-Orford-cedar. Do resistant trees ‘harbor’ *P. lateralis*?

**Summary of Program**

Most Port-Orford-cedar trees are very susceptible to *Phytophthora lateralis*, but useful levels of natural resistance to this non-native pathogen exist. POC is highly amenable to a program to quickly evaluate resistance and produce resistant seedlings. Seed production can be accomplished on very young trees in pots (figure 4) (Elliott and Sniezko 2000), and this permits rapid updating of orchards as new selections are made or as breeding increases the level of resistance. Cooperation among FS, BLM, OSU and others coupled with the silvics of POC have led to rapid early progress in developing resistant populations. Continued progress is expected. Monitoring of field validation plantings will provide information on the durability of genetic resistance. Few, if any, operational resistance programs for forest trees have made such rapid progress.

![figure 4](image)

**Figure 4**—Pollination bags on Port-Orford-cedar in a containerized seed orchard.

**Acknowledgments**

Numerous individuals in the USDA Forest Service, USDI BLM, and Oregon State University have contributed immensely to the progress of the resistance program. State, county, private landowners, as well as National Parks have contributed candidates for resistance testing and/or sites for field validation of resistance. Jodie Sharpe, Lee Riley and Scott Kolpak provided key support with summaries, graphs and analyses used. Angelia Kegley and Leslie Elliott provided support in editing and reviewing this document. A special thanks to both Everett Hansen and Don Goheen for their continual inputs and insights.

**References**


Genetic resistance in Port-Orford-Cedar to the non-native root rot pathogen *Phytophthora* *lateralis*
Fusiform rust caused by the fungus Cronartium quercuum f. sp fusiforme is the primary disease of southern pines in the southeastern U.S. Economic losses are estimated at approximately $28M annually. Data collected over the past ~20 years or more suggests that the fusiform rust–southern pine pathosystem largely conforms to a gene-for-gene system (Powers and others 1977; Nelson and others 1993; Wilcox and others 1996; Stelzer and others 1997). In other words, the presence or absence of a stem gall results from the interaction between both host and pathogen. Infection is the character of this interaction. It is not simply a character of just the host or the pathogen. Resistance and pathogenicity, respectively, designate the character of the host and pathogen, although infection (presence or absence of a gall) is a measure of the interaction of these two characters.

Despite this knowledge, efforts to manage this pathosystem have focused almost exclusively on breeding for resistance in the host. The approach taken has been to screen both open-pollinated and full-sib families of the host with a broad source or bulk inoculum (Anderson and Powers 1985). Although this approach has made it possible to identify trees or families potentially harboring large numbers of resistance genes, control of the attacking pathogen's genetic constitution has not been possible and the genetic variability existing within the pathogen population is not known. As a consequence, little information is available regarding the nature of the resistances currently being deployed.

In an attempt to better understand the nature of these resistances, a concerted effort has been made to identify and 'tag' specific resistance genes with DNA markers (Wilcox and others 1996). To date, as many as eight different resistance genes (Fr1 to Fr8) have been mapped (Henry Amerson unpublished data). The associations between resistance loci and marker loci have been observed to be robust and repeatable, and these markers are being confidently used to identify resistant progenies within these defined host pedigrees.

Although resistance genes have been successfully mapped in many host species, few corresponding genes have been mapped in their pathogens. Only relatively recently were avirulence genes in the wheat stem rust fungus Puccinia graminis mapped (Zambino and others 2000). Mapping resistance genes in the host is only half the picture. To effectively manage gene-for-gene pathosystems, it is essential that the corresponding genes in the pathogen (avirulence genes) be identified and tagged with molecular markers. Once markers tightly linked to pathogen avirulence alleles have been identified and their usefulness confirmed in natural populations, that is, linkage disequilibrium of the markers is confirmed, these markers could be used to more effectively manage these pathosystems by directly estimating avirulence allele frequencies in natural populations of the pathogen hence allowing for more informed decisions regarding host resistance gene deployment.

Cooperative research between the USDA Forest Service's Southern Institute of Forest Genetics in Saucier, Mississippi and the Department of Forestry at North Carolina State University is currently underway to map the corresponding avirulence genes in the fusiform rust pathogen. Crosses between single-spore isolates of the fungus are being conducted to produce lines heterozygous for specific avirulence genes. These fungal lines are being used to challenge host families segregating for the corresponding resistance genes. DNA markers within the host are being used to identify the selecting (resistant) and non-selecting (susceptible) progenies. Fungi from infected trees within these two progeny sets (resistant and susceptible) are being screened with DNA markers to identify markers associated with avirulence.
References


Inherent and Induced Resistance to Pitch Canker in *Pinus radiata*

Thomas R. Gordon and Christopher J. Friel

Pitch canker, caused by *Fusarium circinatum*, was originally described as a disease affecting plantation-grown pines in the southeastern U.S., where it remains a chronic problem. Pitch canker was first recognized in California in 1986 (McCain and others 1987). Although many native California pines are susceptible to pitch canker, *Pinus radiata* (Monterey pine) has been by far the most heavily damaged. Individuals within *P. radiata* vary in their susceptibility to this disease, as a result of both inherent genetic and induced resistance.

The first indication that some Monterey pines were resistant to pitch canker was the presence of disease-free trees in areas where pitch canker was well established. Unaffected trees could be differentiated from those showing symptoms of pitch canker by comparing their response to artificial inoculations. Disease-free trees generally showed a slower rate of lesion expansion than those that were visibly susceptible. Through vegetative propagation it was possible to show that relative susceptibility to pitch canker was determined primarily by the host genotype rather than circumstances under which it was growing (Gordon and others 1998). Thus, the absence of infections in heavily infested areas offers a good indication of resistance to pitch canker. However, it is also true that genetically susceptible trees can remain un-infected for many years, notwithstanding close proximity to diseased trees. The reasons for this variable lag period are unknown.

An assessment of the relative susceptibility of individual trees is routinely accomplished through mechanical inoculations and subsequent measures of lesion length at the site of inoculation. Incubation periods can be as short as three weeks in a growth chamber, but may require three months or more under field conditions. This reflects the influence of temperature on growth of the pathogen. A constant moderate temperature (such as 25° C) is near optimal for the pathogen, whereas cooler temperatures result in slower growth. In either case, time course studies show that after a sufficient incubation period, inoculated branches sustaining long lesions will eventually be girdled, and those with short lesions will not. Girdling kills the branch distal to the point of inoculation, which produces the dieback symptoms typical of pitch canker. Thus, the shorter the lesion at the time of measurement, the lower the probability the tree in question will sustain girdling lesions.

The extent of lesion development is influenced by the host genotype, but potentially by other factors as well. Thus, multiple inoculations of a tree (one inoculation on each of several branches) may yield short lesions on some branches, suggestive of resistance, but one or more long lesions as well. The reasons why some inoculations fail to produce long lesions on trees that are genetically susceptible have not been identified, but may include differences in the physiological status of individual branches.

Although it is clear that Monterey pines differ in susceptibility and that these differences remain apparent in vegetatively propagated clones, the heritability of disease resistance has yet to be established. This question has been addressed, in part, by collecting seed from known resistant and susceptible trees and assessing the susceptibilities of their progeny. Such studies show that both resistant and susceptible trees produce seed that gives rise to seedlings with a wide range of susceptibilities. In both cases, the distribution of reaction types is at least superficially similar to what is found among individuals within most native and planted stands of Monterey pine. If further testing substantiates these observations, one would have to conclude that heritability of resistance to pitch canker is low. However, only seeds from open-pollinated cones have been tested. Progeny of controlled crosses may reveal different estimates of heritability.

The extent to which pitch canker develops in a tree is obviously influenced by the genetically determined susceptibility of that individual. However, susceptibility is not a static characteristic and may change over time. Specifically, trees subjected to repeated inoculations over a period of two years supported progressively lower levels of lesion development (Bonello and others 2001). Thus, trees may manifest systemic induced resistance in response to infections by the pitch canker pathogen. This phenomenon has been confirmed under controlled conditions, but may also occur naturally. In particular, the occurrence of systemic induced resistance is suspected where heavily-infected trees go into remission. Disease remission has been observed in a number of monitoring plots (Gordon and...
Inherited and induced resistance to pitch canker in *Pinus radiata* (Gordon and Friel 2003) that were first established in 1992 (Storer and others 2002).

The physiological mechanisms responsible for resistance to pitch canker have not been identified, but may involve effects of resin on the pathogen. Resin is known to have a primary role in defense against insects and pathogens that attack pines. Thus, the ability to tolerate anti-microbial components of resin, such as monoterpenes, may be a perquisite for pathogenesis on pine. Evidence to support this hypothesis comes from experiments showing differential inhibition of nonpathogenic strains, relative to those that are pathogenic on pine, in the presence beta-pinene and limonene (Friel and Gordon, unpublished data). Studies are underway to determine if the resin composition of trees is predictive of their susceptibility to pitch canker.

**References**

Resistance of Pines to Dwarf Mistletoe

Robert F. Scharpf

Introduction

Last year’s WIFDWC in Powell River, British Columbia emphasized the high level of interest and importance we place on research and development of resistance to forest pathogens in western North America. In those few days alone, seven papers or posters were devoted to some aspect of forest disease resistance. Interestingly, however, none of the presentations involved resistance to dwarf mistletoes (Arceuthobium), one of the most widespread and damaging pathogens in the West. Because of this omission, I think it is appropriate at this meeting in Grants Pass to discuss what is known about resistance to this damaging group of pathogens, mainly because southern Oregon and northern California contain one of the world’s greatest concentrations of dwarf mistletoe taxa (Hawksworth and Wiens 1996). More than a dozen species out of about 40 in North America are found in this rather small geographic area. In addition, most of the studies on dwarf mistletoe resistance have been conducted in these states. A summary of most of this work can be found in Hawksworth and Wiens (1996); Scharpf and Roth 1992, Scharpf, Kinloch and Jenkinson 1992 and Shamoun and DeWald 2002. Unfortunately, relatively little research and development is currently under way. I will summarize two or three examples of past work on resistance in pines and briefly discuss some of the ongoing studies.

Expressions of Resistance

I can think of no other forest pathogen–host combination that shows more variation and expression of resistance than the dwarf mistletoes and their hosts. In an effort to simplify some of these relationships, Hawksworth and Wiens developed “susceptibility classes” based on the percentage of trees infected within 6 m of heavily infected hosts (table 1). Many dwarf mistletoe species not only have primary hosts, but also secondary, occasional, and rare hosts, as well. Some dwarf mistletoes infect only a single host species, whereas others can parasitize several genera. For example, A. laricis primarily infects Larix and Tsuga, but also occurs on Abies, Pinus and Picea. Although Hawksworth and Wiens termed the relationship...

Table 1—Classes of host susceptibility to Arceuthobium

<table>
<thead>
<tr>
<th>Class</th>
<th>Host</th>
<th>Infection Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>principal</td>
<td>&gt;90 percent</td>
</tr>
<tr>
<td>II</td>
<td>secondary</td>
<td>90–50 percent</td>
</tr>
<tr>
<td>III</td>
<td>occasional</td>
<td>50–5 percent</td>
</tr>
<tr>
<td>IV</td>
<td>rare</td>
<td>&lt;5 percent</td>
</tr>
<tr>
<td>V</td>
<td>Immune</td>
<td>0</td>
</tr>
</tbody>
</table>

From Hawksworth and Wiens (1996).

Several other host–parasite reactions, I believe, are also expression of resistance. Excessive swelling of host tissue, restricted endophytic growth of the parasite and lack of mistletoe shoot production have been called an “incompatible host–parasite relationship” by Hawksworth and Wiens. I believe these reactions are expressions of resistance. The wide variation in broom development and morphology also suggests a possible resistance response in some hosts. Hypersensitive reactions to the penetrating seeds have been observed in some host–parasite combination indicating a very specific-resistance mechanism. Also, the formation of a suberized layer at the infection site indicates an active resistance response to infection.

Several investigators have made observations of what appears to be obvious resistance in the field. “Susceptible” hosts with no infections have been reported growing within heavily infected stands. In these cases, the only logical explanation for the absence of infection is resistance. Ponderosa pine in Oregon has been reported to express “juvenile susceptibility.” Among the true firs of California, young red firs appear to be more resistant to infection than larger trees.

Field studies in Oregon also provide strong evidence for resistance within individual, grafted selections of ponderosa pine. In one case, it appeared that the endophytic system of the parasite failed to grow across a graft union between an infected rootstock and the scion of a resistant candidate. In another case, a forked tree that developed from a susceptible rootstock; and, after 22 years, a resistant scion showed no infection on the scion but numerous infections on the rootstock.

Although the field observations and past studies have received little additional attention, it appears that several mechanisms of resistance to Arceuthobium may be present. In order to keep this paper reasonably brief, I will discuss only three studies involving resistance of pines to dwarf mistletoe.
Jeffrey Pine, California

The first study discusses a case in which resistance was observed in a plantation of Jeffrey pines at the Institute of Forest Genetics, Placerville, California. (Scharpf and Parmeter 1967). The plantation was established in 1940 to study the growth of pines from different geographic locations in the Sierra Nevada. Randomly spaced, 16-tree blocks constituted the plantation. Unknowingly, the plantation was located adjacent to a stand of ponderosa pine infected with dwarf mistletoe. Fifty years later, many of the Jeffrey pines were heavily infected with dwarf mistletoe but others much less so. Results of a study indicated that the higher elevation seed sources were the most severely infected; the low elevation sources were much less infected; and the mid-elevation sources showing moderate infection. In a subsequent test to confirm these observations, a young plantation of Jeffrey pines from the same seed sources was inoculated in 1980 and 1981 with a local source of seeds of dwarf mistletoe (A. campylopodium) on ponderosa pine (Scharpf, Kinloch and Jenkinson 1992).

In 1980, 3000 inoculations were made on 50 trees from each of 3 seed sources for a total of 9000 inoculations. In 1981, the same trees were inoculated with a total of 4500 seeds. After five years, results of the study confirm the earlier work by Scharpf and Parmeter (1967) in that the low elevation, the Foresthill source of Jeffrey pine showed significantly fewer trees infected and fewer infections per tree than the other seed sources tested (table 2). Therefore, it appears that different geographic populations of Jeffrey pines express different levels of resistance to A. campylopodium. Unfortunately, the factors that determine resistance among different populations remain unknown.

Table 2—Dwarf mistletoe seed retention and infection of Jeffrey pine inoculated in 1980 and 1981

<table>
<thead>
<tr>
<th>Jeffrey pine seed sources: Site and time of collection a</th>
<th>Trees infected in 1985</th>
<th>Seeds on branches the spring after inoculation b</th>
<th>Infections in 1985–1986 from seeds on branches percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>High Meadows, Fall 1980</td>
<td>50.0</td>
<td>12.7</td>
<td>21.8</td>
</tr>
<tr>
<td>South Lake Tahoe, Fall 1980</td>
<td>24.0</td>
<td>8.7</td>
<td>14.5</td>
</tr>
<tr>
<td>Foresthill, Fall 1980</td>
<td>8.0</td>
<td>6.0</td>
<td>4.4</td>
</tr>
<tr>
<td>High Meadows, Fall 1981</td>
<td>46.0</td>
<td>34.6</td>
<td>13.0</td>
</tr>
<tr>
<td>South Lake Tahoe, Fall 1981</td>
<td>44.0</td>
<td>28.2</td>
<td>7.8</td>
</tr>
<tr>
<td>Foresthill, Fall 1981</td>
<td>20.0</td>
<td>26.2</td>
<td>3.3</td>
</tr>
<tr>
<td>Laguna Mt., Fall 1981</td>
<td>48.0</td>
<td>41.3</td>
<td>9.1</td>
</tr>
</tbody>
</table>

a Fifty trees per source were inoculated, except for 25 trees from Laguna Mountain.
b Inoculation totaled 3000 seeds per source in 1980, and 1500 seeds per source in 1981, except 720 for Laguna Mountain source.

Adapted from Scharpf and others (1992)

Ponderosa Pine, Oregon

Another study that shows a very convincing case for resistance was that conducted in Oregon by Roth in the 1960s (Scharpf and Roth 1992). Thirty resistant candidate ponderosa were selected from various locations in Oregon, and 12 to 15 grafts made from each candidate on nursery grown rootstock. Over 400 of these grafted trees were then out planted in central Oregon from 1967 to 1969 in a random block design in a heavily infested stand of ponderosa pine. The final data on the plots was collected 20–22 years later in 1989. Summaries of the results are as follows:

- A wide variation in resistance was found among the grafted selections ranging from 100 percent infection of some selections to no infection of others.
- The mean number of infections varied widely among the clones. Also the greatest number of infections was found in clone trees with the greatest percentage of trees infected.
- Ungrafted “field run” seedlings from three forests were heavily infected, showed no evidence of resistance and experienced heavy mortality before the end of the test.

The results of these tests in ponderosa pine correspond closely with those in Jeffrey pine in that there was a wide variation in resistance among trees from different geographic locations and that resistance persisted after many years of exposure to the disease.

Ponderosa Pine, California

The last study I want to discuss is that currently being conducted by Paul Stover and Dennis Ringnes at the U.S. Forest Service, Tree Improvement Center, Placerville, California. Details of this study appear in the mistletoe committee report in these proceedings. Based on early observations by Dr. W. W. Wagener on possible resistance in eastside ponderosa pine, guidelines were established to select and test resistant candidate pines. From 1993 to 1996, field crews selected 109 resistant candidate trees from several locations in northern California. In 1997, seedlings from open pollinated resistant candidates, control crosses and susceptible trees were planted in a random block design at the nearby Badger Hill Tree Orchard. In 2001–2002,
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each tree was inoculated at the rate of 10 seeds per tree; and in 2002–2003, the inoculation rate was 20 seeds per tree.

Although it is too soon for any meaningful results to be obtained, the investigators are optimistic that useful information will result from the tests. Infections are developing from the inoculations that we hope will provide sufficient data for a statistically sound determination of resistance among the selections and crosses.

Conclusions

So what are some of the conclusions we can draw from past and present work on dwarf mistletoe resistance?

- Resistance has been observed in the forest by several investigators, and resistance is probably much more common than we realize.
- There is wide variation in resistance within and among conifer species, and variation in resistance within species from different geographic areas.
- Resistance is expressed in many ways, and probably is the result of a complex multigenic process.
- Lastly, we know very little about the mechanisms and inheritance of resistance in conifers to dwarf mistletoe.

So why is there so little research being conducted on resistance to dwarf mistletoes? After all, they are among the most damaging and widespread pathogens in western North America.

- Managers, in general, see little need for widespread use of resistance in the forest against these diseases. Current thinking suggests that most dwarf mistletoes can be managed at tolerable levels through silvicultural or other means. Use of resistance mainly seems applicable only in high use, high value areas.
- Development of resistance is considered to be an expensive long-term commitment. Few research institutions and scientists are willing to devote the time and resources necessary to achieve meaningful results.
- Understanding and developing resistance is perceived to be a high risk, very complex area of research with the chance of success questionable at best. Current evidence suggests that resistance is likely a complex multigenic process, and we do not have the technology or resources to obtain results and provide answers.

So where do we go from here?

- Field workers should continue to report mistletoe resistant candidates they observe in the field. Where appropriate, then candidates should be tagged and located for future examination.
- We should protect the currently known resistant candidates, particularly valuable ones like the ones on the “Roth Pringle Butte Plots”.
- We should continue to propagate and field test candidates as resources allow.
- A centralized database of known resistant candidates should be established. This will allow investigators ready access to test material when resources and technology allow for further research.
- Investigators should keep abreast of new developments in the area of genetics and biotechnology that may apply to testing and development of resistance in dwarf mistletoes.
- A multidisciplinary approach to research and development efforts should be considered as the most efficient way to obtain the desired results.

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References

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