

RESEARCH ARTICLE

Assessing the durability, stability, and usability of genetic resistance to a non-native fungal pathogen in two pine species

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Societal Impact Statement

Damage to white pine forests by white pine blister rust, caused by the non-native invasive fungal pathogen *Cronartium ribicola*, is affecting people across western North America. The loss of white pine forests can impact economic opportunities as well as ecosystem and human well-being. In this paper we quantify the durability, stability, and usability of natural genetic resistance to the disease in two white pine species using long-term field trials. By combining quantitative genetic resistance breeding programs with data from field validation trials, land managers and policy makers will be able to better ensure healthy forests for the future.

Summary

- Many tree species are susceptible to non-native pathogens or pests. Their level of susceptibility can be so severe that they are extirpated, and their use for forest restoration or reforestation is curtailed. Programs to find, enhance, and utilize genetic resistance in tree species are underway. Because trees are long-lived, resistance must be effective for decades to centuries to be useful. Resistance needs to be durable, stable, and present at a useful level. Field plantings provide the best opportunity to assess resistance durability and stability across a range of environments.
- In this paper we examine the durability and stability of resistance levels, previously identified in seedling screening trials, of two white pine species, *Pinus monticola* (western white pine) and *P. lambertiana* (sugar pine) to white pine blister rust, using eight 15 to 20-year-old field trials in Oregon and northern California.
- We found that resistance varies for each host species and in different environments. Major gene resistance (MGR) may have limited utility and the search for resistance should include all types of resistance, as quantitative disease resistance (QDR) appears to be more durable than MGR in the long term at many sites in our study.
- Our data provide encouragement and support for the use of long-term field validation studies in combination with quantitative genetic resistance breeding programs. We advocate for an increased use of field trials to ensure that resistance is effective for restoration and recovery against invasive pathogens and pests.

KEYWORDS

Cronartium ribicola, disease resistance, major gene resistance, *Pinus lambertiana*, *Pinus monticola*, quantitative genetic resistance, white pine blister rust

1 | INTRODUCTION

Tree species in natural, plantation, and urban forests are being damaged or killed by diseases and pests worldwide (Anagnostakis, 2001; Liebhold et al., 2017; Lovett et al., 2016). The damage and mortality are often high enough that they threaten the viability of the species and its function in forest ecosystems throughout its geographic range. The most severe cases often involve non-native pathogens and pests to which the affected tree species has not co-evolved (Budde, Nielsen, Ravn, & Kjær, 2016) and often has little natural genetic resistance. In North America, white pine blister rust (Kinloch, 2003), chestnut blight (Anagnostakis, 1987), Dutch elm disease (D'Arcy, 2000), and emerald ash borer (Poland & McCullough, 2006) are some of the non-native diseases and pests causing mortality to native trees. Fortunately, there is often at least a low level of genetic resistance to these diseases and pests, despite the lack of coevolution (Sniezko, 2006; Sniezko & Koch, 2017). Usually, little can be done to stop the mortality of susceptible trees, and the best solution for retaining these species in future forests will be to find and utilize the genetic resistance that does exist (Boshier & Buggs, 2015; Showalter et al., 2018; Sniezko & Koch, 2017; Telford, Cavers, Ennos, & Cottrell, 2015; Woodcock, Cottrell, Buggs, & Quine, 2018). Identifying and increasing the frequency of genetic resistance to diseases and pests, and deploying them in forests, has been recognized as a key step in maintaining healthy forests (Showalter et al., 2018).

Successful use of genetic resistance will depend on several key considerations including (a) the durability of resistance; (b) the stability of resistance; (c) whether the resistance is passed onto progeny (heritable); and (d) whether the frequency and level of resistance is high enough for land managers to consider its use. Durable resistance refers to whether the pathogen or pest will evolve virulence that renders the resistance ineffective. Durability can often only be determined by planting the materials over many hectares and over many years in the presence of the pathogen or pest (Johnson, 1984; Vale, Parlevliet, & Zambolim, 2001). Stability of resistance refers to whether the resistance is effective over a range of environmental and climatic conditions. In some cases, a change of a few degrees in temperature can impact the efficacy of the resistance (Cheng et al., 2013; Wang, Bao, Zhu, & Hua, 2009; Zhu, Qian, & Hua, 2010). We suggest adding the term “usability” to denote that in the case of using genetic resistance in forestry, the frequency and level of heritable resistance in individuals within a seedlot used for restoration or reforestation may vary and land managers will need to decide whether the current materials can be used to meet their objectives based on all of the available data.

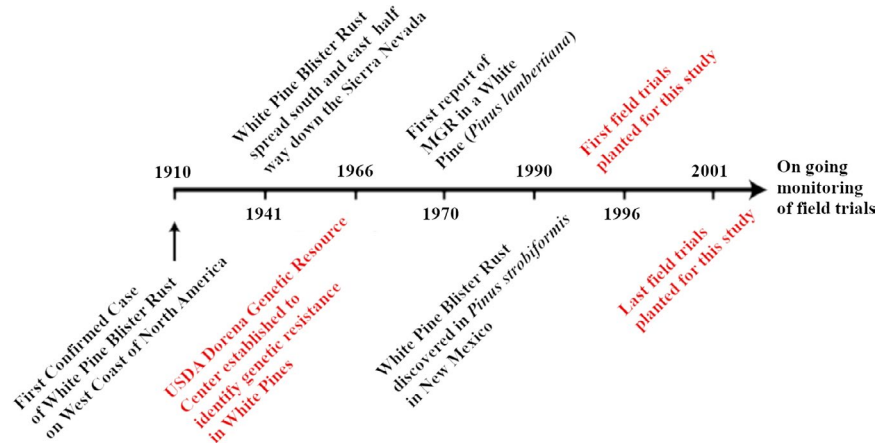
Although durability and stability of resistance in food crops is important, in most cases the crops are annuals and new plantings are

established every year. In these cases, new resistant genotypes, if available, can be planted in future years. The situation is different for forest trees. Trees are long-lived organisms and will be present in the environments for decades to centuries. For native forest restoration efforts, planted seedlings are intended to become parent trees of future generations of seedlings. Thus, it is more important in forestry than in agriculture that genetic resistance be durable, stable and of a useful level to land managers. In addition, most tree species grow over a wide range of environments, with very little management inputs after planting, and they face many abiotic and biotic challenges. Therefore, most restoration and reforestation efforts with forest trees will also need to maintain genetic diversity and future adaptability and thus the seedlings planted should be the heterogeneous product of several to many parent trees rather than a single cultivar (genotype).

The programs to develop genetically resistant populations of *Pinus monticola* (western white pine) and *P. lambertiana* (sugar pine) to white pine blister rust, the disease caused by the fungal pathogen *Cronartium ribicola*, began more than 50 years ago, and are among the longest ongoing programs in North America (McDonald, Zambino, & Sniezko, 2004) with a focus on a non-native disease in forestry (Figure 1). These programs present an ideal opportunity to examine questions on the use of resistance in forestry. In this paper, we examine the field performance of genetic resistance to white pine blister rust in two species of white pines and use these results to discuss the importance of durability, stability and “usability” of genetic resistance in forest trees. Both major gene resistance (MGR) and quantitative disease resistance (QDR) have been documented in *P. monticola* and *P. lambertiana* (Kinloch, 2003; Kinloch, Parks, & Fowler, 1970; Kinloch, Sniezko, Barnes, & Greathouse, 1999; Sniezko, Smith, Liu, & Hamelin, 2014), as well as virulence to the MGR in both species (Kinloch & Dupper, 2002; Kinloch, Sniezko, & Dupper, 2004).

To more fully appreciate the difference in resistance types and their durability, stability, and usability, we must first define the disease symptom expectations of having either MGR or QDR. In the case of white pine blister rust, both MGR and QDR are relatively rare for *P. monticola* and *P. lambertiana* (Kinloch, 2003; Kinloch, Davis, & Burton, 2008; Sniezko, Kegley, & Danchok, 2008). The progeny of parent trees with MGR will segregate for cankered and canker-free phenotypes due to the simple gene inheritance of MGR (Kinloch, 2003; Kinloch et al., 1999). Often, progeny of *P. monticola* and *P. lambertiana* parent trees with MGR show little or no partial resistance in seedling inoculation trials or in the field when exposed to virulent genotypes of the rust (R. Sniezko, unpublished data). Thus, in the presence of virulent rust genotypes all trees with only MGR will be highly susceptible to the disease, but trees in some families may also

FIGURE 1 The timeline of white pine blister rust progression and disease resistance screening in *Pinus monticola* and *Pinus lambertiana* in North America. Black text is used for historical events relevant to white pine blister rust in all white pine species and red text is used for events relevant to this study specifically



have QDR and show a low level of resistance. In contrast to MGR, the progeny of parent trees with QDR, a form of resistance that will often have a more complex pattern of inheritance (King, David, Noshad, & Smith, 2010; Poland, Balint-Kurti, Wissner, Pratt, & Nelson, 2009), exhibit a range of phenotypes (Sniezko et al., 2014). For example, some seedlings from a single QDR family will be highly susceptible while others will be canker-free and in between these two extremes some seedlings will have latent stem infections (Kegley & Sniezko, 2004; Sniezko et al., 2014), some will have fewer infections (Kegley & Sniezko, 2004), some will have complete or partial bark reactions (Hoff, 1986; Sniezko et al., 2014; Struckmeyer & Riker, 1951), some trees will tolerate active cankers (Hunt, 1997; Sniezko et al., 2008) and either die later or continue to live, and some trees may have a combination of these traits (Sniezko et al., 2014). The combination, level, and effectiveness of resistance will vary among the progenies of different QDR parent trees. Many of the components of QDR appear to be slowing the growth of the fungus (Struckmeyer & Riker, 1951; Hoff, 1986; see Kolpak, Sniezko, & Kegley, 2008 for discussion of some of the families included in this study; see table 1 in Sniezko et al., 2014 for examples of families with a range of QDR). Relatively little is known about the durability and stability of QDR to date (Nelson, Wiesner-Hanks, Wissner, & Balint-Kurti, 2018), due to a lack of long-term field trials such as the ones we report here, though it is thought to be more durable than MGR for many host species (Poland et al., 2009).

2 | MATERIALS AND METHODS

Eight field trials planted between 1996 and 2001 in northern California and western Oregon are examined here. Each planting was assigned a trial identity which consists of an abbreviation of the trial purpose (rust validation (RV) in this case), a number, and a letter. Some trials were planted with the same grouping of families or orchard seedlots, and these are identified by a common trial number with different letter suffixes (Table 1). Trials RV1a and RV1b share the same 25 families (12 *P. lambertiana*, 13 *P. monticola*); trials RV6a, 6c, 6e, and 6f have 80 *P. monticola* families

or orchard seedlots in common; and trials RV4c and RV7a have different subsets of families from each other and generally from the other trials. All eight trials include *P. monticola*, and two trials also include *P. lambertiana* families (Dataset S1). Most trials were planted as randomized complete blocks (6 to 12 blocks per trial) using family row plots (1 to 12 seedlings per family), but RV6e was planted as a completely randomized design with 7–24 seedlings per seedlot (Table 1).

The trials have been assessed periodically over the last 15–20 years (Table 1). *Ribes* is the principal alternate host for *C. ribicola* (see Geils, Hummer, & Hunt, 2010 for a discussion). Infection at all sites relied on *Ribes* existing in the areas, except for RV1a, where some *Ribes* plants were inter-planted among rows of pines to help ensure infection. Trials with moderate to very high levels of white pine blister rust incidence were selected for examination here, with the exception of RV6A where infection was only 6.3%.

The seedlots in these trials are representative of those available from the United States Department of Agriculture (USDA) Forest Service's Pacific Northwest Region's (PNW) blister rust resistance program in the mid to late 1990s. All of the seedlots or the parents involved in crosses, have been previously evaluated for MGR and QDR in artificial inoculation trials of seedlings (for details and some results see Bingham, 1983; Kegley & Sniezko, 2004; Kolpak et al., 2008; Sniezko et al., 2014). Seedlots included full-sib families, half-sib families, and bulked seed orchard lots. The parents of most of the Pacific Northwest seedlots were original field selections, but a few seedlots were progeny selections from tested families. One to three susceptible controls (seedlots showing very high susceptibility in one or more seedling inoculation trials) were included in each trial (Dataset S1). Eight families were open-pollinated seedlots collected from surviving trees in the heavily infected Grass Creek and Blodgett plantings, two of the oldest field trials of *P. monticola* planted in Oregon (Sniezko et al., 2004): seven of these were planted in the RV6 trials, two of these (including one also in RV6 trials) were planted in RV7. In general, the level of survival (<5%) and QDR is low in seedling screening trials (Kegley & Sniezko, 2004; Sniezko et al., 2008) in most *P. monticola* and *P. lambertiana* families, including the families

TABLE 1 Trial name, planting year, planting state, organization/collaborator name (NF = National Forest, BLM = Bureau of Land Management), elevation and species planted are listed for each rust validation trial

White pine blister rust infection at eight rust validation field sites											
Trial name	Planting year	Location	Organization/ Cooperator	Elevation (m)	Species	Number of families or orchard lots	Blocks (range of seedlings per family)	Number of assessments (and list year after plantings)	Mean canker percent (family range)	Mean mortality (family range)	Mean non-rust mortality
RV1a ^a	1996	California	Happy Camp	756	<i>P. monticola</i>	13	12 (4–6)	10 (3,4,5,6,7,9,11,16,18,20)	59.9 (23.4–90.5)	27.0 (0–60.9)	5.8 (0–30.6)
RV1b ^{b,c}	1997*	Oregon	Umpqua NF/ Cottage Grove RD	1,057	<i>P. monticola</i>	13	8 (2–12)	9 (2,4,5,6,7,8,11,14,19)	86.0 (36.8–100)	61.5 (15.8–96.3)	16.7 (2.8–45.7)
RV4c ^a	1998	Oregon	Umpqua NF/ Cottage Grove RD	1,145	<i>P. monticola</i>	49	8 (3–4)	6 (4,5,6,9,13,17)	98.9 (77.4–100)	95.1 (32.3–100)	11.4 (0–30.0)
RV7a ^a	2000	Oregon	BLM Tyrrell	320	<i>P. monticola</i>	13	8 (2–6)	8 (5,6,8,9,12,14,16,18)	84.7 (27.3–100)	57.4 (6.8–89.4)	3.5 (0–17.8)
RV6d ^d	1999	Oregon	Confederated Tribes of Warm Springs	1,146	<i>P. monticola</i>	80	6 (2–5)	3 (5,11,15)	6.3 (0–28.6)	1.6 (0–11.1)	32.9 (8.3–65.2)
RV6c ^e	1999	Oregon	Umpqua NF/ Cottage Grove RD	975	<i>P. monticola</i>	80	6 (1–7)	3 (7,12,16)	99.2 (83.3–100)	65.1 (6.7–100)	1.8 (0–8.7)
RV6e ^f	1999	Oregon	BLM/Colton	308	<i>P. monticola</i>	80	1 (7–24)	3 (4,9,16)	65.1 (21.1–100)	2.1 (0–16.7)	1.5 (0–14.3)
RV6f ^g	2001	Oregon	Umpqua NF/ Diamond Lake RD	1,361	<i>P. monticola</i>	81	6 (3–5)	3 (6,11,14)	85.0 (43.8–100)	19.4 (0–53.3)	31.7 (0–70.8)

Notes: The number of families (seedling progeny grown from a seed or orchard source tree) included at each site and species planted are listed. Blocks and the range of seedlings per family per block, in parenthesis is listed. The number of assessments for each trial and the number of years since the trial was planted in parentheses. The mean percent canker and the family range in parentheses indicate the level of infection at a site as well as the mean mortality and family range per site. The mean non-rust mortality indicates the level of non-disease caused mortality that occurred at each site. Sites on the Cottage Grove Ranger District, Umpqua National Forest are close to the first confirmed origin of the *vcr2* strain of rust. Superscripts in trial names correspond to specific experimental designs for each trial.

^aRandomized complete blocks using family row plots.

^bRandomized complete block using family row plots: three seedlots had too few seedlings to occupy each of the eight blocks.

^cOne seedlot had an excess of seedlings and was planted as a ninth block containing 31 seedlings.

^dRandomized complete block noncontiguous seedlings per seedlot per block.

^eRandomized complete block noncontiguous seedlings per block: two seedlots had too few seedlings to occupy each of the six blocks.

^fCompletely randomized design with 7–24 seedlings per seedlot.

*Part planted June 1997, other half October 1997.

planted in the field trials, but notable exceptions in *P. monticola*, such as two open-pollinated half-sib families from field collections (05081–003 (in RV1, RV7), 21105–052 (in RV1, RV6), and the full-sib cross 06024–506 × (06024–504 × 06204–511) (in RV4)) occur. These three families are in the top one percent for QDR of more than 4,500 families evaluated over the first 30 years of the resistance program (R. Sniezko, unpublished data). For some details of these families see Sow# 75, 73, and 74, respectively in table 1 of Sniezko et al., 2014). All trials have families with identified MGR and others known to possess QDR (Dataset S1). Some further background on seedlots, resistance type and early field results in the RV1 and RV4 trials are reported elsewhere (Sniezko et al., 2004 for RV1a; Kolpak et al., 2008 for RV4). In addition, one wind-pollinated F_2 seed orchard lot (Bingham F_2) composed of families previously evaluated for white pine blister rust resistance at the USDA Forest Service's Inland West resistance program was included in the RV6 tests. This QDR Bingham F_2 orchard lot (for a review see Bingham, 1983; sow 71 in table 1, Sniezko et al., 2014) has been widely planted for several decades in the Inland West and elsewhere and has no MGR (R. Sniezko, unpublished data).

Assessments of the trials included data on number of stem infections, type of stem infections, location of infections (main bole and/or branches), severity of overall infection, and mortality. Some small infections (or errors) from early assessment become inactive and not visible in later years, leading to slight fluctuations downward in the percent cankering recorded over time in a few families (and can be seen in the figures found in the results). In this paper we report on family percent stem symptoms and percent mortality. Graphs are produced using R version 3.5.0 (R Core Team, 2018).

3 | RESULTS

For *P. monticola*, the overall level of blister rust infection at the eight field trials varied from 6.3% to 99.2%, while the two sites with *P. lambertiana* were 91.6% and 97.0% (Table 1; Dataset S1). For *P. monticola*, infection levels at RV6a were low (6.3%), those at RV1a, RV1b, RV6e, RV6f, and RV7 were moderate (59.9% to 86.0%), and those at RV4c and RV6c were high (99%) (Table 1). At seven of the eight trials, at least one family had 100% of trees with a canker for *P. monticola* and in both trials with *P. lambertiana* some families were 100% infected (Dataset S1). The temporal dynamics of infection at the sites varied considerably with, for example, RV4c having many *P. monticola* families with >90% infection by the fifth year after planting (Figure 2), while only one *P. monticola* family has reached 90% infection at RV1a after 18 years (Figure 3; Dataset S1). For *P. lambertiana*, infection was relatively rapid at both RV1a and RV1b, with many non-MGR families showing >90% infection at RV1b by the 5-year assessment (Figure 3). Mortality often lagged several years behind infection and few sites had reached high average mortality (Figures 2–4), with the *P. monticola* at RV4c (95.1%) and the *P. lambertiana* at RV1b (83.0%) with the highest mortality. For *P. monticola*, the mean infection varied from 6.3% to 99.2%, while the mean level of rust

related mortality varied from 1.6% to 95.1%. The average number of cankers per alive, infected tree was also highly variable. For instance, at RV6e the average for infected, alive trees were 3.8 cankers per tree, while at RV6c the average was 25.1 cankers per tree.

Relative Resistance of *P. monticola* and *P. lambertiana* to white pine blister rust: The higher susceptibility of *P. lambertiana* compared to *P. monticola* is evident at the two sites, RV1a and RV1b, examined here (Figure 3). The mean infection of the 12 *P. lambertiana* families is 97.0% for RV1a and 91.6% for RV1b. This compares to 59.9% for RV1a and 86.0% for RV1b for the 13 *P. monticola* families. The difference is even greater for mortality. *P. lambertiana* mortality at RV1a is at 71.5% and is 83.0% at RV1b, versus 27.0% and 61.5% for *P. monticola* at these two sites, respectively.

RV1a is known to have the *vcr1* strain of rust present which would overcome MGR in sugar pine, while RV1b is known to have the *vcr2* strain of the rust which would overcome MGR in *P. monticola* (Kinloch et al., 2004). The early effectiveness of MGR played out as expected at the two sites with MGR for *P. lambertiana* being generally ineffective at RV1a and the MGR for *P. monticola* being ineffective at RV1b (Figure 3). However, a small to moderate increase in canker percentage in the most recent assessments in the three *P. lambertiana* MGR families at RV1b and the two *P. monticola* MGR families at RV1a suggests that *vcr1* and *vcr2* may now be having some impact on infection at RV1b and RV1a, respectively, but with little impact yet on mortality.

P. monticola resistance: All sites exhibited variation in percent cankering and mortality among the seedlots planted (Dataset S1). The susceptible control(s) had the highest (or among the highest) infection and mortality at all sites. For percentage of trees with cankers, the range in seedlot variation was the least for RV4c (77.4%–100%) and RV6c (83.3%–100%), the two sites with highest overall infection levels (Figure 2a; Figure 4b). Despite the high infection level at these two sites, there was a large range in mortality from white pine blister rust, with some seedlots at 100% mortality, but others at less than 40% mortality (Figures 2–4). Additionally, the three top families from seedling inoculation trials also had the highest, or among the highest, resistance in the RV1, RV4, RV7, and RV6 trials in which they were included (Dataset S1).

The QDR Bingham F_2 orchard seedlot has been the most widely used *P. monticola* reforestation material for decades, particularly in the Interior West of the United States. The percentage of trees with cankers varies dramatically over the four RV sites where it was planted, with RV6a showing low infection at 6.3% and RV6c showing 100% infection (Figure 4). At this point, despite the high infection at RV6c, only 45% of trees are dead from rust, essentially the same level as one of the susceptible controls. However, the severity of infected living trees is somewhat lower in the Bingham seedlot than the susceptible control.

4 | DISCUSSION

4.1 | Durability of white pine blister rust resistance

The capacity of *C. ribicola* to evolve virulence to MGR or QDR is a concern. Previous work has shown that virulence to the MGR in both *P. lambertiana* and *P. monticola* has arisen (Kinloch et al.,

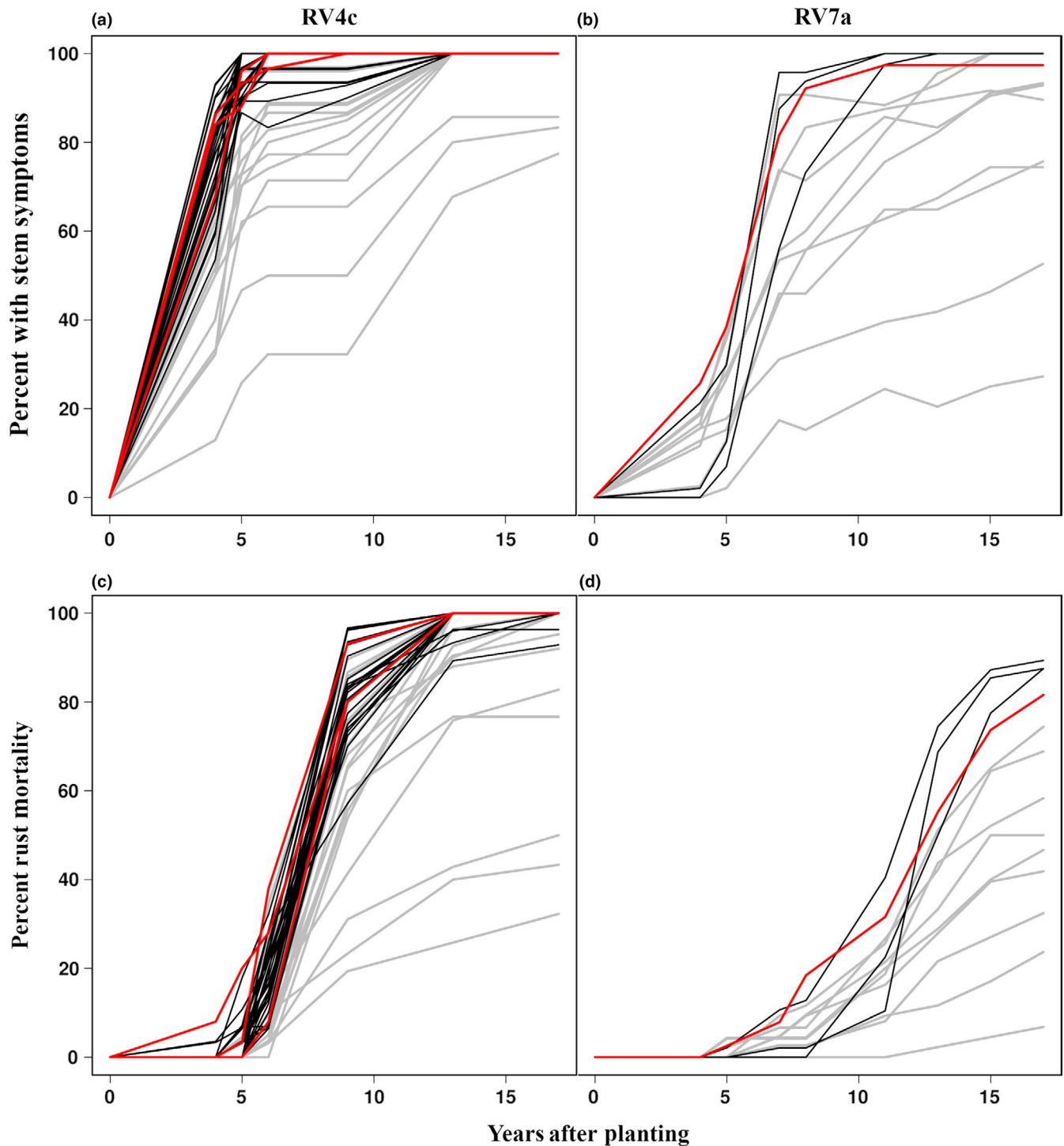


FIGURE 2 The change in *Pinus monticola* disease infection for trials RV4c and RV7a represented by the percentage of trees with stem symptoms (a, b) and the percentage of trees dead from rust (c, d) through time. Red lines show susceptible controls, black lines show major gene resistant families, and gray lines show families with quantitative disease resistance

2004), and that the *vcr2* strain with virulence to MGR in *P. monticola* is more widespread than the *vcr1* strain with virulence that affects MGR in *P. lambertiana* (Kinloch et al., 2004; R. Sniezko, Unpublished data). Our results support this. However, even in the presence of virulence, at some sites, the MGR families are continuing to show some benefit with the level of infection <100% or

less mortality than the susceptible control (e.g., RV6f; Figure 4h). An earlier assay of rust genotypes at RV1a confirmed the presence of *vcr1*, but also indicated that *vcr2* was present at a low frequency (Kinloch et al., 2004). This confirmation at RV1a, in the presence of only several small plantings of *P. monticola* (Kinloch et al., 2008; Sniezko, Bower, & Danielson, 2000), suggests that

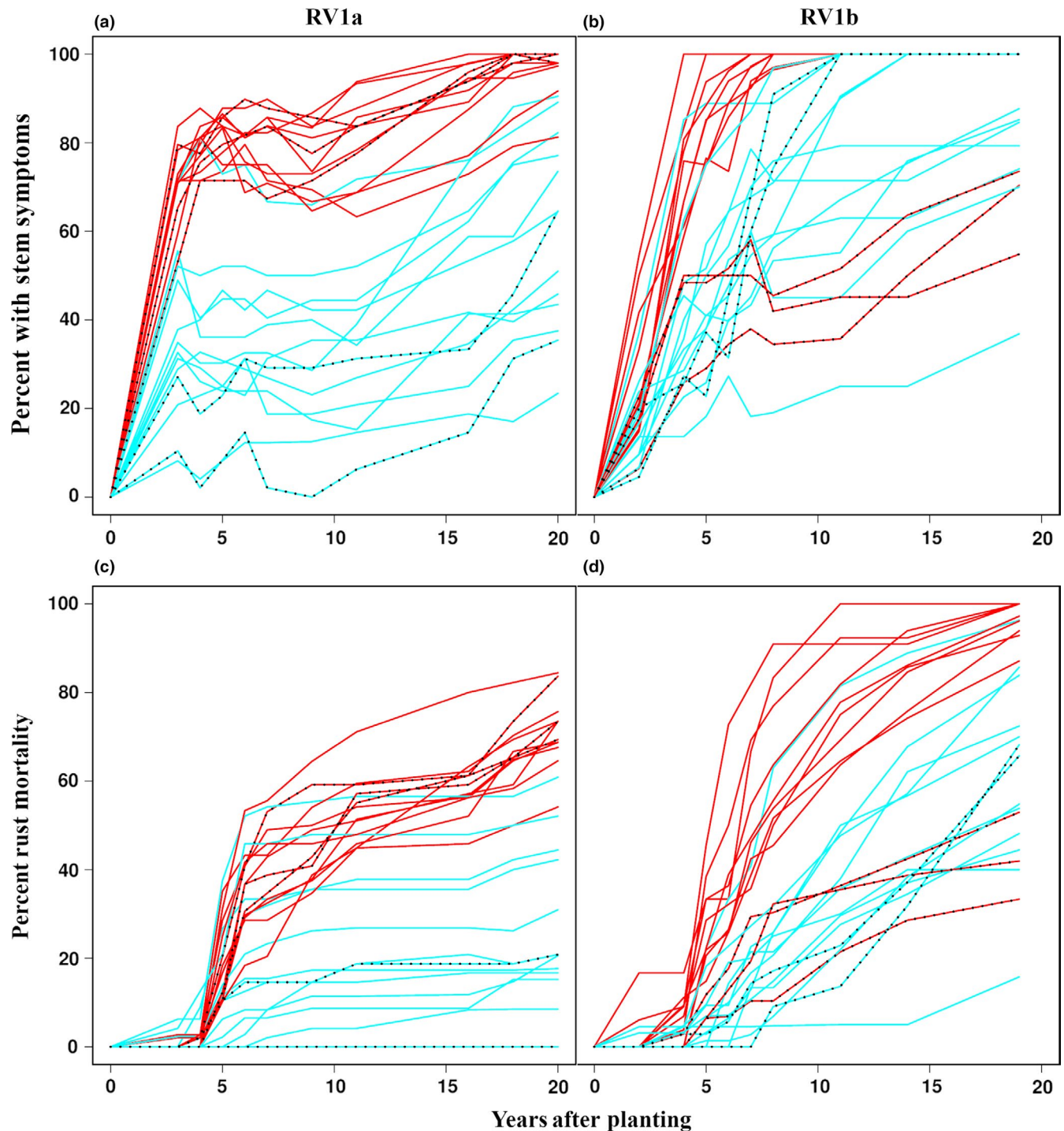


FIGURE 3 The change in *Pinus monticola* and *P. lambertiana* disease infection represented by the percentage of trees with stem symptoms (a, b) and the percentage of trees dead from rust (c, d) through time for trials RV1a and RV1b. Red lines show *P. lambertiana*, cyan lines show *P. monticola* and dotted lines show major gene resistant families in both species

MGR in *P. monticola* can be rapidly overcome by the evolution of the fungus, though this may not occur on all sites (Hunt et al., 2018). The infection of *P. monticola* at RV1a involved some seedlots with MGR and some seedlots with QDR. Encouragingly, QDR tends to be more durable at this stage than MGR in these trials. MGR in *P. lambertiana* appears to be relatively more durable than MGR in *P. monticola*, with data from other trials in Oregon showing

effectiveness through at least age 30, even in trials with >90% cankering (R. Sniezko, Unpublished data). Restoration and reforestation plantings will utilize both types of resistance, for example *P. lambertiana* in the Pacific Southwest Region and *P. lambertiana* and *P. monticola* in Pacific Northwest Region. The combination of resistance types may provide the best scenario for survival of these species from the current generation of seed orchards, while

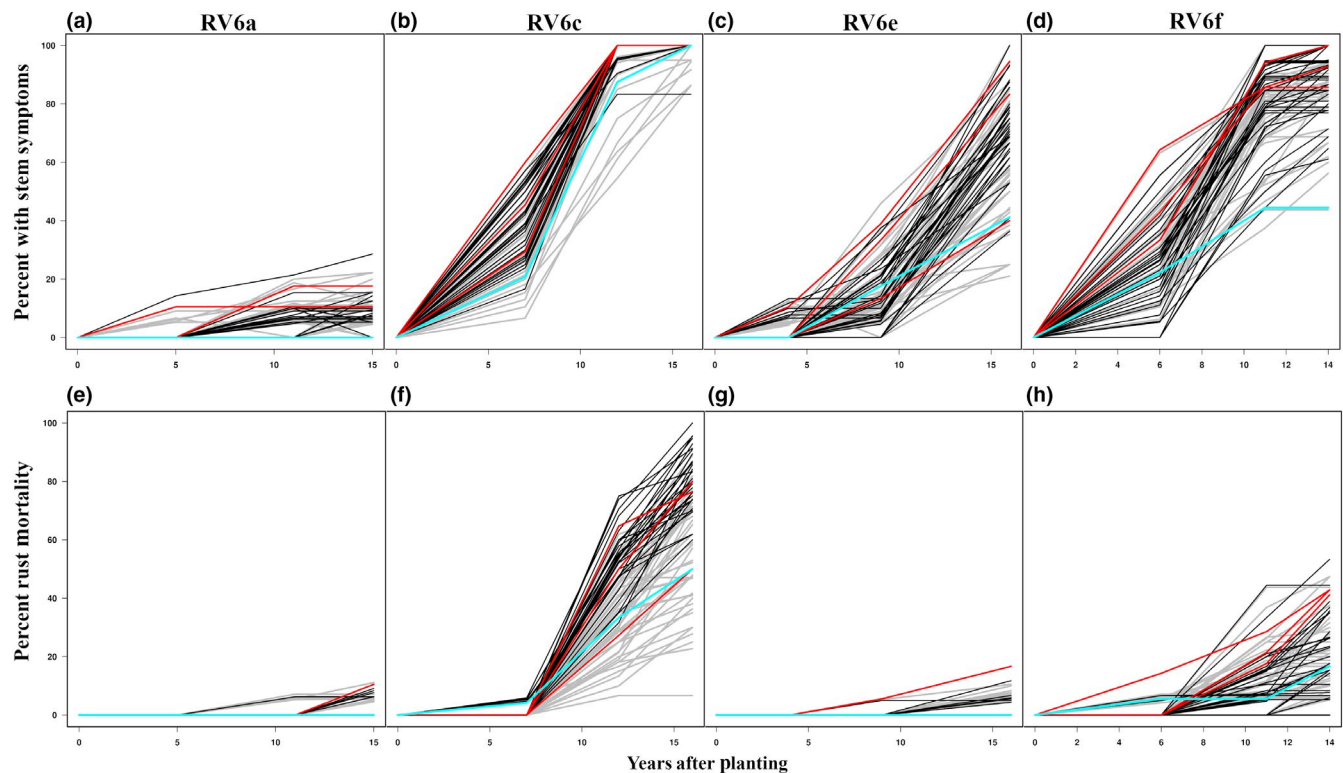


FIGURE 4 The change in *Pinus monticola* disease infection represented by the percentage of trees with stem symptoms (a, b, c, d) and the percentage of trees dead from rust (e, f, g, h) through time at four trials in the RV6 test series. Red lines show susceptible controls, gray lines show known quantitative disease resistant families, black lines show major gene resistant families, and cyan lines show the Bingham F_2 quantitative disease resistant orchard seedlot

breeding efforts are underway to increase the level of QDR in future orchard populations.

4.2 | Stability of white pine blister rust resistance

Hunt (2004) noted that the Bingham F_2 orchard lot with QDR conferred a much higher level of resistance in interior British Columbia (BC) or higher elevation coastal BC than at a low elevation site in BC. Hunt attributed this to variation in environmental conditions (possibly temperature), and not differences in inoculum. Our results are similar, and the 100% infection of this orchard lot in the RV6c site raises some concerns and further work will be needed to understand the resistance of this seedlot better, and the range of sites on which it will be most useful. In seedling inoculation trials at the USDA Forest Service's Dorena Genetic Resistance Center (DGRC), the efficacy of MGR in both *P. monticola* and *P. lambertiana* seems to vary in different years, with canker-free percentages often being lower than expected (Danchok et al., 2012; R. Sniezko, Unpublished data). The findings at DGRC suggest that temperature and/or seedling physiology or phenology in different inoculation years may have an influence on the stability of resistance. Seedling screening assays for resistance for *P. monticola* and *P. lambertiana* often occur at lower elevation environments with different climatic conditions than where many *P. monticola* and *P. lambertiana* grow. Further studies are needed to test the relationship between

environmental and climatic conditions on the stability of resistance. In addition, it is worth noting that in China and South Korea there are examples of where the co-evolved pathogen and white pine species are showing higher levels of susceptibility than found in the past (La, 2009; Zhang, Lu, Sniezko, Song, & Man, 2010), suggesting that changes in management or environment may have altered the previous resistance equilibrium or possibly that new virulence has arisen. This is a reminder that resistance is important but needs on-going field testing and monitoring to determine its level of efficacy over desired environments and through time.

4.3 | Usability of white pine blister rust resistance

In crop species and a few forest species, just one or a few genotypes may be planted to maximize genetic gain in traits such as resistance, yield, and uniformity. However, in most cases in forestry, seed for reforestation or restoration is from a mix of many parent trees in the forest or in seed orchards. Many forest tree programs are in just the first or second cycles of selection for resistance and the efforts to keep the necessary broad genetic base, coupled with durable and stable resistance, may mean modest gains in resistance in early generations. Field trials provide estimates of achievable levels of survival using resistance on sites of varying blister rust hazard. Land managers will be able to use these estimates to choose the number of seedlings they need to plant to achieve desired density of

P. monticola or *P. lambertiana* in their areas. Planting low to moderate hazard sites will require fewer seedlings from resistant parent trees or orchards. For the sites of extreme rust hazard, more seedlings will be needed, or higher levels of resistance may be required. Breeding to increase the level of resistance, and to combine MGR and QDR in orchards, is underway and this next generation of resistant materials would be anticipated to be more suitable for the very high rust hazard areas.

The range of *P. monticola* resistance expressed by the seedlots depends on the types of resistance (MGR or QDR), whether the *vr2* strain of rust is present, the relative rust hazard of the site, and when the white pine blister rust infection was first evident at a moderate or high level. The larger trees are before they are infected may confer advantage by allowing some lower level of partial resistance to be more effective, or simply due to their size it may take more infections to seriously damage or kill trees. The high infection of the susceptible control provides an indication of the susceptibility of the majority of *P. monticola* or *P. lambertiana* present before white pine blister rust was present or what might be available in some wild open-pollinated seedlots used in early reforestation efforts.

4.4 | Implications for *P. monticola* and *P. lambertiana* reforestation and restoration

In this paper, we report on results of eight field trials of *P. monticola* and *P. lambertiana* to give guidance on the durability, stability, and usability of genetic resistance to pests and pathogens. We provide a direct comparison of the relative resistance in the field between the two white pine species. Our results are strengthened by the fact that that disease progression was closely monitored for 15–20 years under different environmental conditions. The subset of trials we examined provide insights about the utility of resistance for this pathogen. In addition, this system provides perhaps the best representation to date of what might be expected from genetic resistance in the first generation of breeding in forest trees.

Characterizing the level of disease resistance and its frequency in a family or seed orchard lot is an important consideration for forest managers deciding on using a specific species or seedlots to meet reforestation or restoration goals. Currently, most data on levels and types of resistance come only from seedling inoculation trials (e.g., Kegley & Snieszko, 2004). Inoculation trials can characterize the type, level, and frequency of resistance, but durability and stability of resistance are only detectable with field validation. There have been few systems in tree resistance programs (and notably to native trees and non-native pathogens or pests) studied long enough or specifically for QDR to make statements on the potential durability of resistance. In hybrid poplar, it is acknowledged that the potential durability of strategies involving qualitative and quantitative resistance is unknown at this point and further investigations are underway (Jorge, Dowkiw, Faivre-Rampant, & Bastien, 2005). There are ongoing studies aimed at identifying resistance genes (e.g., Muchero et al., 2018), but far fewer that report field trials of a decade or more

in duration. Many of the most notable applied programs are noted in Snieszko and Koch (2017) along with the two white pine species reported on in this paper. The *Chamaecyparis lawsoniana* (Port-Orford-cedar) program to develop resistance to *Phytophthora lateralis* may be the most advanced in terms of data from field trials (Snieszko et al. pers. comm.) and results indicate QDR is holding up well.

In the RV trials examined here, both *P. monticola* and *P. lambertiana* had some level of resistance to white pine blister rust, but in general the frequency of seedlings in a family with resistance was higher in *P. monticola* than *P. lambertiana*; a similar result was found in another trial (Kinloch et al., 2008). White pine blister rust infection in the field often occurs a few years after planting the trees but may take 5 or more years for even a susceptible family to approach 100% cankering, and even longer for high mortality to occur (Figures 2–4). The level of resistance provided by MGR in the absence of a race of rust with virulence often approaches 50% to 100% canker-free trees. Even so, as demonstrated in these field trials, the durability of the MGR gene can be relatively short-lived on at least some sites (in several of the trials here, some of the MGR families show as high or higher levels of infection than the susceptible control). For *P. monticola*, the durability of MGR in western Oregon trials was short-lived, though this result may be a product of either a mutation to a virulent strain or migration of virulence into the site, whereas the effectiveness of MGR in *P. lambertiana* is at least somewhat more robust in these trials (RV1b and R. Snieszko, unpublished). For *P. monticola*, many MGR families exhibited 100% cankered trees in some trials, but mortality lags slightly behind cankering. For *P. lambertiana*, only a few locations in California have been noted where MGR is no longer effective (Kinloch et al., 2004).

Quantitative disease resistance in both *P. lambertiana* and *P. monticola* is holding up so far. In the two trials here, the level of QDR in some of the original field selections of *P. monticola* is higher than that found in *P. lambertiana* and this has been noted in inoculation trials (Kegley & Snieszko, 2004; Snieszko et al., 2014) and at least one other field trial (Kinloch et al., 2008). However, in both species, the level of QDR is modest compared to susceptibility and on high hazard sites most trees become infected and many will die. The effectiveness of QDR will be judged by the level of survival in the QDR families compared to the susceptible control families. The results are encouraging, but because of the natural lag between infection and mortality more time will be needed to ascertain what level of survival QDR imparts. Forward selections of QDR families from inoculation trials has begun since these field trials were initiated, and the more recently established field trials will be examined to determine what increase in QDR has been achieved through breeding (e.g., Kinloch et al., 2012 with SP).

Future monitoring of these field trials (and others) will continue to provide managers with data on the efficacy of resistance through time. Depending on management objectives, resistance may be deployable alone, but in other cases the survival of trees may be augmented by use of silvicultural techniques, such as branch pruning to eliminate infections before they reach the bole of the tree (Schwandt, Lockman, Kliejunas, & Muir, 2010; Zeglen, Pronos, &

Merler, 2010). Classification of site rust hazard can be useful in providing land managers information on which sites to emphasize for planting the currently available level of resistance or the number of seedlings to plant to ensure adequate survival (Schoettle, Jacobi, Waring, & Burns, 2018).

4.5 | Implications for resistance programs with other non-native pathogens and pests

A key component to ensure a healthy forest and maintain biodiversity is the presence of genetic resistance to biotic and abiotic factors affecting trees. In the case of non-native pathogens or pests, there may be relatively little natural resistance, and programs to find and utilize this genetic resistance may be needed to retain full utilization of the species on the landscape. However, it is important to realize that not all seedlings from an applied resistance program will be resistant, and to quantify the frequency and level of resistance that might be available for reforestation or restoration efforts. With this information, land managers can decide on whether to use the species and can account for the frequency of resistance and expected survival by planting extra seedlings.

The usability, durability, and stability of resistance will be an important consideration on the ultimate success of reforestation and restoration. For resistance programs involving non-native diseases or pests there is currently little data available to provide guidance to land managers. As non-native pathogens and pests continue to spread and take their toll on forests and tree species worldwide, there has been greater interest in increasing the focus on genetic resistance, the tool with probably the best chance to restore these species. Until recently there have been relatively few decades old field trials to examine some key questions about genetic resistance. The example of *P. monticola* and *P. lambertiana* resistance to white pine blister rust over 15–20 years in the field trials reported here provides guidance on durability, stability, and usability of genetic resistance that can be applied in examining future outputs from resistant programs for other non-native pathogens or pests.

Evaluation of durability, stability, and usability of genetic disease resistance is an on-going process. Therefore, it will be essential to monitor at least a subset of field trials or restoration or reforestation plantings over many decades to gain stronger evidence of long-term durability and stability. The usability of resistance will depend on the species and land managers' objectives. In the case where the disease or insect kills the very high majority of the existing trees, then even a low frequency of resistance may be effective in ensuring survival of the species and the added costs of planting more seedlings may be deemed appropriate.

Identifying and breeding genetic resistance is an approach forest managers can use to keep species present on the landscape or for plantation forestry. Biotechnology and landscape genomic tools to aid non-native pathogen and pest resistance breeding are becoming available and are options being contemplated for use in forestry (Balkenhol et al., 2017; Boshier & Buggs, 2015; National Academies of Sciences, Engineering, and Medicine, 2019; Sniezko & Koch, 2017).

Whether through classical selective breeding or aided by genomics or biotechnology, the resistance approach is not a cure-all and needs to be well-documented to ensure its most effective utilization. In most cases, the level of resistance in the first-generation is limited and increases in resistance may be desired. Further breeding will provide an opportunity to increase the level of resistance, as well as its durability and stability. Breeding for genetic resistance is an invaluable tool for maintaining or restoring forest health. The lessons learned in the ongoing programs like those for white pine blister rust should help lead the way in contemplating some of the questions needed to be addressed and for efficiencies in the development of new resistance programs.

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AUTHOR CONTRIBUTIONS

R.A. Sniezko designed the trials, assisted with assessments and analysis, provided interpretation and led the writing of the manuscript; J.S. Johnson assisted with analysis, provided interpretation, and assisted with writing the manuscript; D.P. Savin assisted with assessments, provided analysis and assisted in writing the manuscript. All authors provided critical review during development of the manuscript and read and approved the submitted version.

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REFERENCES

- Anagnostakis, S. L. (1987). Chestnut blight: The classical problem of an introduced pathogen. *Mycologia*, 79(1), 23–37. <https://doi.org/10.1080/00275514.1987.12025367>
- Anagnostakis, S. L. (2001). The effect of multiple importations of pests and pathogens on a native tree. *Biological Invasions*, 3(3), 245–254.

- Balkenhol, N., Dudaniec, R. Y., Krutovsky, K. V., Johnson, J. S., Cairns, D. M., Segelbacher, G., ... Joost, S. (2017). Landscape Genomics: Understanding relationships between environmental heterogeneity and genomic characteristics of populations. In O. P. Rajora (Ed.), *Population genomics concepts, strategies and applications* (pp. 261–322). Cham, Switzerland: Springer.
- Bingham, R. T. (1983). Blister rust resistant western white pine for the Inland Empire: The story of the first 25 years of the research and development program. Intermountain Forest and Range Experiment Station: General Technical Report, INT-146.
- Boshier, D., & Buggs, R. J. A. (2015). The potential for field studies and genomic technologies to enhance resistance and resilience of British tree populations to pests and pathogens. *Forestry: An International Journal of Forest Research*, 88(1), 27–40. <https://doi.org/10.1093/forestry/cpu046>
- Budde, K. B., Nielsen, L. R., Ravn, H. P., & Kjær, E. D. (2016). The natural evolutionary potential of tree populations to cope with newly introduced pests and pathogens—Lessons learned from forest health catastrophes in recent decades. *Current Forestry Reports*, 2(1), 18–29. <https://doi.org/10.1007/s40725-016-0029-9>
- Cheng, C., Gao, X., Feng, B., Sheen, J., Shan, L., & He, P. (2013). Plant immune response to pathogens differs with changing temperatures. *Nature Communications*, 4, 2530. <https://doi.org/10.1038/ncomm3530>
- Danchok, R. S., Snieszko, R. A., Long, S., Kegley, A., Savin, D. P., Mayo, J. B., ... Hill, J. (2012). Frequency of hypersensitive-like reaction and stem infections in a large full-sib family of *Pinus monticola*. Proceedings of the 4th international workshop on genetics of host-parasite interactions in Forestry, 281–285.
- D'Arcy, C. J. (2000). *Dutch elm disease* [Online]. St. Paul, MN: The American Phytopathological Society
- Geils, B. W., Hummer, K. E., & Hunt, R. S. (2010). White pines, Ribes, and blister rust: A review and synthesis. *Forest Pathology*, 40(3–4), 147–185.
- Hoff, R. J. (1986). Inheritance of the bark reaction mechanism in *Pinus monticola* infected by *Cronartium ribicola*. USDA Forest Service, Intermount Research Station, Research Note INT-361.
- Hunt, R. S. (1997). Relative value of slow-canker growth and bark reactions as resistance responses to white pine blister rust. *Canadian Journal of Plant Pathology*, 19(4), 352–357. <https://doi.org/10.1080/07060669709501059>
- Hunt, R. S. (2004). Environmental and inoculum-source effects on resistance of Idaho F₂ western white pine in British Columbia. *Forest Pathology*, 26, 351–357. <https://doi.org/10.1080/07060660409507152>
- Hunt, R. S., Murray, M., Reich, R., Rusch, D., Woods, A., & Zeglen, S. (2018). Persistence of major gene resistance in western white pine (*Pinus monticola*) in British Columbia. In: A. W. Schoettle, R. A. Snieszko, & J. T. Kliejunas (Eds.), Proceedings of the IUFRO joint conference: Genetics of five-needle pines, rusts of forest trees, and Strobosphere; 2014 June 15–20; Fort Collins, CO. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station RMRS-P-76, 149–150.
- Johnson, R. (1984). A critical analysis of durable resistance. *Annual Review of Phytopathology*, 22(1), 309–330. <https://doi.org/10.1146/annurev.py.22.090184.001521>
- Jorge, V., Dowkiw, A., Faivre-Rampant, P., & Bastien, C. (2005). Genetic architecture of qualitative and quantitative *Melampsora larici-populina* leaf rust resistance in hybrid poplar: Genetic mapping and QTL detection. *New Phytologist*, 167(1), 113–127. <https://doi.org/10.1111/j.1469-8137.2005.01424.x>
- Kegley, A. J., & Snieszko, R. A. (2004). Variation in blister rust resistance among 226 *Pinus monticola* and 217 *P. lambertiana* seedling families in the Pacific Northwest. In R. A. Snieszko, S. Samman, S. E. Schlarbaum, & H. B. Kriebel (Eds.), *Breeding and genetic resources of five needle pines: Genetics, breeding and adaptability*, Proceedings of the IUFRO 2.02.15 Working Party Conference Medford, OR: USDA Forest Service, Rocky Mountain Research Station RMPS-P-32. Fort Collins, CO, 209–225.
- King, J. N., David, A., Noshad, D., & Smith, J. (2010). A review of genetic approaches to the management of blister rust in white pines. *Forest Pathology*, 40(3–4), 292–313. <https://doi.org/10.1111/j.1439-0329.2010.00659.x>
- Kinloch, B. B. (2003). White pine blister rust in North America: Past and Prognosis. *Phytopathology*, 93(8), 1044–1047. <https://doi.org/10.1094/PHYTO.2003.93.8.1044>
- Kinloch, B. B., Burton, D., Davis, D. A., Westfall, R. D., Dunlap, J., & Vogler, D. R. (2012). Strong partial resistance to white pine blister rust in sugar pine. In: R. A. Snieszko, A. D. Yanchuk, J. T. Kliejunas, K. M. Palmieri, J. M. Alexander, & S. J. Frankel (Eds.), *Proceedings of the fourth international workshop on the genetics of host-parasite interactions in forestry: Disease and insect resistance in forest trees* (pp. 80–91). Albany, CA: Pacific Southwest Research Station, Forest Service, U.S. Department of Agriculture.
- Kinloch, B. B., Davis, D. A., & Burton, D. (2008). Resistance and virulence interactions between two white pine species and blister rust in a 30-year field trial. *Tree Genetics & Genomes*, 4(1), 65–74. <https://doi.org/10.1007/s11295-007-0088-y>
- Kinloch, B. B., & Dupper, G. E. (2002). Genetic specificity in the white pine-blister rust pathosystem. *Phytopathology*, 92(3), 278–280. <https://doi.org/10.1094/PHYTO.2002.92.3.278>
- Kinloch, B. B., Parks, G. K., & Fowler, C. W. (1970). White pine blister rust: Simply inherited resistance in sugar pine. *Science*, 167(3915), 193–195. <https://doi.org/10.1126/science.167.3915.193>
- Kinloch, B. B., Snieszko, R. A., Barnes, G. D., & Greathouse, T. E. (1999). A major gene for resistance to white pine blister rust in western white pine from the western Cascade range. *Phytopathology*, 89(10), 861–867. <https://doi.org/10.1094/PHYTO.1999.89.10.861>
- Kinloch, B. B., Snieszko, R. A., & Dupper, G. E. (2004). Virulence gene distribution and dynamics of the white pine blister rust pathogen in Western North America. *Phytopathology*, 94, 751–758. <https://doi.org/10.1094/PHYTO.2004.94.7.751>
- Kolpak, S. E., Snieszko, R. A., & Kegley, A. J. (2008). Rust infection and survival of 49 *Pinus monticola* families at a field site six years after planting. *Annals of Forest Research*, 51, 67–80.
- La, Y. J. (2009). Korean successes in controlling blister rust of Korean pine. In D. Noshad, E. Noh, J. King, & R. A. Snieszko (Eds.), *Breeding and Genetic Resource of Five-Needle Pines Conference* (pp. 1–9). Yangyan, Republic of Korea: Korea Forest Research Institute.
- Liebold, A. M., Brockerhoff, E. G., Kalisz, S., Nuñez, M. A., Wardle, D. A., & Wingfield, M. J. (2017). Biological invasions in forest ecosystems. *Biological Invasions*, 19(11), 3437–3458. <https://doi.org/10.1007/s10530-017-1458-5>
- Lovett, G. M., Weiss, M., Liebold, A. M., Holmes, T. P., Leung, B., Lambert, K. F., ... Weldy, T. (2016). Nonnative forest insects and pathogens in the United States: Impacts and policy options. *Ecological Applications*, 26(5), 1437–1455. <https://doi.org/10.1890/15-1176>
- McDonald, G. I., Zambino, P. J., & Snieszko, R. A. (2004). Breeding rust-resistant five-needle pines in the western United States: lessons from the past and a look to the future. In R. A. Snieszko, S. Samman, S. E. Schlarbaum, & H. B. Kriebel (Eds.), *Breeding and genetic resources of five-needle pines: Growth, adaptability and pest resistance* (pp. 28–50). Fort Collins, CO: USDA Forest Service, Rocky Mountain Research Station.
- Muchero, W., Sondreli, K. L., Chen, J.-G., Urbanowicz, B. R., Zhang, J., Singan, V., ... LeBoldus, J. M. (2018). Association mapping, transcriptomics, and transient expression identify candidate genes mediating plant-pathogen interactions in a tree. *Proceedings of the National Academy of Sciences*, 115(45), 11573. <https://doi.org/10.1073/pnas.1804428115>

- National Academies of Sciences, Engineering, and Medicine. (2019). *Forest health and biotechnology: Possibilities and considerations*. Washington, DC: The National Academies Press. <https://doi.org/10.17226/25221>
- Nelson, R., Wiesner-Hanks, T., Wisser, R., & Balint-Kurti, P. (2018). Navigating complexity to breed disease-resistant crops. *Nature Reviews Genetics*, 19, 21. <https://doi.org/10.1038/nrg.2017.82>
- Poland, J. A., Balint-Kurti, P. J., Wisser, R. J., Pratt, R. C., & Nelson, R. J. (2009). Shades of gray: The world of quantitative disease resistance. *Trends in Plant Science*, 14(1), 21–29. <https://doi.org/10.1016/j.tplants.2008.10.006>
- Poland, T. M., & McCullough, D. G. (2006). Emerald ash borer: Invasion of the urban forest and the threat to North America's ash resource. *Journal of Forestry*, 104(3), 118–124.
- R Core Team. (2018). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.
- Schoettle, A. W., Jacobi, W. R., Waring, K. M., & Burns, K. S. (2018). Regeneration for resilience framework to support regeneration decisions for species with populations at risk of extirpation by white pine blister rust. *New Forests*. <https://doi.org/10.1007/s11056-018-9679-8>
- Schwandt, J. W., Lockman, I. B., Kliejunas, J. T., & Muir, J. A. (2010). Current health issues and management strategies for white pines in the western United States and Canada. *Forest Pathology*, 40(3–4), 226–250. <https://doi.org/10.1111/j.1439-0329.2010.00656.x>
- Showalter, D. N., Raffa, K. F., Sniezko, R. A., Herms, D. A., Liebhold, A. M., Smith, J. A., & Bonello, P. (2018). Strategic development of tree resistance against forest pathogen and insect invasions in defense-free space. *Frontiers in Ecology and Evolution*, 6(124). <https://doi.org/10.3389/fevo.2018.00124>
- Sniezko, R. A. (2006). Resistance breeding against nonnative pathogens in forest trees—Current successes in North America. *Canadian Journal of Plant Pathology*, 28(sup1), S270–S279. <https://doi.org/10.1080/07060660609507384>
- Sniezko, R. A., Bower, A. D., & Danielson, J. (2000). A comparison of early field results of white pine blister rust resistance in sugar pine and western white pine. *Horttechnology*, 10, 519–522. <https://doi.org/10.21273/HORTTECH.10.3.519>
- Sniezko, R. A., Kegley, A. J., & Danchok, R. (2008). White pine blister rust resistance in North American, Asian and European species—results from artificial inoculation trials in Oregon. *Annals of Forest Research*, 51(1), 53–66.
- Sniezko, R. A., Kinloch, B. B. Jr, Bower, A. D., Danchok, R. S., Linn, J. M., & Kegley, A. J. (2004). Field resistance to *Cronartium ribicola* in full-sib families of *Pinus monticola* in Oregon. In R. A. Sniezko, S. E. Schlarbaum, S. E. Kriebel, & B. Howard (Eds.), *Breeding and genetic resources of five-needle pines: Growth, adaptability and pest resistance; 2001 July 23–27; Medford, OR. IUFRO Working Party 2.02.15. Proceedings RMRS-P-32* (pp. 243–249). Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station.
- Sniezko, R. A., & Koch, J. (2017). Breeding trees resistant to insects and diseases: Putting theory into application. *Biological Invasions*, 19(11), 3377–3400. <https://doi.org/10.1007/s10530-017-1482-5>
- Sniezko, R. A., Smith, J., Liu, J.-J., & Hamelin, R. (2014). Genetic resistance to fusiform rust in southern pines and white pine blister rust in white pines—A contrasting tale of two rust pathosystems—Current status and future prospects. *Forests*, 5(9), 2050. <https://doi.org/10.3390/f5092050>
- Struckmeyer, B. E., & Riker, A. J. (1951). Wound-periderm formation in white pine trees resistant to blister rust. *Phytopathology*, 41, 276–281.
- Telford, A., Cavers, S., Ennos, R. A., & Cottrell, J. E. (2015). Can we protect forests by harnessing variation in resistance to pests and pathogens? *Forestry*, 88(1), 3–12. <https://doi.org/10.1093/forestry/cpu012>
- Vale, F. X. R. D., Parlevliet, J. E., & Zambolim, L. (2001). Concepts in plant resistance. *Fitopatologia Brasileira*, 26, 577–589.
- Wang, Y., Bao, Z., Zhu, Y., & Hua, J. (2009). Analysis of temperature modulation of plant defense against biotrophic microbes. *Molecular Plant-Microbe Interactions*, 22(5), 498–506. <https://doi.org/10.1094/MPMI-22-5-0498>
- Woodcock, P., Cottrell, J. E., Buggs, R. J. A., & Quine, C. P. (2018). Mitigating pest and pathogen impacts using resistant trees: A framework and overview to inform development and deployment in Europe and North America. *Forestry: An International Journal of Forest Research*, 91(1), 1–16. <https://doi.org/10.1093/forestry/cpx031>
- Zeglen, S., Pronos, J., & Merler, H. (2010). Silvicultural management of white pines in western North America. *Forest Pathology*, 40(3–4), 347–368. <https://doi.org/10.1111/j.1439-0329.2010.00662.x>
- Zhang, X. Y., Lu, Q., Sniezko, R. A., Song, R. Q., & Man, G. (2010). Blister rusts in China: Hosts, pathogens, and management. *Forest Pathology*, 40(3–4), 369–381.
- Zhu, Y., Qian, W., & Hua, J. (2010). Temperature modulates plant defense responses through NB-LRR proteins. *PLoS Path.*, 6(4), e1000844. <https://doi.org/10.1371/journal.ppat.1000844>

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