White Pine Blister Rust Resistance of 12 Western White Pine Families at Three Field Sites in the Pacific Northwest

Richard A. Sniezko,[1] Robert Danchok,[1] Jim Hamlin,[2] Angelia Kegley,[1] Sally Long,[1] and James Mayo[1]

Abstract

Western white pine (*Pinus monticola* Douglas ex D. Don) is highly susceptible to the non-native, invasive pathogen *Cronartium ribicola*, the causative agent of white pine blister rust. The susceptibility of western white pine to blister rust has limited its use in restoration and reforestation throughout much of western North America. Fortunately, some genetic resistance to blister rust exists in western white pine, and several operational programs to select for resistance and develop orchards to produce resistant seed exist. These programs, such as that of the U.S. Department of Agriculture, Forest Service (USDA FS) Pacific Northwest Region, utilize short-term artificial inoculation trials of seedlings to evaluate resistance. However, until recently, there have been very few multi-site field trials to examine resistance closely. In this paper, we report on 12 western white pine families common to three field trials in western Oregon and northern California. The sites have been assessed for white pine blister rust infection and mortality at multiple ages since they were planted from 1996 to 2003. The 12 families had been selected based on results in previous artificial inoculation trials of seedlings of more than 4,000 parent trees at Dorena Genetic Resource Center (DGRC). Most of the families were from wind-pollinated seedlots from forest stands and represent some of the top ranked families from a first round of selection. Timing and frequency of field trial assessments varied to coincide with the appearance and development of rust infection at the three sites. By 2010, all three sites had moderate to high levels of blister rust infection, and moderate levels of mortality were present at some sites. The susceptible control family had 92 to 100 percent of trees with stem symptoms at the three sites, while only 29 to 43 percent of the trees in the top resistant family showed stem symptoms in those trials. The high level of stem symptoms in the two families with the Cr2 gene, which conditions a hypersensitive-like reaction (HR) in the needles, suggests that it is likely that a virulent *vcr2* strain of rust is present at all three sites. A wide range in family variation is present at all three sites for the proportion of trees with stem symptoms. Mortality lags behind stem infection, and more time is needed to assess the full impact of current stem infections. Infected trees in families with putative partial resistance traits such as bark reaction noted in previous screening trials will continue to be followed to examine their subsequent survival under field conditions. In general, moderate to high correlations were found between current levels of resistance at the three sites and seedling screening results from DGRC, providing some of the first field validation of results of short-term testing. These current results and future assessments will provide valuable information on the level of rust resistance and survival that land managers can expect over a range of sites. Long-term monitoring of these sites will be essential to evaluate durability of the various resistance types. Ongoing breeding efforts are aimed at increasing the levels of resistance to provide greater opportunities for successful reforestation and restoration use.

Key words: *Pinus monticola*, *Cronartium ribicola*, rust resistance, field trials, operational program

Introduction

Western white pine, *Pinus monticola* Dougl. ex D. Don, a wide ranging forest tree species in western North America, is highly susceptible to the non-native, invasive pathogen *Cronartium ribicola*, the fungus causing white pine blister rust (Geils et al. 2010). Fortunately, some natural genetic resistance

[1] USDA Forest Service, Dorena Genetic Resource Center, Cottage Grove, Oregon, USA.
[2] USDA Forest Service, Umpqua National Forest, Roseburg, Oregon, USA.
Corresponding author: rsniezko@fs.fed.us.

exists in white pine populations (Bingham 1983, Kegley and Sniezko 2004, King et al. 2010, Kinloch et al. 1999, Sniezko 2006). The development of populations of white pine with genetic resistance to white pine blister rust offers an invaluable management tool to restore this species and its utilization in managed forests. In the Pacific Northwest (Oregon and Washington), the U.S. Department of Agriculture, Forest Service (USDA FS) has a regional program at Dorena Genetic Resource Center (DGRC) in Oregon to develop genetically resistant populations of western white pine to blister rust. Resistance is identified through artificial inoculation of 2-year-old seedlings and subsequent assessments, using seed collected from parent trees selected in the forest or from control crosses among selected parents. Thousands of parent trees have been evaluated since the 1960s and several types of complete and partial resistance have been documented in these seedling tests (Kegley and Sniezko 2004; Kinloch et al. 1999, 2003; Sniezko and Kegley 2003a, 2003b; Sniezko 2006).

Western white pine is a long-lived conifer, and in many environments it will be exposed to *C. ribicola* for decades or even hundreds of years. Field testing is essential to validate the results of short-term rust resistance screening by artificial inoculation on very young seedlings and to monitor the durability of blister rust resistance. There are few summaries of field trials that examine white pine blister rust resistance in individual families of western white pine in the Pacific Northwest, and generally these reports encompass only one field site or have a more limited range of resistance types available (Hunt 2002, Kinloch et al. 2008, King et al. 2010, Kolpak et al. 2008; Sniezko et al. 2000, 2004a, 2004b). Until relatively recently, there were few multi-site field trials that closely monitored the field performance of families with the different types of putative resistance observed in artificial inoculation trials of seedlings. However, a series of field trials have been established in the Pacific Northwest since 1996 to examine the effectiveness and durability of resistance under field conditions. In this paper, we examine the incidence of blister rust stem symptoms and mortality in a common set of 12 western white pine families established in three field trials between 1996 and 2003 in western Oregon and northern California. These trials include families from diverse geographic and resistance backgrounds. We also examine the correlation of field performance to seedling test results following artificial inoculation at DGRC.

Materials and Methods

Seed from western white pine parents with varying types and levels of resistance observed in previous seedling artificial inoculation trials at DGRC were used to establish a series of field trials since 1996. We examine the subset of 12 families that are common to three trials in northern California and western Oregon. The seedlots include 11 half-sib families from wind pollination of parent trees in natural stands from a wide range of geographic areas in Oregon and Washington (10 families), or in an orchard at DGRC (one family); the remaining family is a full-sib from a control cross between two parent trees in an orchard at DGRC (table 1). The families were chosen to represent varying levels and types of complete and partial resistance identified in seedling screening, and include a susceptible control (family 4) that showed little or no resistance in previous testing at DGRC. In addition, two of the 12 families (families 11 and 12) have R gene resistance conveyed from the *Cr2* gene, which imparts complete resistance (from a hypersensitive-type reaction in the needles, HR) in the absence of a strain of rust with corresponding virulence gene, *vcr2* (Kinloch et al. 1999, 2003, 2004). The inheritance of HR resistance is due to a single dominant gene and, based on results from a previous artificial inoculation trial at DGRC, these families would be expected to have 75 percent or more of their progeny canker free in the absence of a virulent strain of rust (Sniezko et al. 2004b). The remaining nine families had previously displayed varying types and levels of partial resistance in seedling tests at DGRC (see Kegley and Sniezko 2004, Sniezko et al. 2004a; Sniezko and Kegley 2003a, 2003b). Seedlings with partial resistance often develop stem symptoms, but not to the degree of the most susceptible seedlings. In the seedling trials at DGRC most wind-pollinated families with partial resistance show high levels of stem symptoms (70 to 100 percent of seedlings), but in contrast to the high-susceptible families, partial-resistance families often have fewer stem symptoms per

seedling, more of the stem symptoms appear at a later assessment (latent), more stem symptoms manifest as bark reactions (complete or partial bark reactions) rather than normal cankers, and there is longer survival of seedlings with stem symptoms. More information is needed on the underlying basis of partial resistance(s) and its inheritance. Family 6 displays many of the partial resistant traits, but in the seedling testing it also has a relatively high level of seedlings with no stem symptoms (Sniezko et al. 2004a; Sniezko and Kegley 2003a, 2003b).

Table 1—Family identities, origin, and resistance type for 12 western white pine families: 11 wind-pollinated and one control crossed

Family code	Family	Parent origin	Resistance[a]
1	11053-552 x w	OR	pr
2	03023-509 x w	WA	pr
3	03024-510 x w	WA	pr
4	03024-532 x w	WA	susceptible control
5	03024-793 x w	WA	pr
6	05081-003 x w	WA	high canker free, non-HR
7	06023-521 x w	OR	pr
8	18034-140 x w	OR	pr
9	18035-150 x w	OR	pr
10	21105-052 x w	WA	high bark reaction
11	(15045-816 x 15045-841) x w_{so}	OR	HR
12	15045-823 x 15045-840	OR	HR

w= wind pollinated (in forest stand); w_{so} =wind pollinated in orchard; OR=Oregon, WA=Washington; HR= hypersensitive-like reaction in needles; pr= low levels of partial resistance (Sniezko et al. 2004a).
[a] Based on previous screening trials at Dorena Genetic Resource Center.

Periodic visits to each field site were made, but formal assessments began only after blister rust infection was noted in each trial (figs. 1 and 2). Trees were assessed in multiple years for growth as well as number of stem symptoms (on main bole or branches), types of stem symptoms (incipient infections that appear as small orange circles at the base of some infected needles, normal cankers, bark reactions, partial bark reactions), severity of infection, and mortality. For this paper, we report on family variation, 8 to 11 years after planting (depending on site), only on the proportion of trees (1) with stem symptoms from blister rust and (2) surviving. Analyses of the other traits such as number of stem symptoms are pending. We report on cumulative proportion of stem symptoms, since some stem symptoms present in 1 year may not be seen in some future years (e.g., the tree may outgrow small bark reactions or branches with stem symptoms may self prune).

Happy Camp (HC)

The site is located on the Happy Camp Ranger District on the Klamath National Forest in northern California (N41° 53' 08.51'; W123° 24 09.82', 777 m elevation). This location is the main field test site for sugar pine (*Pinus lambertiana* Dougl.) for the USDA FS, Pacific Southwest Region's blister rust resistance program, but western white pine has also previously been tested on the site (Kinloch et al. 2008). Seedlings from the spring 1995 sowing of 12 sugar pine and 13 western white pine families were planted in April 1996 in a randomized complete block (RCB) design with 12 blocks and up to four trees per family per block in row plots. Trees were planted at 0.60 x 0.75 m spacing. *Ribes sanguineum*, an alternate host to *C. ribicola*, was inter-planted among the pines to augment existing sources of rust spores and to help ensure uniform exposure to rust infection. Assessments of blister rust stem symptoms, mortality, and growth were done annually from 1999 to 2003, and also in 2005 and 2007. Additional background and earlier results are presented elsewhere (Sniezko et al. 2000, 2004a).

Kerbluey (K)

The site is located on the Cottage Grove Ranger District (CGRD), Umpqua National Forest, in southern Oregon (N 43°39.765', W 122° 39.711', 974 m elevation). Western white pine occurs naturally as well as in reforestation areas. *Ribes* species are present in the area, and white pine blister rust had previously been documented in the area. The parents for the two families with HR resistance were selected during the 1950s on CGRD. Seedlings sown in 1995 for the same 25 families used for HC were planted in June 1997 (blocks 1 to 4) and October 1997 (blocks 5 to 8) in a RCB design; a few trees that died in blocks 1 to 4 were replaced in October 1997. In most cases, there were four trees in each family row plot per block (varied from two to five). Eleven families are included in all eight blocks, and one of the 12 families is included only in four blocks. Trees are planted at 3 x 3 m spacing. Assessments of blister rust infection and mortality were done in 1999, 2001 to 2005, and 2008.

Figure 1—Western white pine trial at Coast Plum site in 2010/2011: (a) overview, (b) incipient stem symptom with the 'point-of-entry' needle still alive in the center, (c) expanding canker, (d) older canker with aecia and girdling stem, (e) partial bark reaction, (f) partial bark reaction. (Photo credits: Richard Sniezko (a to e), Robert Danchok (f))

Coast Plum (CP)

The site is located on Plum Creek Corporation land in the Coast Range of Oregon (N 44° 49.966', W 123° 44.790', 533 m elevation). One-year-old seedlings of 28 western white pine families, sown in early March 2002, grown in containers at Plum Creek Corporation's nursery in Cottage Grove, Oregon were planted in early February 2003 in a RCB design with nine blocks. Nine of the 12

families are included in all nine blocks, one family is included in seven blocks, and two families are included in six blocks. Seedlings were planted in family row plots with generally five trees per family plot (varied from three to six trees per family row plot). Trees were planted at 1.8 x 3 m spacing. Blister rust incidence was very low until the first assessment in 2010 (figs. 1 and 2); the trial was also assessed in 2011. There is little or no other known western white pine in the general vicinity, but abundant *R. sanguineum*, an alternate host to *C. ribicola*, was noted in the surrounding areas.

DGRC Seedling Artificial Inoculation Trial (DGRC1995)

All 12 families had previously been evaluated for rust screening in various seedling trials at DGRC, and 10 of the families are included in a common test sown in 1995 (inoculated in 1996). Two-year-old seedlings are used in the artificial inoculation trials, and these seedlings often have primary needles as well as secondary needles. Kegley and Sniezko (2004) provide detailed information on seedling screening trials at DGRC. We use the results from the 1995 test to examine the relationship between short-term seedling screening and field results at the three sites (Sniezko et al. 2004a). The two families with HR resistance are not included in the 1995 seedling test. The three variables used in this paper from the screening trial results include needle lesion mean (NLC), proportion of trees with stem symptoms (SSp), and proportion of trees dead from rust (RMORT) 5 years after inoculation. NLC is based upon number of needle lesions or "spots" on all secondary needles, and ranges from 0 to 4 for individual seedlings. Generally, the scale is setup, after examining a subset of seedlings, to have approximately 25 percent of the seedlings in each needle lesion class; seedlings with no needle lesions are usually rare and are assigned to class 0. The NLC and a preliminary estimate of SSp were used in an earlier summary of early mortality at the Happy Camp site (Sniezko et al. 2004a).

Statistical analysis procedures were conducted using SAS (SAS Institute Inc., V 9.2, 2008) where the trials were analyzed as a RCB. Family differences in survival (and proportion of trees with stem symptoms) were analyzed using family-plot means in Proc Glimmix (binomial distribution); plot means were adjusted by 0.01 for those values of 1.0 (adjusted to 0.99) and 0.0 (adjusted to 0.01) in order to avoid the non-convergence issue in Proc Glimmix when numerous values of 1 or 0 are encountered in the dataset. Site, family, and site x family interaction were considered as fixed effects in the model. Proc Corr (product moment correlation) was used to examine family mean correlations among the respective field trials as well as between these three field trials and the 1995 seedling screening trial at DGRC.

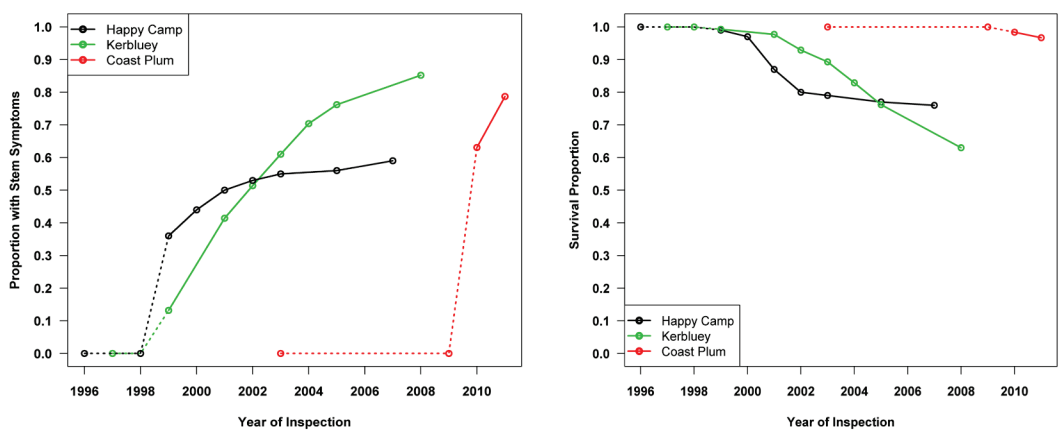

Figure 2—Time trend for proportion of white pines with (a) stem symptoms and (b) surviving, at three sites: Happy Camp (planted 1996), Kerbluey (planted 1997), and Coast Plum (planted 2003).

Results

A moderate (0.60 at HC) to high proportion (0.78 at CP and 0.84 at K) of trees displayed stem symptoms (SSp) by the latest assessment (fig. 2; table 2) at the three sites. The temporal dynamics of blister rust infection varied among the three sites. For the 1996 HC trial, over 35 percent of trees had stem symptoms by 1999, but there has been only a gradual increase in infection through 2007; for the 1997 K planting, over 35 percent of trees had stem symptoms by 2001 and there has been a continued steady increase in infection through the last assessment (2008); for the 2003 CP planting, over 60 percent of trees had infection by the first assessment in 2010 and a further jump in infection was recorded in 2011 (fig. 2). The severity codes associated with the first assessments at each site indicated that only 7 (of 311) trees at K, and 14 (of 556) trees at HC had larger and presumably older active cankers at their first assessments, while 105 trees (of 465) at CP had relatively large active cankers at that site's first assessment (fig. 1). Most of the symptoms noted at CP in 2010 were on the 2007 and 2008 portions of the main stem or branches. At their first assessment, 2, 6, and 8 trees were dead from blister rust at K, HC, and CP, respectively. The number of stem symptoms per living tree varied by site and year from a maximum of 0.60 at HC to 7.18 at K, and 9.53 at CP (table 3).

Table 2—Overall site means (and range in variation among the 12 families) for proportion of trees with blister rust stem symptoms and survival at three field sites

Site	Stem symptom	Survival
Coast Plum (CP)	.78 (.37 to 1.0)	.95 (.90 to 1.0)
Kerbluey (K)	.84 (.43 to 1.0)	.64 (.19 to .94)
Happy Camp (HC)	.60 (.29 to. 92)	.75 (.43 to 1.0)

Highly significant differences ($p < .0001$) existed among sites and families in the inter-site analysis for SSp. In addition, there was a significant site x family interaction ($p = .0001$). This interaction appears to be influenced by the relatively low proportion of trees in families 11 and 12 with stem symptoms at HC relative to that at CP and K (fig. 3). When these two families with HR resistance are excluded from the inter-site analysis, the site x family interaction is not significant for SSp ($p = .056$). A wide range of family variation in SSp was evident at all three sites (table 2, fig. 3).

Table 3—Number of stem symptoms (SS) per living tree at several assessments for three western white pine blister rust resistance trials

Site	Year	Number of living trees	Mean number of stem symptoms (SS) per living tree	Range in SS per tree
Happy Camp	1999	551	0.60	0 to 9
Happy Camp	2003	438	0.15*	0 to 4
Kerbluey	2001	305	1.08	0 to 11
Kerbluey	2005	244	2.39	0 to 20
Kerbluey	2008	192	7.18	0 to 48
Coast Plum	2010	457	4.95	0 to 91
Coast Plum	2011	449	9.53	0 to 80

Happy Camp planted in 1996; Kerbluey in 1997; Coast Plum in 2002.
*Decrease in SS at HC reflects mortality of many of the trees with early stem symptoms.

SSp for the susceptible control (family 4) was very high (0.92 to 1.0) at all sites (fig. 3; table 2). At HC, the difference in SSp between the susceptible family and the other 11 families was particularly notable (fig. 3). At K, four families showed 100 percent stem symptoms, including the susceptible control (family 4), the two families with HR resistance (families 11 and 12), and family 2. Family 2 was the only one to have 100 percent stem symptoms at CP, which was slightly higher than the 98 percent for the susceptible control (fig. 3). Family 6 had the lowest proportion of trees with stem

symptoms (0.29 to 0.43) at all three sites (fig. 3). At K, the two families (11 and 12) with HR resistance showed delayed, but then rapid, steady increase in infection over time (fig. 4).

Despite the moderate to high level of stem infection, the proportion of trees surviving (SURV) was moderate (0.64 at K, 0.75 at HC) to high (0.95 at CP) at the three sites (table 2, fig. 3), with family survival varying from 0.19 to 1.0 (table 2, fig. 3). Through the latest assessments, mortality has just begun at CP, it has nearly plateaued at HC, and it appears to be steadily increasing at K (fig. 2). There were significant differences among both sites and families ($p < .0001$), but the site x family interaction for SURV was not significant ($p=.50$). The susceptible control (family 4) has the lowest survival at all sites (fig. 3). Family 6 had the highest survival at CP and K, and second highest at survival HC (fig. 3).

Survival of the 12 families was significantly correlated among the three sites (0.64 to 0.81), but for SSp, only the family mean correlation between CP and K (0.92) was significant (table 4). SSp at HC

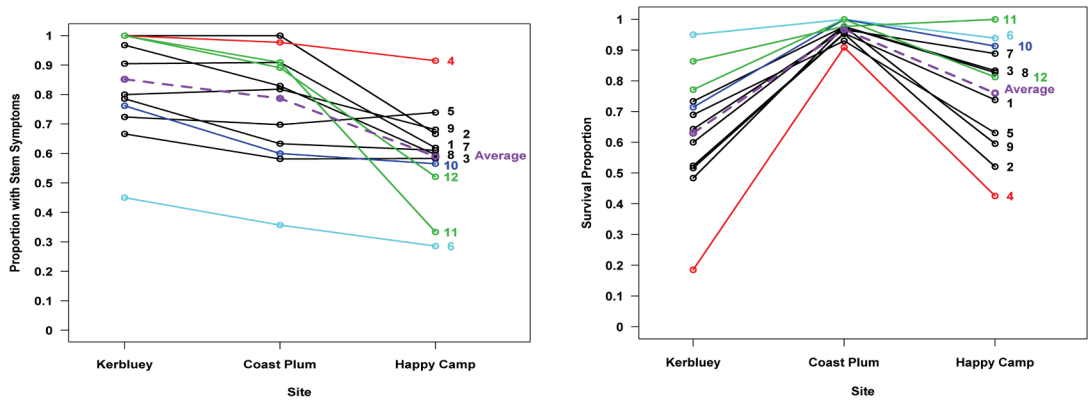

Figure 3—Family means at last assessment over three sites for 12 families for (a) proportion of trees with stem symptoms and (b) proportion of trees surviving.

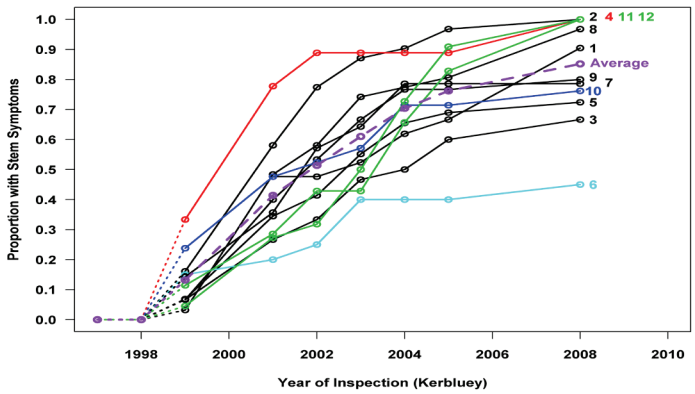

Figure 4—Time trend for 12 western white pine families for 1997 Kerbluey planting for proportion of trees with stem symptoms from blister rust infection (first assessment in 1999).

was significantly correlated with survival at all three sites (-0.78 to -0.88); there was a moderate family mean correlation of SSp at CP with survival at both K (- 0.59, $p = .04$) and HC (-0.56, $p = .06$); there was a moderate, but non-significant correlation of SSp and survival at K (-0.51, $p = .09$ (table 3, fig. 4)). At K, the majority of the stem symptoms on families 11 and 12, the two families with HR resistance, appear much later than other families, but reached 100 percent by 2008 (fig. 4). When families 11 and 12 are excluded (n=10), many of the family mean correlations for SURV and SSp were higher and additional ones were significant (14 of the 15 correlations ranged from 0.59 to 0.92, with only SSp at K with Surv at CP being somewhat lower (-0.40)).

For the 10 families included in the DGRC artificial inoculation trial, there were significant moderate- to high-family mean correlations (0.60 to 0.87) of SSp and RMORT at DGRC with both SSp and SURV at each of the three field trials (table 5, fig. 5). The correlations of seedling results at DGRC (SSp and RMORT) were slightly higher for SSp (0.82 to 0.87) at the field sites than for SURV (-0.60 to -0.78) (table 4). The correlations of seedling needle lesion class (NLC_DGRC) with SSp and SURV in the field were only low to moderate, and were non-significant (table 5).

Table 4— Family mean correlations (p-values) between three field trials for proportion of trees with white pine blister rust stem symptoms (SSp) and survival (Surv)

	Surv_CP	SSp_K	Surv_K	SSp_HC	Surv_HC
SSp_CP	-0.42 (0.178)	0.92 (0.0001)	-0.59 (0.044)	0.45 (0.138)	-0.56 (0.059)
Surv_CP		-0.19 (0.546)	0.64 (0.025)	-0.78 (0.0028)	0.76 (0.004)
SSp_K			-0.51 (0.088)	0.34 (0.271)	-0.31 (0.329)
Surv_K				-0.88 (0.0001)	0.81 (0.0015)
SSp_HC					-0.85 (0.0005)

Based on 12 families.
HC, K, and CP field trials assessed through 2007, 2008, and 2011, respectively.

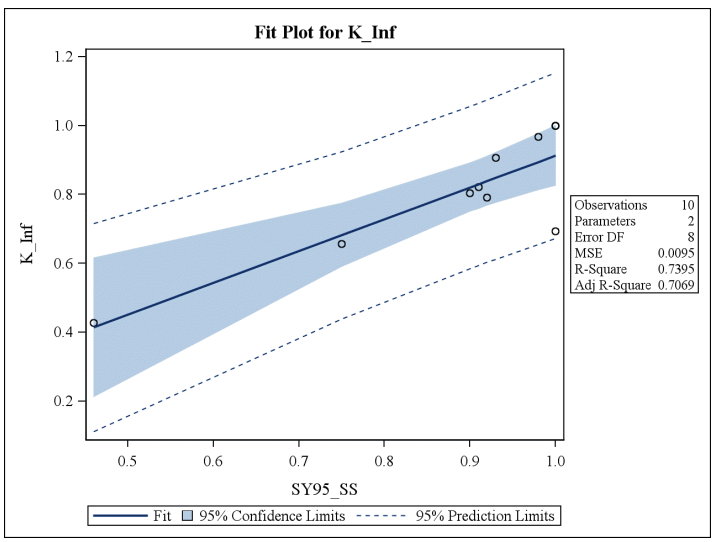

Figure 5—Relationship between proportion of seedlings with stem symptoms in artificial inoculation trial at DGRC (SY95_SS) and proportion of trees with stem symptoms at Kerbluey field trial (K_Inf) for 10 western white pine families (excludes two families with HR resistance).

Table 5—Family mean correlations (p-values) between results from the 1995 artificial inoculation test of seedlings at DGRC and proportion of trees with white pine blister rust stem symptoms (SSp) and survival (Surv) at three field trials

	SSp_CP	Surv_CP	SSp_K	Surv_K	SSp_HC	Surv_HC
NLC_DGRC	0.34 (.338)	-0.33 (.346)	0.46 (.185)	-0.59 (.074)	0.48 (.157)	-0.35 (.318)
SSp_DGRC	0.82 (.004)	-0.61 (.059)	0.86 (.001)	-0.72 (.019)	0.84 (.002)	-0.60 (.067)
MORT_DGRC	0.87 (.001)	-0.72 (.018)	0.83 (.003)	-0.78 (.008)	0.84 (.002)	-0.68 (.029)

10 families included (excludes the two families with HR resistance, refer to text for details).

NLC = needle lesion rating after artificial inoculation (see text for details), MORT=mortality proportion 5 years after inoculation.

Discussion

As evidenced by the high proportion of stem symptoms on the susceptible control (family 4), all three sites have had moderate to heavy exposure to blister rust infection. The incidence of rust at the Coast Plum 2003 planting was low until the first assessment in 2010. From the early data, CP would have appeared to be a low rust hazard, but the abundance of *Ribes* spp., the alternate host of blister rust, and favorable climate eventually led to a large jump in stem symptoms by 2010 and again in 2011. The rust hazard of this site likely increased since 2003 as tree harvest operations in the surrounding areas removed the forest canopy, permitting more abundant natural regeneration of *Ribes*, the alternate host. The levels of infection and family variation in proportion of trees with stem symptoms in these trials appears to be somewhat comparable to results from other field trials with mostly different families (at Happy Camp and in Idaho) discussed by Kinloch et al. (2008), in British Columbia (Hunt 2002), and somewhat lower than the 0.93 (six years after planting) for another trial on CGRD (Kolpak et al. 2008). It will be important to monitor these sites over time since western white pine is a long-lived tree species and exposure to blister rust infection will continue to occur at periodic intervals.

There are large differences in blister rust resistance among the 12 families tested. In the absence of the *vcr2* strain of rust, families 11 and 12, with HR resistance, were expected to show little or no incidence of stem symptoms. The moderate to high incidence of stem symptoms on families 11 and 12 confirms the presence of the *vcr2* strain of rust at all three sites. Several other trials on CGRD also show a very high incidence of stem symptoms on families with HR resistance (Kolpak et al. 2008, Sniezko et al. 2004b). Previous monitoring has indicated that a low to high frequency of this strain of rust is present in much of western Oregon, as well as at HC (Kinloch et al. 2004). At two of the three sites, K and CP, the proportion of trees with stem symptoms for these two families is now very similar to that of the susceptible control. At the third site, HC, the level of stem symptoms in these two families has increased since the last report (Sniezko et al. 2004a). At this stage, the survival of the two families with HR resistance is relatively high, but this may be at least partially a function of the more recent infection at CP and the relatively recent increase in incidence of *vcr2* at K. Future monitoring of these families at these sites will indicate whether they have any resistance beyond HR. The airborne spread of rust spores and the current geographic distribution of *vcr2* will limit the utility of HR resistance in many areas of western Oregon. At some sites where *vcr2* is not yet present, HR resistance may offer utility in delaying stem infections until trees are larger which may benefit overall survival. Combining HR resistance with partial resistances would offer an attractive option and has been implemented in the design of current seed orchards.

The other nine families are thought to have varying levels of partial resistance. Two of these, families 6 (05081-003 x w) and 10 (21105-052 x w), have been tested many times and have repeatedly shown moderate to high resistance in artificial inoculation trials of seedlings at DGRC (Kegley and Sniezko 2004; Sniezko and Kegley 2003a, 2003b). In the DGRC trials, these two families have been among the most outstanding for partial resistances. These two wind-pollinated families are probably in the top 1 percent for partial resistance of the thousands of forest selections evaluated in seedling testing. In these three field trials, these two families are also among the top for rust resistance. In seedling testing at DGRC, these two families show a higher incidence of seedlings free of stem symptoms and seedlings with bark reactions than do many other families (Kegley and Sniezko 2004; Sniezko and Kegley 2003a, 2003b). They also show comparable or higher levels of resistance in rust screening at DGRC to the full-sib checklots from the Interior West rust resistance program based in Idaho (see Table 5 in Kegley and Sniezko 2004) and higher resistance in the field at the CP site than the full-sib seedlots from the British Columbia program (Sniezko et al. 2010). Progeny of these parents have been grafted into orchards, and clone banks, pollinations made with them, and other clones will help increase resistance and help us understand more about inheritance of some of the mechanisms involved.

The significant correlations among some resistance traits at DGRC and those in the field are encouraging, and validate short-term testing. Only a low, non-significant correlation of the needle spot class trait (at DGRC) with the proportion of trees infected or dead was found in this study; a somewhat similar result has been found elsewhere (Hunt 2002). However, in another study, needle spot class correlated with number of stem symptoms in the field (Kolpak et al. 2008). Data analyses for the relationship between needle lesion mean (at DGRC) and number of stem symptoms at these three field sites has not yet been done. A significant amount of additional data to examine this relationship will also be available in the near future as assessments on many other field trials are completed and analyzed.

Continued observations from these field trials will help confirm the type of resistances and their effectiveness on larger trees under repeated exposure to *C. ribicola* infection. Of particular interest will be what level of bark reaction in these families is manifested in the field and whether it contributes significantly to survival. Some further discussion of possible resistances and their inheritance in western white pine is reported for an older trial at HC (Kinloch et al. 2008) and from the early results from another trial on CGRD (Kolpak et al. 2008). However, more research is needed into the underlying mechanisms and the inheritance of partial resistance.

Mortality from blister rust usually lags at least several years behind stem infection, so future assessments will be needed to provide a more definitive indication of the full impact of blister rust infection. Most infections at CP were observed for the first time in 2010 or 2011, 7 and 8 years after planting, and it is expected to be several years before these larger, older trees show much mortality. Coast Plum showed a relatively large jump in stem symptoms from 2010 to 2011, probably due to some latent infections not being visible during the February 2010 assessment on trees with partial resistance as well as new infections from what now appears to be a fairly high-hazard site for rust. The incidence of stem symptoms and mortality at HC has been relatively static between 2002 and 2007, and many of the previous infections appear to be inactive. At CP and K, the incidence of stem symptoms has been increasing and a commensurate increase in mortality may occur depending on the effectiveness of the partial resistances.

Somewhat surprisingly, blister rust mortality is only low to moderate at HC and K. The susceptible control shows the highest mortality of the 12 families, but only at one of the three sites is its mortality greater than 60 percent. The effectiveness of partial resistance may depend on several factors, including the size and age of the seedlings when infection occurs and the rust hazard of the site. In the three trials reported here, there is a moderate to high level proportion of trees with stem symptoms and a low to moderate number of stem symptoms per tree, but most of the symptoms occur several years or more after planting. On an extreme high-hazard site, there may be high incidence of infection events and a much higher number of stem infections per tree. Experience with 2-year-old seedlings in artificial inoculation trials at DGRC shows much higher mortality in most families, usually within 2 to 4 years of appearance of stem symptoms. Small seedlings in the field that are repeatedly infected or trees with dozens to hundreds of stem symptoms may not survive. Rating sites for rust hazard is still problematic in many cases, and more information is needed to provide land managers rust hazard ratings for lands being considered for planting western white pine.

These trials are the first tests in the Pacific Northwest to closely follow field performance of specific families at multiple sites. Results from short-term screening using artificial inoculation of 2-year-old seedlings correlates well with field performance at these three sites. However, the field sites need to be monitored over the long term to evaluate the impacts of continued new exposures to infection by *C. ribicola*, as well as the subsequent impact from existing stem symptoms. Additional field trials have been established in Oregon and Washington, including some with control crossed progenies as well as seedlots from the Interior West and British Columbia blister rust resistance programs. This set of trials will provide baseline information on the utility of current levels of blister rust resistance under a wide range of site conditions and the adaptability of the species under changing climatic conditions. The data from these trials will provide key information to allow land

managers to utilize resistant western white pine. Long-term monitoring of these sites will be essential to evaluate durability of the various resistance types.

Most of the families represented here are wind-pollinated progenies from original field selections. The top parents or some of their progeny surviving rust testing at DGRC have been grafted into orchards managed by several groups, including the USDA FS; U.S. Department of the Interior, Bureau of Land Management; Washington Department of Natural Resources; Quinault Indian Nation; and British Columbia Ministry of Forests. Many of these orchards for the breeding zones in the Pacific Northwest have reached reproductive age and bulked seedlots to use in reforestation and restoration have been collected and are in use for some of them. Some of these orchard seedlots are now in testing at DGRC and in field trials. The level of resistance expected from the first cycle of orchards developed will depend on several factors, but will be less than that of the top families in these trials. Control crossing between orchard selections is ongoing. It is anticipated that the breeding and subsequent selections from artificial inoculation trials will supply higher levels of resistance from advanced-generation orchards. These anticipated levels and combinations of resistances in future orchards should provide land managers with higher levels of success in restoration and reforestation plantings.

Acknowledgments

The authors thank the USDA Forest Service for funding and personnel for the many phases of this work. We thank Jim Smith and Plum Creek Corporation for establishment of Coast Plum site, and Jerry Hill for his work on all these trials; and Douglas Savin for providing some of the graphs. We also acknowledge the help of Deems Burton and Dean Davis with many aspects of the Happy Camp trial. We thank Holly Kearns, Amy Kroll, Michael Murray, and Tom DeSpain for their reviews of an earlier version of this paper.

Literature Cited

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. Gen. Tech. Rep. INT-146. Ogden, UT: U.S. Department of Agriculture, Forest Service, Intermountain Forest and Range Experiment Station. 45 p.

Geils, B.W.; Hummer, K.E.; Hunt, R.S. 2010. White pines, *Ribes*, and blister rust; a review and synthesis. Forest Pathology. 40: 147–185.

Hunt, R.S. 2002. Relationship between early family-selection traits and natural blister rust cankering in western white pine families. Canadian Journal of Plant Pathology. 24: 200–204.

Kegley, A.; Sniezko, R.A. 2004. Variation in blister rust resistance among 226 *Pinus monticola* and 217 *P. lambertiana* seedling families in the Pacific Northwest. In: Sniezko, R.A.; Samman, S.; Schlarbaum, S.E.; Kriebel, H.B., eds. Breeding and genetic resources of five-needle pines: growth, adaptability, and pest resistance; IUFRO Working Party 2.02.15. Proceedings RMRS-P-32. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station: 209–226. http://www.fs.fed.us/rm/pubs/rmrs_p032/rmrs_p032_209_226.pdf. (14 January 2012).

King, J.N.; Noshad, D.A.; Smith, J. 2010. A review of genetic approaches to the management of blister rust in white pines. Forest Pathology. 40: 292–313.

Kinloch, B.B., Jr.; 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 and Genomes. 4: 65–74.

Kinloch, B.B., Jr.; Sniezko, 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.

Kinloch, B.B., Jr.; Sniezko, R.A.; Dupper, G.E. 2003. Origin and distribution of Cr2, a gene for resistance to white pine blister rust in natural populations of western white pine. Phytopathology. 93(6): 691–694.

Kinloch, B.B., Jr.; Sniezko, R.A.; Dupper, G.E. 2004. Virulence gene distribution and dynamics of the white pine blister rust pathogen in western North America. Phytopathology. 94(7): 751–758.

Kolpak, S.E.; Sniezko, R.A.; Kegley, A.J. 2008. Rust infection and survival of 49 *Pinus monticola* families at a field site six years after planting. In: Blada, I.; King, J.; Sniezko, R., eds., 2008: Proceedings of the IUFRO Working Party 2.02.15 Breeding and genetic resources of five-needle pines conference. Annals of Forest Research. 51: 67–80. http://www.editurasilvica.ro/afr/51/1/kolpak_1.pdf. (19 January 2012).

Sniezko, R.A. 2006. Resistance breeding against nonnative pathogens in forest trees — current successes in North America. Canadian Journal of Plant Pathology. 28: S270–S279.

Sniezko, R.A.; Bower, A.; Danielson, J. 2000. A comparison of early field results of white pine blister rust resistance of sugar pine and western white pine. HortTechnology. 10 (3): 519–522.

Sniezko, R.A.; Bower, A.; Kegley, A. 2004a. Variation in *Cronartium ribicola* field resistance among *Pinus monticola* and 12 *P. lambertiana* families: early results from Happy Camp. In: Sniezko, R.A.; Samman, S.; Schlarbaum, S.E.; Kriebel, H.B., eds. Breeding and genetic resources of five-needle pines: growth, adaptability, and pest resistance; IUFRO Working Party 2.02.15. Proceedings RMRS-P-32. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station: 203–208. http://www.fs.fed.us/rm/pubs/rmrs_p032/rmrs_p032_203_208.pdf. (15 January 2012).

Sniezko, R.A.; Hill, J.; Danchok, R.; Kegley, A.; Long, S.; Mayo, J.; Smith, J. 2010. White pine blister rust resistance in a seven year old field trial of 28 western white pine (*Pinus monticola*) families in the Coast Range of Oregon. [Poster]. In: American phytopathology society meeting; 2010 August, Charlotte, NC.

Sniezko, R.A.; Kegley, A. 2003a. Blister rust resistance experiences in Oregon and Washington: evolving perspectives. In: Stone J.; Maffei, H., comps. Proceedings of the 50th Western International Forest Disease Work Conference. Bend, OR: U.S. Department of Agriculture, Forest Service, Central Oregon Service Center: 111–117. http://www.fs.usda.gov/Internet/FSE_DOCUMENTS/stelprdb5280665.pdf .(15 January 2012).

Sniezko, R.A.;Kegley, A.J. 2003b. Blister rust resistance of five-needle pines in Oregon and Washington. In: Xu, M.; Walla, J.; Zhao, W., eds. Proceedings of the second IUFRO rusts of forest trees working party conference. Forest Research. 16 (Suppl.): 101–112. http://www.fs.usda.gov/Internet/FSE_DOCUMENTS/stelprdb5280720.pdf. (15 January 2012).

Sniezko, R.A.; Kinloch, B.B., Jr.; Bower, A.D.; Danchok, R.S.; Linn, J.M.; Kegley, A.J. 2004b. Field resistance to *Cronartium ribicola* in full-sib families of *Pinus monticola* in Oregon. In: Sniezko, R.A.; Samman, S.; Schlarbaum, S.E.; Kriebel, H.B., eds. Breeding and genetic resources of five-needle pines: growth, adaptability, and pest resistance; IUFRO Working Party 2.02.15. Proceedings RMRS-P-32. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station: 243–249. http://www.fs.fed.us/rm/pubs/rmrs_p032/rmrs_p032_243_249.pdf. (15 January 2012).