

STATUS OF WHITE PINE BLISTER RUST IN THE INTERMOUNTAIN WEST

Jonathan P. Smith¹ and James T. Hoffman²

ABSTRACT.—During 1995–1997 we conducted a white pine blister rust (WPBR) disease survey in white pines of the Intermountain West. Incidence of WPBR in white pines was 59% overall, 73% in the northern Rocky Mountains, 55% in the middle Rocky Mountains, and 67% in the Sierra Nevada sample stands. Intensity within infected stands averaged 35% and ranged from 2% to 100%. Southward spread of the disease along the western slopes of the Rocky Mountains appears to have slowed or stopped, and the disease was found at the northern and western edges of, but not within, the Great Basin region. Smaller-diameter trees infected with WPBR sustained more severe damage than larger-diameter trees. Mortality and top kill caused by WPBR were very low across the entire study area, but incidence and intensity of the disease appear to have increased substantially in the northern and middle Rocky Mountains since the 1960s.

Key words: white pine, white pine blister rust, *Cronartium ribicola*, tree diseases, Great Basin forests, Rocky Mountain forests, subalpine forests.

Most, if not all, white pines (genus *Pinus* L., subgenus *Strobus* Lemm., section *Strobus* subsections *Cembrae* Loud. and *Strobi* Loud., and section *Parrya* Mayr subsection *Balfouriana* Engelm.) are susceptible to white pine blister rust disease (WPBR) caused by the introduced fungus *Cronartium ribicola* J.C. Fisch. ex Rabenh (Hoff et al. 1980, Keane and Arno 1993). *Cronartium ribicola* causes only minimal damage to its primary *Ribes* spp. (ribes) host but can produce cankers that girdle and kill its alternate white pine host, or destroy the reproductive potential of white pines by killing the uppermost, cone-bearing branches (Keane et al. 1994, Krebill and Hoff 1995).

Within 30 yr of its 1910 introduction into Vancouver, British Columbia, the fungus had spread throughout most of the range of mid-elevation white pine forests, which contain *Pinus monticola* Dougl. ex D. Don. (western white pine) and *P. lambertiana* Dougl. (sugar pine; Mielke 1943). By 1960 it had spread throughout much of the range of the subalpine white pine species, *P. albicaulis* Engelm. (whitebark pine), concentrated in areas where its distribution coincides with that of *P. lambertiana* and *P. monticola* (Hoff et al. 1994).

Recent studies have shown that the WPBR epidemic is devastating *P. albicaulis* in the northern Rocky Mountains (Keane and Arno

1993, Keane and Morgan 1994, Kendall et al. 1996). In western Montana, for example, Keane et al. (1994) reported that of a sample of 2503 *P. albicaulis* trees, 83% were infected with WPBR. Estimates of *P. albicaulis* mortality are as high as 90% for portions of the Selkirk Range in northern Idaho and for the east side of Glacier National Park (Kendall and Arno 1990).

Less is known about the status of WPBR in the area we refer to here as the Intermountain West. A formal WPBR survey has not been conducted in this area since 1967 (Brown and Graham 1969). From that survey and other recorded observations, it appears that the disease was present at low levels throughout much of the area in the late 1960s (Krebill 1964, Brown and Graham 1969).

There is a growing body of evidence to suggest that WPBR has the capacity to intensify in subalpine white pine forests and spread to new, uninfected areas in the western United States. Results of a recent study in Grand Teton National Park suggest that WPBR incidence is increasing in *P. albicaulis* and *P. flexilis* of the middle Rocky Mountain region (Kendall et al. 1996). Relatively recent infections have been reported in southeastern Wyoming (Brown 1978) and South Dakota (Lundquist and Geils 1992), and in 1990 WPBR was discovered in *P. strobiformis* Engelm. (southwestern white pine)

¹Northern Arizona University, School of Forestry, Box 15018, Flagstaff, AZ 86011.

²USDA Forest Service, Forest Health Protection, 1249 S. Vinnell Way, Boise, ID 83709.

in the Sacramento and adjoining White Mountains of New Mexico (Hawksworth 1990, Conklin 1994). The New Mexico infection center lies over 900 km away from any other known WPBR infections. It is not known whether the disease spread to this location via a corridor of infected pines and *Ribes*, by long-distance spore dispersal, or by the accidental introduction of infected nursery stock (Conklin 1994).

This recent intensification and spread of WPBR prompted us to question whether the disease would become better established in the Intermountain West. Thus, a disease survey was conducted in 1995–1997 to document WPBR epidemic characteristics (incidence, intensity, damage, and mortality) in the Intermountain West, and to use these characteristics to investigate whether WPBR has increased, intensified, or spread since the 1960s.

METHODS

Study Area

The study area encompasses the Great Basin physiographic province and adjoining areas of the Colorado Plateau and Sierra Nevada, and portions of the middle Rocky Mountain and northern Rocky Mountain provinces (Fennemans 1931). We surveyed the portion of the middle Rocky Mountains that lies south of the Yellowstone Plateau and the portion of the northern Rocky Mountains that lies south of the westward course of the Salmon River at approximately 45°N latitude (Fig. 1). Physiographic regions were subdivided into sections based on geology, geomorphology, and climate (Steele et al. 1981, 1983).

Throughout the study area the white pine species, *P. albicaulis*, *P. flexilis*, *P. monticola*, and *P. longaeva* D.K. Bailey [= *P. aristata* var. *longaeva* (D.K. Bailey) Little] (Great Basin bristlecone pine), occur in high-elevation subalpine forests up to the highest elevations of tree growth at the boundary with the alpine zone. *Pinus flexilis*, however, has the unique capability of occupying lower, dry treeline sites as well as upper, cold treeline sites (Arno and Hammerly 1984), especially in the northern and eastern portions of the study area. Along the western boundary of the Great Basin and Sierra Nevada provinces, *P. flexilis* is less common at lower treeline, but *P. lambertiana* occasionally occupies mid-elevation forests. *Pinus monticola* occurs in subalpine

forests in the Sierra Nevada and, therefore, is referred to here as a subalpine white pine species. In the Rocky Mountains *P. monticola* tends to occupy mid-elevation forests but does not grow south of the Salmon River in Idaho, the approximate northern boundary of the study area.

Survey Procedures

During the summers from 1995 through 1997, we inspected white pines for WPBR in 100 subalpine locations throughout the study area. Sample locations were randomly chosen from a list of areas identified by local forest managers as having white pine species present. In each white pine area, the 1st patch, or stand, of trees encountered that appeared to have at least 50 white pines >1.37 m (4.5 ft) tall was sampled. During the 1995 field season we installed 10 rectangular plots according to methods specified by the Whitebark Pine Monitoring Network (Kendall 1995). For the 1996 and 1997 field seasons, 90 strip transects were used to delineate sample trees. We switched to transects because white pine species in the Intermountain West tend to grow as dispersed woodlands or as infrequent seral components in subalpine forests. Obtaining 50 white pines in a rectangular plot of a reasonable size was often impossible. Once established in the stand, both rectangular plots and strip transects precluded a biased tree selection by imposing bounds on which trees were inspected. Location criteria and data collection procedures were identical for plots and transects, and so the data were combined for our analysis.

Strip transects were 4.6 m wide and oriented along the contour of the slope or perpendicular to the contour from an arbitrary point on the edge of the stand. We traversed the transect until at least 50 white pines (at least 30 live or recently killed trees) >1.37 m tall had been inspected, or until we reached the edge of the stand. In open woodland stands, or where white pines were minor seral components, the edge of the stand was defined as a change in habitat type or phase (Steele et al. 1981, 1983), horizontal canopy structure, aspect (>10°), slope (>10%), elevation (>100 m), or topographic position. If the edge of the stand was encountered before 50 trees had been inspected, a 2nd segment of the transect was initiated 2.3 m to the left or right of the

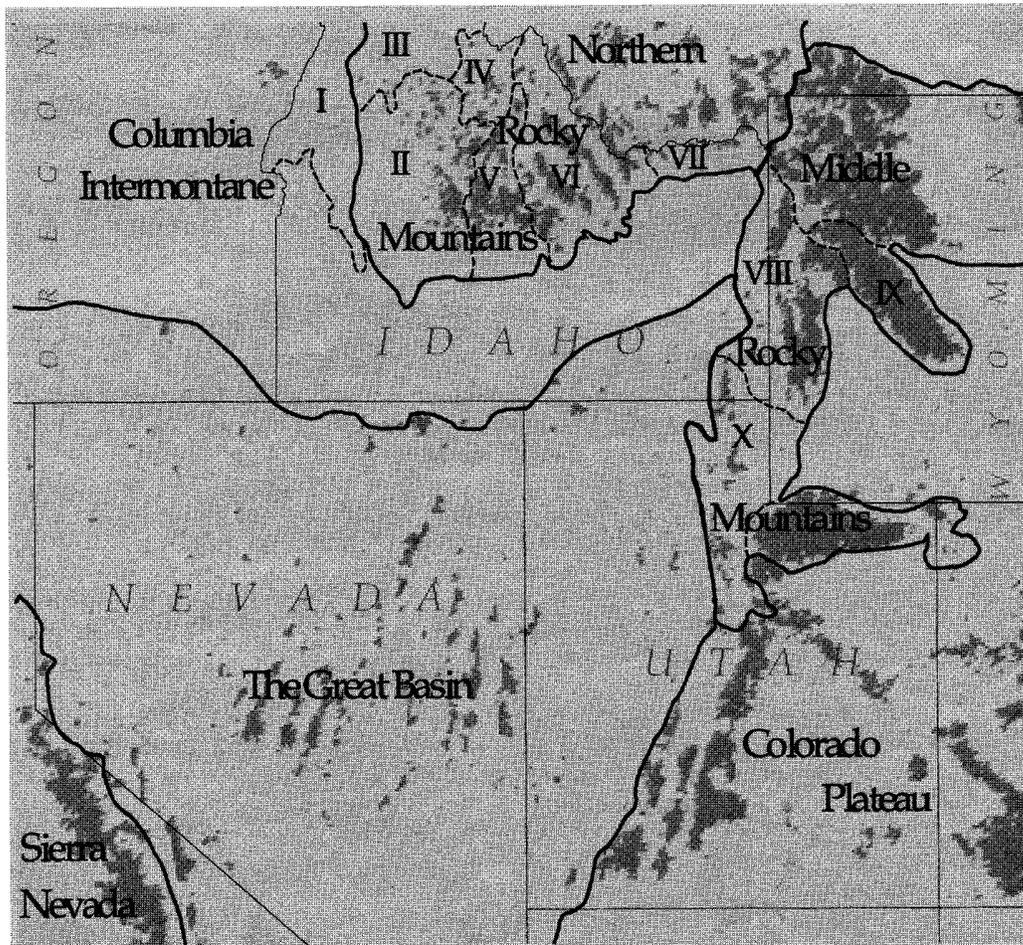


Fig. 1. Physiographic regions and sections of the Intermountain West adapted from Steele et al. (1981, 1983). Sections of the northern and middle Rocky Mountains: I, Wallowa–Seven Devils; II, Idaho Batholith; III, western Salmon Uplands; IV, eastern Salmon Uplands; V, Challis; VI, open Rocky Mountains; VII, mountains of the upper Snake River headwaters; VIII, Wyoimide Ranges; IX, Wind River; X, Wasatch. Shaded areas are elevations >2440 m above sea level.

1st segment (toward the center of the stand) and traversed in the opposite direction of the 1st segment. Thus, the left edge of the 2nd segment corresponded with the left edge of the 1st segment, but care was taken not to inspect the same tree twice. Sampling continued in this manner until 50 trees had been inspected.

Using binoculars for foliar and stem disease signs and symptoms, we inspected each white pine within the strip transect or plot boundaries. Tree diameter at breast height (DBH) was measured at 1.37 m aboveground in 5-cm-diameter classes. Stems that forked below 1.37 m were considered individual trees. For infected

trees the distance from the main stem to the closest (most proximal) branch canker was estimated and used to assign each tree to 1 of 5 damage classes.

An additional 27 transects were established where white pines were encountered en route to predetermined sample areas. Most of these incidental samples were located in lower tree-line rather than subalpine white pine stands. We treated them separately because they were often located near roads or trails and usually did not meet the sampling criterion of at least 50 trees. Sampling methods were identical to the methods described above except that damage and mortality data were not collected.

Since 11 of these samples contained <30 trees, and 8 of those had <15 trees, intensity estimates are probably less accurate than the transect and plot estimates. Nevertheless, data from these samples were deemed useful for describing the geographic extent of WPBR throughout the study area and for generating hypotheses about the spread and intensification of the disease. We clearly distinguish how these supplemental data are used throughout the paper.

Analyses

Our definitions of incidence, intensity, and damage are as follows: WPBR *incidence* refers to presence or absence of WPBR in a sample stand. *Intensity* is the percentage of live trees in infected sample stands that were infected with WPBR. Incidence and intensity were calculated only for live trees, and since most stands had at least some dead trees, these calculations were usually based on <50 trees. Thirteen samples had <40 live trees, but only 1 had <30 live trees. *Damage* is based on the location of a permanent infection, or canker, within a tree crown (minor damage = branch canker >60 cm from stem; moderate damage = branch canker 15–60 cm from stem; severe damage = branch canker within 15 cm or on the main stem; top kill = foliage dead above stem canker; mortality = no live foliage visible). Cankers within 15 cm of the main stem, or on the main stem, were considered potentially lethal.

We used contingency table analysis as an omnibus test to investigate whether WPBR incidence is independent of the physiographic region in which stands were surveyed. Analysis of variance (ANOVA) and Fisher's Least Significant Difference test (Fisher's LSD) were employed to test for a relationship between physiographic region and WPBR intensity. Means of proportions were normalized with an arcsine square root transformation (Zar 1996).

Differences between WPBR incidence in 1967 and 1995–1997 were analyzed using contingency table analysis. The 1967 survey encompassed 4 national forests within our much broader study area. Therefore, we used only the 1995–1997 samples that corresponded with these same 4 national forests. The Mantel-Haenszel test was used to perform a contingency table test of independence between

WPBR incidence and survey year, stratified by national forest (Systat 1992). Coincidentally, because many of our incidental stands were sampled in these national forests, we performed a second Mantel-Haenszel test with a 1995–1997 data set that included 20 of our incidental samples.

RESULTS

Southward Spread of White Pine Blister Rust

MIDDLE ROCKY MOUNTAINS.—The most southerly location of WPBR in the middle Rocky Mountains that we found was at 42.5°N latitude in the Gannett Hills of Wyoming, near the Idaho border (Fig. 2). The 4 *P. flexilis* inspected at this lower treeline location had numerous WPBR cankers. The site, at the entrance to Allred Flat campground, is only about 45 km farther south of the southern WPBR limit reported by Brown and Graham (1969). The disease may have spread further south. Our only sample location south of the Allred site, however, was >40 km away, on Commissary Ridge (Fig. 2), the southernmost stand of *P. albicaulis* in western Wyoming (R. Lanner personal communication). We found no WPBR at this location.

NORTHERN AND EASTERN GREAT BASIN.—No WPBR was found south of Skinner Canyon at approximately 42.5°N latitude, the southernmost location in Idaho reported by Krebill (1964; see Fig. 2). We also found no evidence of WPBR infection in any of our Utah sample locations.

GREAT BASIN.—WPBR was found in no forest islands associated with the Basin and Range geomorphology that occurs throughout most of Nevada. However, WPBR was found in *P. monticola* and *P. albicaulis* at 2 locations in the Carson Range (Fig. 3). This was the 1st report of WPBR in Nevada (Smith et al. 2000). However, the Carson Range is not typical of isolated forests elsewhere in the state because it is linked by nearly continuous forest cover to the Sierra Nevada ecosystem.

EASTERN SLOPES OF THE SOUTHERN SIERRA NEVADA.—WPBR has been present in *P. lambertiana* in the southern Sierra Nevada since at least the 1960s and now extends throughout almost the entire range of that species (Kliejunas 1996). Southward spread of WPBR in *P. monticola* and *P. albicaulis* has been somewhat

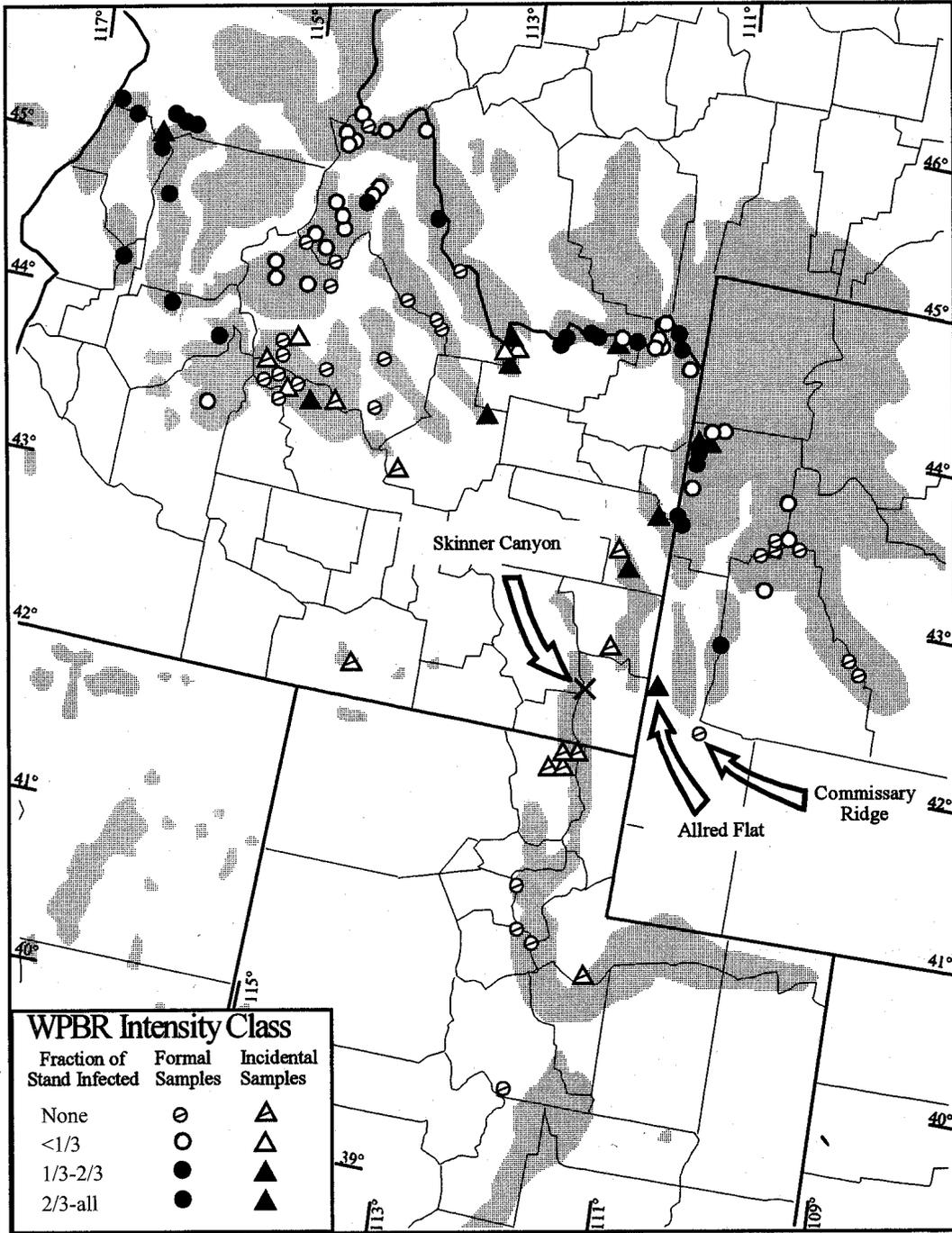


Fig. 2. Northern and middle Rocky Mountain sample locations and WPBR intensity for 100 formal samples and 27 incidental samples. X marks the location of Skinner Canyon, the southernmost WPBR pine infection location in the Rocky Mountains reported by Kriebill (1964). Distribution of white pine species (shaded areas) derived from Little (1971).

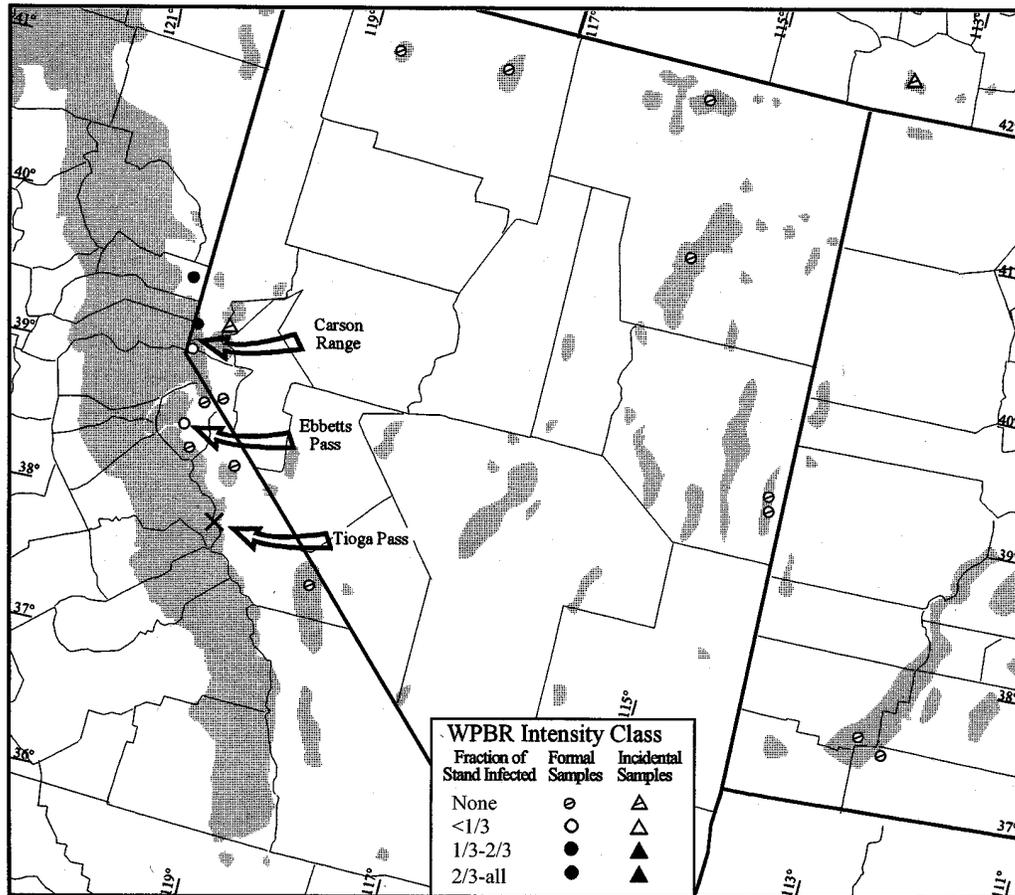


Fig. 3. Sierra Nevada and Great Basin sample locations and WPBR intensity for formal and incidental samples. Distribution of white pine species (shaded areas) derived from Little (1971).

slower. The southernmost location of WPBR in *P. albicaulis* that we observed was in the Sierra Nevada at Ebbetts Pass, at about 38°N latitude (Fig. 3). We know of no other reports of WPBR in *P. albicaulis* south of this location in the Sierra Nevada. However, *P. monticola* infected with WPBR have been observed much farther south, in the Sequoia National Forest, in recent years (J. Pronos personal communication). We did not find WPBR in *P. albicaulis* and *P. monticola* at our Carson-Iceberg Wilderness sample site, which was our southernmost sample location in the Sierra Nevada at a latitude approximately 7 km south of Ebbetts Pass (see Fig. 3). The disease also was not found during informal inspections of *P. albicaulis* and *P. flexilis* near Mammoth Lakes, California, and along the

North Fork of Big Pine Creek, near the town of Big Pine, California (J. Smith, August 1997, personal observation). Both locations are farther south than Tioga Pass, in Yosemite National Park, where Hoff et al. (1994) reported that no WPBR was observed in *P. albicaulis* in 1992 (see Fig. 3).

White Pine Blister Rust Incidence and Intensity

Region-wide incidence of WPBR in sub-alpine white pine samples was 59% (59 of 100 sampled stands). Average intensity in infected stands was approximately 36%. Addition of 27 incidental samples does not substantially change these overall infection values (Table 1); however, only the 100 formal samples were used in the statistical analysis.

TABLE 1. Incidence of white pine blister rust cankers, potentially lethal cankers, and intensity in formal white pine sample stands and in all sample stands during 1995–1997 in the Intermountain West.

Physiographic region/ Section ^c	Formal sample stands ^a				All sample stands ^b		
	Stands sampled (N)	Stands infected (%)	Average intensity (%)	Average incidence of potentially lethal cankers (%)	Stands sampled (N)	Stands infected (%)	Average intensity (%)
Northern Rocky Mountains	60	73.3	34.9	9.8	73	74.0	38.4
I. Wallowa–Seven Devils	3	100	59.0	20.9	3	100	59.0
II. Idaho Batholith	12	75.0	25.6	6.4	14	71.4	24.7
III. Western Salmon Uplands	4	100	52.9	19.1	5	100	52.3
IV. Eastern Salmon Uplands	14	85.7	16.6	6.3	14	85.7	16.6
V. Challis	6	0	0	0	9	22.2	52.0
VI. Open Rocky Mountains	8	25.0	21.1	1.4	14	50.0	51.0
VII. Mtns. of the upper Snake River hdwtrs.	13	100	52.0	21.0	14	100	51.9
Middle Rocky Mountains	22	54.5	38.8	6.8	34	50.0	51.8
VIII. Wyommide Ranges	10	90	48.4	16.2	17	82.3	60.8
IX. Wind River	8	37.5	9.9	0.3	8	37.5	9.9
X. Wasatch	4	0	0	0	9	0	0
Great Basin ^d	12	0	0	0	14	0	0
Slopes and associated ranges of the Sierra Nevada	6	66.7	32.6	9.4	6	66.7	32.6
TOTALS	100	59.0	36.1	8.1	127	58.3	41.7

^an = 100 transect and plot samples only.^bn = 100 transect and plot samples plus 27 incidental samples.^cPhysiographic regions and sections adapted from Steele et al. (1981, 1983).^dIncludes 2 samples from the western edge of the Colorado Plateau physiographic province in Utah.

Results of the contingency table analysis suggested that WPBR incidence is not independent of physiographic region (chi square = 21.57, $P < 0.001$). Using Fisher's LSD, we found a significant pairwise comparison between the Great Basin, where no WPBR was found, and the other 3 regions: northern Rocky Mountains ($P < 0.001$), middle Rocky Mountains ($P = 0.001$), and Sierra Nevada ($P = 0.003$). Significant differences in disease incidence among these latter 3 regions were not detected. Observed levels of significance ranged from 0.126 to 0.793 for pairwise comparisons among these regions.

We found no evidence to suggest a difference in WPBR intensity among any of the infected regions. Analysis of variance showed no significant relationship between the arcsine square root transformed mean of the proportion of trees infected and the physiographic

region in which the sample originated ($P = 0.970$).

Damage and Mortality Caused by White Pine Blister Rust

Severe damage or potentially lethal infections were found in 61% (630 of 1029) of WPBR-infected trees. Incidence of potentially lethal canker infections was highest in the Wallowa–Seven Devils Mountains of the upper Snake River headwaters and Wyommide sections (Table 1). Most potentially lethal Wyommide infections were in the Teton Mountains.

Of 5209 trees sampled, 452 (8.7%) were standing dead trees, and 154 of these had died recently (i.e., their bark and fine limbs were still present). We did not attempt to diagnose the cause of death of the 298 "old dead" trees (those with no bark or fine limbs remaining). Of the new dead trees, 34 (22%) had definite

signs of WPBR canker girdling on the main stem, such as residual aecial peridia, pycnial (spermagonial) scars, swollen, cracked bark, and evidence of rodent feeding on spermagonial exudate. The other 120 trees appeared to have died from physical damage, other diseases, and unknown causes. Twelve (35%) of 34 trees killed by WPBR were in the 5-cm-DBH class, and all trees killed by WPBR were <~30 cm DBH (see Fig. 4). Severe damage, top kill, and mortality were proportionally more prevalent in smaller-diameter trees, while minor damage was more common in larger-diameter trees. The overall mortality attributable to WPBR, calculated as the 34 WPBR-killed trees divided by the total number of live and new dead (diagnosable) trees, was 0.7%.

Comparison with 1967 Disease Levels

In the 1967 survey of Intermountain West white pines in eastern Idaho and western Wyoming, 9 of 31 sample locations had WPBR in either white pines or *Ribes* (Brown and Graham 1969). From the data in that report, we calculated an overall incidence of WPBR in pines to be about 12% (3 of 26 white pine sample locations; Table 2). Results of the Mantel-Haenszel test suggest that WPBR incidence is independent of the survey year (chi square = 3.09, $P = 0.079$). However, incorporating our 20 incidental samples into the analysis resulted in a significant test of independence result (chi square = 8.42, $P = 0.0004$).

We did not statistically analyze WPBR intensity between survey years because of the prohibitively small number of sample locations in the 1967 data. However, we inferred that the average intensity of WPBR in pines was approximately 38% in the 1967 survey, compared to 51% in 1995–1997. Overall, slightly more than 1% (14 of 1078) of white pines inspected in the 1967 survey had WPBR. In comparison, 31% (785 of 2546) of white pines had WPBR in our survey of the same area.

DISCUSSION

Increase in White Pine Blister Rust Incidence and Intensity

Results of the initial statistical analysis of WPBR incidence in the 1967 and 1995–1997

surveys were somewhat inconclusive. When only the formal 1995–1997 sample data were used, we rejected the hypothesis that WPBR incidence has changed significantly between the 2 periods. However, when the 20 incidental plot data points were added, we found evidence to indicate an increase in WPBR incidence. We feel that the test of independence using only formal sample data was underpowered, and that the addition of more samples in the Caribou and Sawtooth National Forests increased the ability of the test to detect a significant change in WPBR incidence. Additionally, the 1 high-intensity sample in the 1967 survey consisted of only 6 white pines, which were all infected. This sample is probably not representative of overall WPBR intensity in the 1967 study area. The other 2 infected samples from that survey, with 5% and 10% intensities, had sample sizes of 20 and 30 trees, respectively.

Our analysis and anecdotal observations indicate an increase in incidence and intensity of WPBR in the middle and northern Rocky Mountain portions of our study area over the past 30 yr. Results from our WPBR damage assessment support this hypothesis. We found that the vast majority of infected trees had cankers close to branch tips, near points of initial infection, the needles. Had WPBR levels remained constant since the 1960s, we presumably would have found cankers more evenly distributed throughout the canopy and main stem of infected trees. Furthermore, we found that smaller-diameter (and usually younger) white pines suffered more serious damage, including top kill and mortality, than larger-diameter trees. This DBH–damage class relationship is very likely related to distance between foliage (the point of infection) and main stem, and the amount of cambium circumference that must be girdled in order to kill the top of the tree, or cause mortality. Assuming cankers progress at a roughly similar rate in smaller- and larger-diameter trees, if the rate of infection had remained constant over the past several decades, we should have seen more serious infections in larger-diameter trees.

Implications of an Increase in Damage Caused by White Pine Blister Rust

In some heavily infected stands most, if not all, small-diameter trees are infected. It is

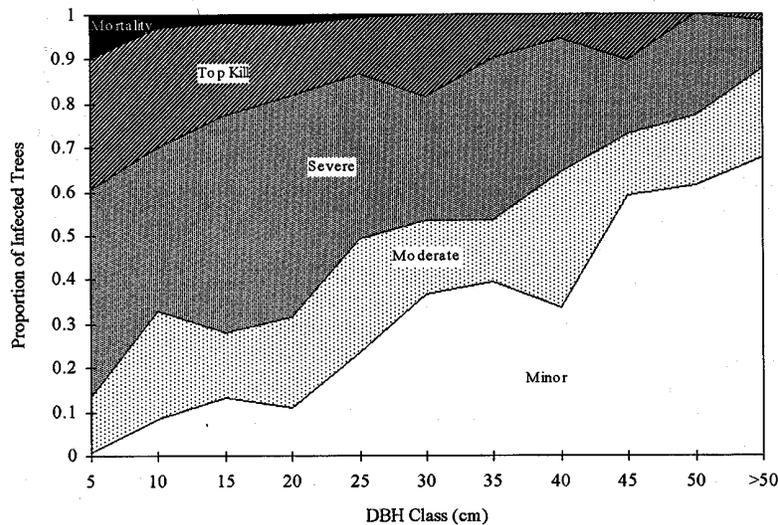


Fig. 4. WPBR damage on infected white pines by DBH (diameter at breast height) class. White pines were surveyed during 1995–1997 in the Intermountain West.

difficult to say with certainty whether any of these trees will live long enough to reproduce. Estimates of canker growth rates and natural inactivation of cankers are derived from *P. monticola* and *P. lambertiana* studies (Harvey 1967, Kimmey 1969, Hungerford 1977). Relatively little is known about the inactivation rate and growth rate of cankers in subalpine white pine species. Thus, infections far out on the end of a limb are also potentially lethal. We should note that the number of cankers on each tree affects the probability of a single canker reaching the main stem (Slipp 1953), and we did not collect data on this disease characteristic. Yet, regardless of the severity of infection, we rarely found inactive cankers and have no reason to believe that trees will escape serious damage even if they have only a few cankers. Research has shown that genetic resistance to infection or canker growth is rare in many subalpine white pine species (Hoff et al. 1980, 1994). Thus, damage from WPBR infection will likely increase in most currently infected trees, and smaller trees will succumb to top kill and mortality more quickly than larger trees. In high-intensity areas it is likely that only those few trees genetically resistant to WPBR will survive to maturity. Furthermore, we observed WPBR cankers high in the crowns of trees at most sites. Cones of at least 1 white pine species, *P. albicaulis*, are pro-

duced primarily in the upper 1/3 of the crown (Keane et al. 1994). Nonresistant trees that do survive to maturity in high-intensity areas may lose their reproductive capability long before they die.

Of immediate concern to forest managers is the area in the northeastern portion of the study area where WPBR incidence is high, intensity is high on many sites, and mortality is beginning to occur. Increased mortality in the western and southern portions of the Greater Yellowstone Ecosystem is of particular concern. This area is home to the threatened grizzly bear (*Ursus arctos horribilis*) that uses *P. albicaulis* seeds as an autumn food source. Abundance of *P. albicaulis* seeds is linked to grizzly bear cub production and to frequency of bear-human conflicts (Kendall and Arno 1990, Mattson and Reinhart 1994). The west slope of the Teton Range, where we found very high WPBR incidence and intensity, is also of particular concern. Loss of *P. albicaulis* in this prime grizzly bear habitat would be severely detrimental to future grizzly bear conservation efforts (D. Mattson personal communication).

Other areas of high WPBR incidence and intensity include the Idaho Batholith, eastern Salmon River Mountains, and Seven Devils Mountains in the northwestern part of the study area. We associated recent mortality of

TABLE 2. Comparison of 1967 and 1995–1997 white pine blister rust surveys in 4 Idaho and Wyoming national forests.

National forest	1995–1997 survey ^a			1967 survey ^b		
	Stands sampled (N)	Stands infected (%)	Average intensity ^c (%)	Stands sampled (N)	Stands infected (%)	Average intensity (%)
Bridger-Teton	12	50.0	34.5	16	18.8	38.3
Caribou ^d	3	33.3	78.1	4	0	0
Sawtooth	13	23.1	43.8	5	0	0
Targhee	30	96.7	55.3	1	0	0
TOTALS	58	67.2	51.5	26	11.5	38.3

^aTransect data (n = 38) and supplemental transect data (n = 20)

^bBrown and Graham (1969)

^cSum of percentage of trees infected with WPBR in infected samples divided by number of infected samples.

^dIncludes the Idaho portion of the Cache National Forest surveyed by Brown and Graham (1969).

P. albicaulis with *Dendroctonus ponderosae* (mountain pine beetle) and root pathogens (unidentified). The interaction of these opportunistic parasites and WPBR may accelerate white pine mortality in this area.

Further Spread of White Pine Blister Rust

Whether WPBR will cause problems for white pines in areas that are now free of the disease is unknown. The southward spread of WPBR in the southern portion of the middle Rocky Mountains and along the northern boundary of the Great Basin has proceeded very slowly, if at all, since the 1960s. Furthermore, we found no evidence of WPBR in any of the isolated Great Basin forests closest to infected regions, even those that are only a few kilometers from moderately high infection centers in the Sierra Nevada. Isolation from *Ribes* populations, or infected pine populations, may be a primary factor in the absence of WPBR in the Great Basin.

Lack of climatic conditions conducive to WPBR spread and intensification may also be an important factor in explaining the absence of WPBR in the Great Basin. Climates of the Great Basin and central Utah are generally more arid than elsewhere within the range of WPBR. In Great Basin mountain ranges, it is unknown whether moisture events necessary for infection by WPBR, such as fog, rain, and dew (Mielke 1943), occur frequently enough for WPBR to persist or intensify.

The apparent absence of WPBR in the Great Basin and the Rocky Mountains in Utah suggests that there is not a continuous, or even broken, corridor of infected pines between

infection centers in the Sierra Nevada or northern and middle Rocky Mountains to the New Mexico infection site described by Hawksworth (1990). We did not investigate the eastern Rocky Mountain ranges; however, our review of WPBR survey literature documents a long-known WPBR infection in the southern end of the Laramie Range in southeastern Wyoming (Brown 1978). This range is the northwestern extension of the Colorado Front Range (Lageson and Spearing 1988). Limber pines grow in both ranges, indicating a possible route for spread of the disease. The mountains in New Mexico receive relatively abundant moisture during late summer due to a monsoonal climate (Baker 1944). The disease also may have spread via long-distance spore dispersal and become established only where climatic conditions and alternate host distribution were favorable.

We hypothesize that the combination of isolation from reservoirs of infected pines and environmental conditions which are relatively unfavorable to initial WPBR infection has thus far been a barrier to WPBR establishment in the Great Basin. But, the potential for spread and subsequent intensification of WPBR should not be underestimated. During our survey we observed that, at some sites, most WPBR cankers were located at a similar distance from the main tree stem. Since we recorded only the most proximal canker found on each tree, we could not perform a statistical analysis of this apparent clustering of canker distances. However, the observation alone suggests that infections occurred during the same time period. This type of pattern is indicative of a wave year phenomenon wherein

most WPBR infections occur only during years when environmental conditions are favorable for infection (Mielke 1943, Peterson 1971). The probability of a wave year may be lower in the relatively arid Intermountain West than in the maritime provinces and might help explain the slow spread of WPBR in our study area. However, we observed several sites in which distances of cankers from the main stem were more evenly distributed, indicating a higher frequency of years in which environmental conditions are favorable enough for at least some infections to occur.

A recent climate analysis of the Greater Yellowstone Ecosystem suggests that years during which the climate is favorable to WPBR infection are common in the subalpine zone. If montane climates in other portions of the Intermountain West yield frequent wave years, factors such as the timing of *Ribes* leaf emergence and distribution of white pine and susceptible *Ribes* may be more important in explaining the spread of WPBR in the Intermountain West. Additional research to age cankers and to clarify the roles of climate, site conditions, and the distribution of host species will help researchers more accurately predict where WPBR is likely to occur and intensify in the immediate future, and under long-term climate change scenarios.

Management Implications

Training land managers to identify WPBR and establishing a frequent, regular monitoring regime would help in the early detection of new infection centers. If, as we presume, years in which the climate is conducive to infection are rare in the Great Basin, early detection and implementation of control measures, such as canker removal or selective tree removal, might slow or even stop a small infection center in the isolated white pine populations of the region. In heavily infected areas of the Intermountain West, silvicultural or prescribed fire treatments could be used to reduce inoculum potential or to promote natural reproduction of phenotypically resistant white pines by removing infected white pines or other competing tree species. Identifying and collecting seed from resistant trees could provide stock for breeding programs and for reintroduction of native resistant stock. In any situation, management of white pine ecosystems should be based not only on WPBR

epidemiology, but also on preservation of white pine genetic diversity and local disturbance ecology.

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LITERATURE CITED

- ARNO, S.F., AND R. HAMMERLY. 1984. Timberline: mountain and arctic forest frontiers. The Mountaineers, Seattle, WA. 304 pp.
- BAKER, F.S. 1944. Mountain climates of the western United States. Ecological Monographs 14:225-254.
- BROWN, D.H. 1978. Extension of the known distribution of *Cronartium ribicola* and *Arceuthobium cyanocarpum* on limber pine in Wyoming. Plant Disease Reporter 62:905.
- BROWN, D.H., AND D.A. GRAHAM. 1969. White pine blister rust survey in Wyoming, Idaho, and Utah: 1967. U.S. Department of Agriculture, Forest Service, Northern Region, State and Private Forestry Report. 11 pp.
- CONKLIN, D.A. 1994. White pine blister rust outbreak on the Lincoln National Forest and Mescalero-Apache Indian Reservation, New Mexico. U.S. Department of Agriculture, Forest Service, Forest Pest Management Report R3-94-2. 12 pp.
- FENNEMAN, N.M. 1931. Physiography of western United States. McGraw-Hill, New York. 534 pp.
- HARVEY, G.M. 1967. Growth rate and survival probability of blister rust cankers on sugar pine branches. U.S. Department of Agriculture, Forest Service Research Note PNW-54. 6 pp.
- HAWKSWORTH, F.G. 1990. White pine blister rust in southern New Mexico. Plant Disease 74:938.
- HOFF, R.J., R.T. BINGHAM, AND G.I. McDONALD. 1980. Relative blister rust resistance of white pines. European Journal of Forest Pathology 10:307-316.
- HOFF, R.J., S.K. HAGLE, AND R.G. KREBILL. 1994. Genetic consequences and research challenges of blister rust in whitebark pine forests. Pages 118-126 in W.C. Schmidt and F.K. Holtmeir, compilers, Proceedings of the international workshop on subalpine stone pines and their environment: the status of our

- knowledge. U.S. Department of Agriculture, Forest Service Intermountain Research Station publication INT-GTR-309.
- HUNGERFORD, R.D. 1977. Natural inactivation of blister rust cankers on western white pine. *Forest Science* 23:343-350.
- KEANE, R.E., AND S.F. ARNO. 1993. Rapid decline of whitebark pine in western Montana: evidence from 20-year measurements. *Western Journal of Applied Forestry* 8(2):44-47.
- KEANE, R.E., AND P. MORGAN. 1994. Decline of whitebark pine in the Bob Marshall Wilderness Complex of Montana, U.S.A. Pages 245-253 in W.C. Schmidt and F.K. Holtmeir, compilers, Proceedings of the international workshop on subalpine stone pines and their environment: the status of our knowledge. U.S. Department of Agriculture, Forest Service, Intermountain Research Station publication INT-GTR-309.
- KEANE, R.E., P. MORGAN, AND J.P. MENAKIS. 1994. Landscape assessment of the decline of whitebark pine (*Pinus albicaulis*) in the Bob Marshall Wilderness Complex, Montana, USA. *Northwest Science* 68: 213-229.
- KENDALL, K.C. 1995. June 29 memorandum on monitoring methods, field forms, and instructions for the Whitebark Pine Monitoring Network. On file at U.S. Department of Agriculture, Forest Service, Forest Health Protection, Boise [Idaho] Field Office.
- KENDALL, K.C., AND S.F. ARNO. 1990. Whitebark pine: an important but endangered wildlife resource. Pages 264-273 in W.C. Schmidt and K.J. McDonald, compilers, Proceedings of a symposium on whitebark pine ecosystems: ecology and management of a high mountain resource. U.S. Department of Agriculture, Forest Service, Intermountain Research Station, General Technical Report INT-270.
- KENDALL, K., D. SCHIROKAUER, E. SHANAHAN, R. WATT, D. REINHART, R. RENKIN, S. CAIN, AND G. GREEN. 1996. Whitebark pine health in northern Rockies national park ecosystems: a preliminary report. Page 16 in R.E. Keane, editor, U.S. Department of Agriculture, Forest Service, Intermountain Research Station, Nutcracker Notes 7.
- KIMMEY, J.W. 1938. Susceptibility of ribes to *Cronartium ribicola* in the West. *Journal of Forestry* 36:312-320.
- _____. 1969. Inactivation of lethal-type blister rust cankers on western white pine. *Journal of Forestry* 67:296-299.
- KLEJUNAS, J. 1996. An update of blister rust incidence on the Sequoia National Forest. U.S. Department of Agriculture, Forest Service, Pacific Southwest Region, State and Private Forestry, Forest Pest Management Report R96-01. 3 pp.
- KREBILL, R.G. 1964. Blister rust found on limber pine in northern Wasatch Mountains. *Plant Disease Reporter* 48:532.
- KREBILL, R.G., AND R.J. HOFF. 1995. Update on *Cronartium ribicola* in *Pinus albicaulis* in Rocky Mountains, USA. Pages 119-126 in Proceedings of the 4th IUFRO rusts of pines working party conference, Tsukuba, Japan. USDA Forest Service, Intermountain Research Station, Ogden, UT.
- LAGESON, D.R., AND D.R. SPEARING. 1988. Roadside geology of Wyoming, 2nd edition. Mountain Press Publishing, Missoula, MT. 271 pp.
- LITTLE, E.L., JR. 1971. Atlas of United States trees. Volume 1. Conifers and important hardwoods. Miscellaneous Publication 1146. U.S. Department of Agriculture, Forest Service, Washington, DC. 200 pp.
- LUNDQUIST, J.E., AND B.W. GEILS. 1992. White pine blister rust on limber pine in South Dakota. *Plant Disease* 76:538.
- MATTSON, D.J., AND D.P. REINHART. 1994. Bear use of whitebark pine seeds in North America. Pages 212-220 in W.C. Schmidt and F.K. Holtmeir, compilers, Proceedings of the international workshop on subalpine stone pines and their environment: the status of our knowledge. U.S. Department of Agriculture, Forest Service Intermountain Research Station publication INT-GTR-309.
- MIELKE, J.L. 1943. White pine blister rust in western North America. *Yale University School Forestry Bulletin* 52. 155 pp.
- PETERSON, R.S. 1971. Wave years of infection by western gall rust on pine. *Plant Disease Reporter* 55:163-167.
- SLIPP, A.W. 1953. Survival probability and its application to damage survey in western white pine infected with blister rust. University of Idaho, Forest, Wildlife, and Range Experiment Station, Research Note 7. 13 pp.
- SMITH, J.P., J.T. HOFFMAN, K. SULLIVAN, E.P. VAN ARSDEL, AND D. VOGLER. 2000. First report of white pine blister rust in Nevada. *Plant Disease* 84(5):594 (note 2053).
- STEELE, R., S.V. COOPER, D.M. ONDOVE, D.W. ROBERTS, AND R.D. PFISTER. 1983. Forest habitat types of eastern Idaho-western Wyoming. U.S. Department of Agriculture, Forest Service, Intermountain Forest and Range Experiment Station General Technical Report INT-144. 122 pp.
- STEELE, R., R.D. PFISTER, A.R. RUSSELL, AND J.A. KITTAMS. 1981. Forest habitat types of central Idaho. Forest habitat types of eastern Idaho-western Wyoming. U.S. Department of Agriculture, Forest Service, Intermountain Forest and Range Experiment Station General Technical Report INT-114. 138 pp.
- SYSTAT. 1992. Systat for Windows: statistics. Statistical analysis software reference manual. Version 5 edition. Systat, Inc., Evanston, IL. 750 pp.
- ZAR, J.H. 1996. Biostatistical analysis. 3rd edition. Prentice Hall, NJ. 662 pp.

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APPENDIX. White pine species sampled, incidence of white pine blister rust, mortality, and geographic location of stands sampled during 1995–1997 in the Intermountain West.

Sample ^a	Species sampled ^b	Live trees (N)	Infected trees (N)	WPBR mortality ^c (%)	Coordinates		Sample location
					Latitude	Longitude	
BOI01	A	39	3	0	43.61235	115.44104	Near Trinity Mountain, ID
BOI02	A	49	20	2.0	43.99066	115.32479	N of Shepherd Peak, ID
BOI03	A	47	30	1.9	44.17672	115.75901	E of Scott Mtn. Lookout, ID
BOI04	A	57	31	0	44.41286	116.12706	Snowbank Mountain, ID
BRI01	A	48	17	0	43.16663	110.18464	Spring Creek near The Rim, WY
BRI02	A	52	0	0	43.39252	110.08593	Bacon Ridge, W of Mosquito Lake, WY
BRI03	A	50	3	0	43.46214	109.93764	Near Fish Creek Work Center, WY
BRI04	A	50	9	0	43.75610	110.07015	Togwotee Pass, WY
BRI05	A	51	22	0	42.84406	110.58272	McDougal Gap, WY
BRI06	A	53	0	0	42.07242	110.57198	Commissary Ridge, WY
GRO01	A	53	3	0	43.44329	110.06318	Buffalo Meadow, WY
WIN01	A	51	0	0	42.69115	109.23433	Upper S Temple Creek, WY
WIN02	A	53	0	0	42.67272	109.25683	Big Sandy Opening, WY
WIN03	A	37	0	0	43.45941	109.94090	Near Fish Creek, WY
WIN04	A	39	0	0	43.29536	109.93423	Near Gypsum Creek, WY
LIM18	F	4	4	—	42.49976	110.91625	Allred Flat, WY
LIM05	F	15	0	—	43.36980	111.49372	Fall Creek, ID
LIM11	F	30	0	—	42.65791	111.62572	Soda Springs, ID
LIM12	F	32	25	—	43.20678	111.21272	Jensen Pass, ID
CED01	F, L	50	0	0	37.56362	112.84558	Cedar Canyon, UT
BRY01	F, L	38	0	0	37.78900	112.14900	Church Garden, UT
HUM01	A	38	0	0	41.83000	115.46200	Jarbridge Mountain, NV
PPR01	A, F	50	0	0	41.68096	118.74585	Pine Forest Range, NV
SAN01	F	47	0	0	41.78800	117.55075	Santa Rosa Mountains, NV
RUB01	A, F	36	0	0	40.63174	115.40645	Ruby Mountains, NV
SNA01	F	41	0	0	39.00517	114.30749	Wheeler Peak, NV
SNA02	F	34	0	0	38.96481	114.27636	Baker Creek, NV
CAR01	A	49	24	0	39.31264	119.89728	Mt. Rose Summit, NV
CAR02	M	50	6	0	39.06936	119.89367	Spooner Summit, NV
SIE01	A, M	49	0	0	38.51056	119.56856	Carson-Iceberg Wilderness, CA
SIE02	A, M	45	7	0	38.54268	119.81133	Ebbetts Pass, CA
SIE03	M	50	0	0	38.67728	119.59634	Monitor Pass, CA
PNM01	M	51	0	0	38.81221	119.51499	Pine Nut Mountains, NV
BAL01	M	50	27	0	39.60208	120.10436	Bald Mtn. Range, CA
SWE01	A	52	0	0	38.41627	119.26680	Sweetwater Range, CA
WHI01	L	47	0	0	37.39044	118.17978	Schullman Grove, CA
VIR01	M	6	0	—	39.33252	119.63949	Virginia Range, NV
SAL15	A	51	27	0	45.37411	115.86560	Marshall Lake, ID
SAL16	A	50	30	2.0	45.34305	115.84562	Near California Lake, ID
SAL17	A	46	30	0	45.31940	115.79160	War Eagle Peak, ID
SAL18	A	44	35	0	44.87426	115.94709	Boulder Peak, ID
SAL19	A	15	5	0	45.01326	116.11976	Brundage Mountain, ID
SEV01	A	45	38	0	45.35110	116.50976	Seven Devils, ID
SEV02	A	42	16	16.0	45.34903	116.49246	Heaven's Gate, ID
WBP04	A	50	25	—	45.18590	116.13010	Hazard Lake, ID
BIT01	A	49	1	0	45.46384	114.32236	Continental Divide–Spring Creek, ID
BIT02	A	48	0	2.0	45.47249	114.35710	Blue Note Lookout, ID
BIT03	A	50	7	3.8	45.55438	114.51407	East of Reynolds Lake, ID
BIT04	A	48	7	0	45.51721	119.83699	Morgan Mountain, ID
BIT05	A	51	0	0	44.78314	113.35547	Grizzly Hill, ID
BIT06	A	52	21	0	45.08084	113.54208	Headwaters of Kenney Creek, ID
CLE01	A	47	15	4.1	45.39577	114.61340	Corn Lake, ID
CLE02	A	52	10	0	45.38938	114.55931	Long Tom Ridge/Swamp Creek, ID
CLE03	A	50	10	1.9	45.35919	114.57914	Long Tom Ridge/Bear Camp Spring, ID
LEM02	A	44	0	0	44.43026	113.32289	W of Meadow Lake Campground, ID
LEM03	A	49	0	0	44.44096	113.31661	N of Meadow Lake Campground, ID
LEM04	A	31	0	0	44.65323	113.65154	Mill Lake, ID

APPENDIX. Continued.

Sample ^a	Species sampled ^b	Live trees (N)	Infected trees (N)	WPBR mortality ^c (%)	Coordinates		Sample location
					Latitude	Longitude	
SAL01	A	49	6	0	45.25773	114.01104	Salmon R. Mountain/Wallace Lk., ID
SAL02	A	48	8	0	45.19917	114.04678	Salmon R. Mountain/Turner Gulch, ID
SAL03	A	53	21	0	45.13484	114.06392	Salmon R. Mountain/Phelan Mtn., ID
SAL04	A	51	6	0	45.00075	114.42997	Red Rock Peak, ID
SAL05	A	52	6	0	45.06037	114.44690	Quartzite Mountain, ID
SAL06	A	52	3	0	45.09697	114.51502	Crags Campground, ID
SAL07	A	50	0	0	45.08478	114.52858	S of Crags Campground, ID
LOS01	F	31	0	0	44.13672	113.81795	W Slope, Mt. Borah, ID
BOU01	A	42	0	0	43.91627	114.36801	North Fork of Lost River Headwaters, ID
MAC01	A	52	1	0	43.88511	113.68992	White Knob Mountains, ID
SAL08	A	51	1	0	44.46408	114.73555	Loon Creek Summit, ID
SAL09	A	52	0	0	44.47163	114.48829	Mill Creek Summit, ID
SAL10	A	34	2	0	44.75359	114.67964	Sleeping Deer Lookout, ID
SAL11	A	52	1	0	44.67445	114.55989	Fly Creek Point, ID
SAL12	A	52	0	0	44.60336	114.47234	Twin Peak, ID
SAL13	A	52	4	0	44.58764	115.00826	Sheep Mountain, ID
SAL14	A	47	10	2.1	44.56863	115.01072	Fontez Creek, ID
SAW01	A	71	0	0	43.96262	114.69275	Pole Creek, ID
SAW02	A	62	0	0	43.88052	114.71073	Galena Summit, ID
SAW03	A	49	0	0	43.59235	114.68143	Dollarhide Summit, ID
SAW04	A	52	0	0	44.02695	114.65040	Phyllis Lake, ID
SAW05	A	89	0	0	43.81082	114.80664	Frenchman Creek, ID
SAW06	A	46	0	0	43.84080	114.50401	Boulder City, ID
WBPO1	A	30	5	—	43.32808	114.61948	Boulder View, ID
WBPO2	A	50	2	—	44.17230	114.57488	Railroad Ridge, ID
WBPO3	A	20	0	—	43.85551	114.88476	Beaver Creek, ID
LIM01	F	15	0	—	43.82085	114.25056	Trail Creek, ID
LIM03	F	50	50	—	43.80094	114.41991	Murdock Creek, ID
LIM02	F	50	0	—	43.42440	113.54859	Craters of the Moon, ID
LIM19	F	50	0	—	42.31617	113.64109	Mt. Harrison, ID
CEN01	A	48	32	4.0	44.41939	112.34467	Pleasant Creek Summit, W, ID
CEN02	A	51	33	0	44.41944	112.34460	Pleasant Creek Summit, E, ID
CEN03	A	38	8	0	44.55312	111.42022	Sawtelle Creek, ID
CEN04	A	42	5	0	44.56013	111.44394	Sawtelle Krumholtz site, ID
CEN05	A	49	45	2.0	44.53543	112.05843	Big Table Mountain, W, ID
CEN06	A	48	45	0	44.53213	112.03794	Big Table Mountain, E, ID
CEN07	A	33	10	0	44.41944	112.34460	Sawtelle Peak, ID
CEN08	A	41	33	2.3	44.56038	111.58557	Blair Lake, ID
CEN09	A	50	11	0	44.53138	111.85361	Hancock Lake, ID
HEN01	A	33	32	15.2	44.68883	111.29229	Targhee Pass/Avalanche Gulch, ID
HEN02	A	42	31	6.0	44.62466	111.25966	Mt. Two Top, ID
HEN03	A	52	11	0	44.69912	111.39685	Black Mountain, ID
LEM01	A	42	0	0	44.34804	113.26162	Spring Mountain Canyon, ID
MCP01	A	52	1	0	44.51267	111.15442	Moose Creek Plateau/Black Canyon, ID
TET01	A	52	9	0	44.11943	110.89848	Flagg Ranch Road, WY
TET02	A	46	34	2.1	43.49712	110.95485	Teton Pass-East, WY
TET03	A, F	49	20	0	43.88905	110.96675	Badger Creek, WY
TET04	F	49	43	0	43.49131	110.95602	Teton Pass-West, WY
TET06	A	53	16	0	44.12063	110.85708	Camp Loll, WY
TET07	A	51	27	1.9	44.03884	110.90426	Hominy Peak, WY
WBPO5	A	9	7	—	44.04221	110.94600	Hominy Peak, W, WY
WBPO6	A	40	20	—	44.51400	111.86200	Alduous Lake, ID
WBPO7	A	10	8	—	44.07353	110.97240	Jackass Loop, WY
LIM04	F	50	40	—	44.37300	112.70800	Webber Creek, ID
LIM06	F	30	24	—	43.52364	111.25986	Mike Spencer Canyon, ID
LIM07	F	15	4	—	44.27560	112.75000	Crooked Creek, ID
LIM08	F	50	4	—	44.30068	112.92534	Nicholia Canyon, ID
LIM09	F	20	20	—	43.93972	112.92534	Deer Canyon, ID
LIM10	F	30	30	—	44.24532	112.80766	Buckhorn Canyon, ID

APPENDIX. Continued.

Sample ^a	Species sampled ^b	Live trees (N)	Infected trees (N)	WPBR mortality ^c (%)	Coordinates		Sample location
					Latitude	Longitude	
WAS03	F	44	0	0	39.86788	111.74625	Mt. Nebo, UT
WAS01	F	40	0	0	40.60006	111.59843	Solitude Ski Area, UT
WAS02	F	41	0	0	40.65176	111.59385	Silver Peak, UT
WAS04	F	52	0	0	40.56368	111.65012	Snowbird Ski Area, UT
STA01	F	52	0	0	40.48520	112.62066	Stansbury Range, UT
LIM13	F	4	0	—	37.49200	111.17500	Yellow Pine, UT
LIM14	F	10	0	—	41.77800	111.63200	Logan Cave, UT
LIM15	F	5	0	—	41.82700	111.59500	Ricks Spring, UT
LIM16	F	10	0	—	41.93900	111.54900	Beaver Mtn., UT
LIM17	F	30	0	—	41.90900	111.45200	Old Limber Trail, UT

^aSamples with LIM or WBP prefixes are incidental samples of *Pinus flexilis* (limber pine) and *P. albicaulis* (whitebark pine).

^bF = *P. flexilis*, A = *P. albicaulis*, M = *P. monticola*, L = *P. longaeva*.

^cMortality data not collected in incidental plots.