

Annual Observations of Conspicuous Canker Activity on Whitebark Pine (2003 to 2007)

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Introduction

Whitebark pine's (*Pinus albicaulis*) notable ecological values, combined with its precarious state, underscore the need for monitoring its health and dynamics. Populations of whitebark pine are in decline throughout most of its range. White pine blister rust, caused by the fungus *Cronartium ribicola*, has denuded stands since introduction during the early 1900s (Tomback and Achuff 2010). The exclusion of fire, which historically promoted whitebark pine over its competitors, has further lessened its proportional abundance with competitors (Murray 2007; Murray and others 2000). Mountain pine beetle epidemics have killed vast acreages since 2003 (Haeussler 2008; Kegley and others, these proceedings; Logan and others 2010). Due to one or more of these threats, whitebark pine has declined in areal cover up to 98 percent in places (Schwandt 2006).

Rust-induced mortality is becoming well-documented across the natural range of whitebark pine (Schwandt and others 2010). However, at a finer scale (i.e., individual tree), the biological interactions taking place between the infected host (whitebark pine) and disease are not well-studied. Understanding dynamics pertaining to natural inactivation of cankers may improve our ability to model death rates, monitor virulence, and develop measures for blister rust control (Kimmey 1969). Natural inactivation of blister rust cankers on western white pine has been described on young trees (Hungerford 1977; Kimmey 1969). Knowledge of canker activity from mature western white pine (*Pinus monticola*), or whitebark pine of any age, appear absent from the literature.

The objective of this study was to track the most apparent signs of canker activity on a sample of whitebark pine over a five-year period. The magnitude and duration of inactive periods were documented. Further insight regarding possible differences between canker locations (branch vs. stem, location on Cascade Crest) was also sought.

Methods

The study location is centered around Crater Lake, Oregon, located on the crest of the southern Cascade Mountain Range. Whitebark pine achieves co-dominance from 2,080 m to the highest elevation in the study area (2,722 m). Mountain hemlock (*Tsuga mertensiana*) and

lodgepole pine (*Pinus contorta* var. *murrayana*) are common co-dominants. Shasta red fir (*Abies × shastensis*), western white pine, and ponderosa pine (*P. ponderosa*) are occasional associates. Common understory flora include California needlegrass (*Achnatherum occidentale*), squirreltail (*Elymus elymoides*), Ross' sedge (*Carex rossii*), Davidson's penstemon (*Penstemon davidsonii* var. *davidsonii*), knotweed (*Polygonum davisiae*), buckwheat (*Eriogonum umbellatum*), woodrush (*Luzula hitchcockii*), and spreading phlox (*Phlox diffusa*). Soils are relatively young, having been derived from the volcanic eruption of Mt. Mazama about 7,700 years ago. Composition is pumice-ash with varying amounts of cobble-size cinder stone, resulting in rapid permeability and loose structure. The climate is typified by heavy snowfall (average = 12.2 m/year) with depths commonly peaking at about 4.5 meters in April (Crater Lake National Park, unpublished data). Winter temperatures are somewhat mild (average = -3.0 °C (26.6 °F)) with summer days averaging 11.0 °C (52.0 °F). Because the study area straddles the crest of the southern Cascade Range, a climate gradient occurs between the east and west portion of the study area. The east side receives less precipitation and has cooler temperatures.

All monitored trees were associated with a set of seven permanent sampling plots established in 2003. The vicinity of each plot was pre-determined to represent the whitebark pine communities present in the study area. Next, each plot was placed within each vicinity based on field reconnaissance of the area, then choosing a plot center location that appeared typical for the vicinity and community (Mueller-Dombois and Ellenberg 1974). Plots were circular, encompassing 300 square meters. Within each plot, all trees were mapped for ease in relocating each subsequent year. In many instances, labelled aluminum tags were affixed to trees to provide additional reference. The following information was recorded for all live and dead standing trees: a unique alphanumeric identifier, species, diameter at breast height (dbh), and overall status (healthy, sick, recently dead, and dead). Trees classified as sick were infected by one or more biotic agents. Also included were instances of mechanical damage that caused foliage-kill. Where white pine blister rust was identified, the following data were recorded: status (active or inactive) and location (distance from ground and main stem) of each canker, plus percent of crown killed. Cankers were noted as occurring on either live branches or stems, but those that grew on both were classified as stem cankers. Blister rust cankers were noted as active when one



Figure 1. Active canker with fruiting structures, resinous, orange-tinted bark, and rodent gnawing.

or more of the following symptoms were present: resin on surface, fungal fruiting structures, or yellow to orange coloured bark (figure 1). Rodent gnawing deemed to occur during the current year's sampling season was also indicative of an active canker.

Results were tabulated and tested for statistical significance. Specifically, annual tallies of active cankers were tested with χ^2 (chi-square) to determine potential differences in activity according to each year (Fowler and Cohen 1990). The number of active vs. inactive cankers were also compared across location in the study area (east vs. west sides) and position on the tree (branch vs. stem) using the Mann-Whitney U -test (Fowler and Cohen 1990). The magnitude of inactivity (percentage of years inactive) was also tested to see if cankers tended to be significantly inactive over their entire span of observations. For this, the randomization goodness-of-fit test was applied (McDonald 2009).

Results

A total of 52 cankers from 46 trees were tracked but tallies varied in any given year due to new infections detected or tree mortality. Trees varied in height from 0.19 m to 7 m (0 to 28.7 cm dbh). Overall, 42 percent of cankers changed their status (active vs. inactive) at least once. Active cankers were more numerous than inactive cankers in every year except 2004 (table 1). Cankers were significantly more active in 2003 and 2006 (χ^2 , Yates correction, $P < 0.05$). The east side tended to have lower activity, however, the disparity between sides was not significant ($U = 8$, $P < 0.05$). Branch cankers were observed to be more active than stem cankers in all years except 2003, although not with significance.

The degree of inactivity for each canker was examined (table 2). Seven cankers showed no activity during the study period and may be devoid of the *C. ribicola* inoculum. For those cankers that had activity, years of idleness tended to be skewed low. For example, 32 of 52 cankers were inactive for less than 50 percent of annual observations. Correspondingly, in comparing inactivity among all percentage classes (table 2), both stem and branch cankers were more likely to be active than inactive ($P = 0.005$ and $P = 0.000$ respectively). A subset of cankers went inactive, and then returned to activity. For these re-activated cankers, eight were latent for only a single year. Another five cankers were inactive two consecutive years. None were found to re-activate after three years. Half of all inactive cankers (at any time during the study) failed to re-activate.

Discussion and Conclusions

This study presents the first published account of annual canker activity of white pine blister rust on whitebark pine. Based on these observations, canker activity appears to vary each year. Activity was significantly higher in 2003 and 2006, but a cause was not investigated. Hungerford (1977) found activity for branch cankers to decrease consistently every year—possibly due to aging trees and/or aging cankers. That result was not observed in this study. Also in contrast, Kimmey (1969) and Hungerford (1977) observed more activity among stem cankers. Because they did not define a classification for individual cankers occurring on both the stem and adjoining branch, these comparisons are of limited merit. While Hungerford (1977) found that 78 percent of inactive cankers failed to re-activate, in this study half of inactive cankers re-activated. During his most active two years, 60 and 72 percent of all cankers were active. Similarly, 67 and 83 percent were active during peaks in my observations.

The reliability of outward signs of activity closely reflecting fungal virulence is not well-documented in literature. The disease may continue to thrive, and possibly spread beneath the bark without conspicuous resin, sporulation, rodent-gnawing, or bark discoloration. Notwithstanding, no cankers appeared to re-activate after three years of inactivity.

Table 1. Percentage of individual blister rust cankers found active.

	2003	2004	2005	2006	2007
All Cankers	83.3	45.2	59.5	66.7	59.5
West-Side Plots	77.3	56.7	71.0	66.7	68.8
East-Side Plots	87.5	22.2	40.0	70.0	45.5
Branch Cankers	80.0	62.5	66.7	75.0	66.7
Stem Cankers	83.3	25.0	56.3	57.9	55.0

Table 2. Proportion of inactivity for individual blister rust cankers (2003-2007).

	Percentage of Years Canker Found Inactive										
	0%	20%	25%	33%	40%	50%	60%	66%	75%	80%	100%
No. Stem Cankers	8	1	0	1	2	3	2	1	0	2	2
No. Branch Cankers	14	2	0	0	4	3	0	1	0	1	5

Additional studies could explore the utility of this potential threshold in determining when a canker is no longer infected with inoculum. Also, half of inactive cankers failed to re-activate. Thus, classifying a tree as having blister rust based on the existence of a single canker, which appears inactive, would risk over-estimating disease incidence.

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