Long Infection Period for White Pine Blister Rust in Coastal British Columbia

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SUMMARY. For the white pine blister rust disease (WPBR), reports conflict concerning the time of year the pathogen, *Cronartium ribicola* J.C. Fisch., infects western white pine (*Pinus monticola* D. Don) and what needle age increments are most susceptible. To determine timing of infection, western white pine seedling were placed under infected currants (*Ribes nigrum* L.) for 1-week periods from May to November. Needles became spotted and stems cankered after exposure to diseased currants from early summer until leaf drop in November. To determine what foliage age increment was most susceptible, 5-year-old seedlings were placed in a disease garden, and older trees were inoculated *in situ*. All age increments of pine foliage were susceptible to infection. For young seedlings, all age increments were about equally susceptible, but on some older seedlings and trees, the current year's foliage appeared more resistant than older foliage.

Much has been written about the seasonal timing of infection and the age increment of foliage infected by the introduced WPBR pathogen on eastern white pine (*Pinus strobus* L.), western white pine, and sugar pine (*P. lambertiana* Dougl.). Hirt (1935) found teliospores germinated up to 22 °C (72 °F) and their basidiospores up to 21 °C (70 °F). Basidiospores exposed to sunlight or drying conditions survive a few hours (Hirt, 1935; Spaulding and Rathbun-Gravatt, 1926). In Wisconsin, Van Arsdel et al. (1956) found diurnal summer temperature fluctuations of 15 to 25 °C (59 to 77 °F) prevented formation of fertile teliospores, and that cooler, more stable, temperatures of September were necessary to permit viable inoculum development and infection of white pine. In eastern North America, York et al. (1927) and Hirt (1942) obtained good infection of artificially inoculated seedlings in late, but not early August. Similarly, in Idaho and Montana, copious telia were formed under fall conditions and regularly used to produce inoculum for pines (Bingham, 1983). In British Columbia, copious telia were noted in late August to early September and cool moist conditions were favorable for infecting white pines in the fall (Hunt, 1988; Lachmund, 1933; Porter, 1960). Therefore, artificial inoculations for resistance screening typically occurred in September in Wisconsin, British Columbia, Idaho, and Oregon. However, natural production of copious telia occurred in August in California (Kimmey and Wagener, 1961), July in India (Bagchee, 1950), and June on *Ribes lacustre* (Pers.) Poir. in Pacific northwestern North America (Mielke, 1943).

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The weather in coastal British Columbia in June is often cool and moist, so it is possibly conducive to basidiospore formation and white pine infection. If pine infection occurs early at the Pacific coast, it may permit the pathogen to become well established in host needles before the spots-only resistance (McDonald and Hoff, 1971) can function. This may explain why trees with this trait are resistant in the British Columbia interior (Hunt, 1994) but are susceptible at the Pacific coast (Hunt and Meagher, 1989).

Screening programs in British Columbia, Idaho, and Oregon inoculate only the current foliage of 2-year-old seedlings. However, the age of these infection courts may not be comparable to those occurring in nature. Reports are conflicting concerning which age increment of foliage is the most important infection court. For eastern white pine, Patton (1961) reported the current year's needles were more susceptible than older needles, whereas Hirt (1936) reported the reverse. Similarly for western white pine, Chapman (1934) and Snell (1936) found current year's needles susceptible while Pierson and Buchanan (1938) found current year's needles resistant. York et al. (1927) and Snell (1936) found current and 1-year-old needles to be about equally susceptible; Chapman (1934) found the 2-year-old needles to be more susceptible; and Hunt (1991) traced infection on inoculated seedlings only to current foliage.

The objectives of this study were to determine when WPBR infection occurs in coastal British Columbia and if current foliage of 2-year-old seedlings was a realistic inoculation target when screening seedling for resistance.

Methods and materials

To determine approximate timing for pine infection, *Ribes* were examined for telia when encountered in the field during the past 13 years. To determine when infection occurred, groups of potted western white pine seedlings were placed in a coastal disease garden (cultivated currants infected with *C. ribicola*) under ambient conditions at weekly intervals. In 1991, groups of 12 seedlings were placed in the garden each week from 3 Aug. to 27 Nov. In 1997, groups of 10 seedlings were placed in the garden each week from 21 June to 5 Sept. They were then removed to locations where they could not become infected (safe locations). Control seedlings were maintained in the safe locations. Infection spots were tallied the following spring and the seedlings were observed for canker production for an additional 2 years.

To determine the susceptibility of needles of various ages, 87 5-year-old seedlings were placed in a disease garden in 1992 and infection spots tallied the following summer. They were observed for cankering over the next 2 years. In natural settings on Vancouver Island (Renfrew, Cowichan and NorthWest Bay), 14 western white pines, aged 5 to 12 years, were inoculated by suspending telia-bearing *R. bracteosum* Doug. leaves on wire trays over their branches. Needle spotting and branch cankering were observed as described above.

Results

Telial columns were observed on coastal *R. bracteosum*, *R. lacustre* and *R. lobbi* Gray, sparingly or in abundance from May through to November in all years (1985-1998). Needle spotting and subsequent cankering occurred in all groups of seedling exposed for weekly intervals in 1991. In the last week, few leaves were left on the currant plants. Rain occurred in only 6 of the 11 weeks in which infection occurred. However, the least spotting occurred in late September after a month without rain. In 1997, the greatest incidence of spotting and cankering occurred in groups from the first 3 weeks (Table 1). Some of the early exposed seedlings produced cankers by the following March and most produced pycnia in the spring and summer. Control seedlings in safe locations showed no signs or symptoms of WPBR.

Inoculation of the 87 5-year-old seedlings resulted in cankers developing on both 1-year-old (1991) and current (1992) growth increments in 72% of the seedlings, on only the current growth increment in 20% of the seedlings, and on only the 1-year-old growth increment in 8% of the seedlings. Field inoculations of older trees produced variable results (Table 2). Both the Renfrew and Cowichan locations were observed several times for needle infection spots. Spots were observed only at Cowichan, but not cankers, while at Renfrew and NorthWest Bay cankers were observed, but not needle spots.

Discussion

Lachmund (1933) suggested that infection occurred in wave years (about 1 year in 3); however, Kimmey and Wagener (1961) contended that infection occurred every year. In this study, telial columns were observed sparsely or in abundance on *Ribes* under field conditions from as early as May to as late as November. The spotting and cankering data on the pines placed out for one-week intervals indicate these telial columns lead to many infection episodes over a long time each year, starting in the spring. Condensation must be sufficient for basidiospore germination as infection oc-

Table 1. White pine blister rust needle spotting and cankering on western white pine seedlings placed for weekly intervals in a disease garden in 1997.

Placement date[z]	Seedlings infected (%)	Mean spots (no.)	Cankering (%)[y]
June 21	100	88	80
June 27	100	52	100
July 4	100	52	100
July 11	100	3	30
July 18	100	6	40
July 25	60	1	10
Aug. 1	60	2	0
Aug. 8	60	1	0
Aug. 15	50	1	0
Aug. 22	60	1	0
Aug. 29	30	0	10
Sept. 5	0	0	0
Controls	0	0	0

[z]10 seedlings / week.
[y]Observations through May 1999.

Table 2. Age of branch increments bearing white pine blister rust cankers from artificial inoculations in field situations on Vancouver Island, British Columbia.

Location	Inoculation year	Tree Age (yr)	Tree No.	Canker incidence Branch increment Current	1-year	2-year
Renfrew	1989	5	3	2 (0)[z]	12 (0)	1 (0)
NorthWest Bay	1991	5-11	9	10 (0)	14 (0)	16 (0)
Cowichan	1991	12	2	0 (5)	0 (54)	0 (29)

[z]Spotting data in parentheses. Mean spot counts for 1-cm (0.39-inch) intervals on all inoculated branches; noninoculated branches lacked spots and cankers.

curred in the absence of rain. Wave infections may occur when the pathogen spreads to new areas (Mielke, 1943), but once the disease is established, multiple infection episodes per year would nullify wave years, at least in coastal British Columbia.

The spotting and cankering frequency demonstrate that some infection episodes in the early summer are more successful than fall infection episodes (Table 1). This early coastal infection may explain why spots-only resistance fails on the coast (Hunt and Meagher, 1989), but is effective in the British Columbia interior (Hunt, 1994).

The conflicting results reported in the literature (Chapman, 1934; Hirt, 1936; Patton, 1961; Pierson and Buchanan, 1938; Snell, 1936; York et al., 1927), for the age increment of foliage most susceptible may be partially explained by the age of the host. Patton (1961) inoculated seedlings and found current foliage to be highly susceptible, which parallels observations for screened seedlings (Bingham, 1983; Hunt, 1988). In contrast, we observed the current foliage to be most resistant on a few inoculated 5-year-old trees and on older trees in the field at two locations; these observations parallel those for 6- to 8-year-old plants by Chapman (1934); 4-year-old plants by Hirt (1936); and 5- to 7-year-old plants by Pierson and Buchanan (1938). At one field location all foliage increments were similarly susceptible and this observation parallels that of York et al. (1927) and Snell (1936). The reason for this variability in susceptibility of foliage increments on older plants needs further study. The observation that cankers result from infection spots only from the current foliage (Hunt, 1991) could not be repeated.

In screening for resistance to blister rust in western white pine, infection spot numbers are tallied and families with reduced needle lesion frequency (RNLF) are considered more resistant than families with high needle spotting frequencies (Hoff and McDonald, 1980). However, for this trait to be important for much of the life of the tree, RNLF of current year's foliage on seedlings must correlate to RNLF of older foliage, particularly on older trees. The variability reported in the literature, and observed here on older plants, suggests that such correlations are unlikely, and may explain why Hunt (1999) did not find any correlation between field resistance and RNLF resistance. Perhaps RNLF is only a juvenile resistance mechanism. This, combined with the variability reported here, the apparent resistance of current foliage in some cases, and the lack of a correlation with field data (Hunt, 1999) suggests that RNLF should be reevaluated as a selection trait for WPBR resistance.

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