NOTE
Forest pathology / Pathologie forestière

Effect of plant age and length of growing season on the development of blister rust cankers in western white pine

R.S. Hunt

Abstract: The hypothesis that resistance to blister rust in western white pine increases with tree age and a short growing season was tested. For a short growing season, 18 clones (40 ramets) from mature parent trees were inoculated and placed at a high-elevation site (1000 m). Resistant and susceptible seedlings (68 and 214, respectively) were also inoculated and established at the high-elevation site. After 3 years, there were cankers on the resistant (97%) and susceptible (99%) seedlings, while none of the 40 older ramets were infected. For a longer growing season, the surviving 38 ramets (17 clones) were reinoculated and maintained at a low-elevation site (50 m) and observed for canker development. After 3 years, 11 ramets (10 clones) became infected. Eight of the cankered ramets (73%) had slow-canker-growth resistance responses. These observations are consistent with the above hypothesis. There are implications for the deployment of certain resistant stocks, as the effectiveness of resistance to disease may vary with elevation and latitude as these factors influence the length of the growing season.

Key words: western white pine, blister rust, cankers, tree age, growing season, resistance to blister rust.

Résumé : Nous avons testé l'hypothèse selon laquelle la résistance du pin argenté à la rouille vésiculeuse augmente avec l'âge et lors d'une saison de végétation courte. Pour le traitement de saison courte, 18 clones (40 ramets) obtenus d'arbres parents matures furent inoculés et placés sur un site en haute altitude (1000 m). Des semis résistants et sensibles (respectivement 68 et 214) furent aussi inoculés et établis au site de haute altitude. Après 3 ans, des chancres s'étaient développés sur les semis résistants (97%) et sensibles (99%), alors qu'aucun des 40 ramets n'était infecté. Pour le traitement de saison longue, les 38 ramets survivants (17 clones) furent de nouveau inoculés et gardés à un site en basse altitude (50 m), puis surveillés pour le développement de chancres. Après 3 ans, 11 ramets (10 clones) étaient infectés. Huit des ramets affectés de chancres (73%) manifestaient des réponses de résistance du type développement lent des chancres. Ces observations sont cohérentes avec l'hypothèse énoncée plus haut. Il en résulte des implications pour l'utilisation de certains stocks résistants puisque l'efficacité de la résistance à la maladie peut varier avec l'altitude et la latitude, des facteurs qui influencent la longueur de la saison de croissance.

Mots clés : pin argenté, rouille vésiculeuse, chancres, âge des arbres, saison de croissance, résistance à la rouille vésiculeuse.

Introduction

White pine blister rust [*Cronartium ribicola* J.C. Fisch.], a disease introduced to British Columbia, causes branch and stem cankers resulting in extensive mortality to western white pine (*Pinus monticola* D. Don) (Hunt 1997a). Resistance to the disease was screened in open-pollinated seedlings originating from field selected parent trees in British Columbia (Canada) and California, Oregon, and Idaho (United States) (Hoff et al. 1973; Eramian 1999; Hunt 1999; Kitzmiller and Samman 1999; Sniezko 1999), with the latter State possessing the oldest seed orchard (Bingham 1983). This orchard is commonly used as a resistant-seed source for the British Columbia interior, because British Columbia's interior orchard is still immature. Plantations established from this Idaho source have reduced incidence of blister rust in the British Columbia interior (Hunt 2004a). For example, a 20-year-old plantation had 65% of the established trees from the Idaho orchard lacking cankers, while those established from a local British Columbia source were 100% cankered and dead. The original explanation for resistance to blister rust in the Idaho trees was mainly the premature shedding of infected needles, i.e., needles shed within 1 year of infection, which prevented *C. ribicola* from reaching the branch or stem. Forty percent of the resistant seedlings and 14% of the control seedlings appeared to

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R.S. Hunt. Pacific Forestry Centre, Natural Resources Canada, 506 West Burnside Road, Victoria, BC V8Z 1M5, Canada (e-mail: rhunt@nrcan.gc.ca).

have this resistance response after a September inoculation (Hoff et al. 1973; Hoff and McDonald 1980). In contrast, when trees originating from the Idaho seed orchard were established in low-elevation coastal trials, they were as cankered as local unselected stock (Hunt and Meagher 1989; Hunt 2004a). Because coastal British Columbia experiences wet and cool weather during spring and early summer, basidiospore production occurs and causes pine infection over a long time (Hunt and Jensen 2000). These conditions were believed to negate the premature needle-shed response, normally seen from September-only inoculations. However, thunderstorms and cool weather can also occur in the British Columbia interior, and it was recently shown that basidiospores could develop and infect pine seedlings as early as June (Hunt 2004b). Moreover, many of the seedlings inoculated in September possessed incipient stem cankers by July or August (Hunt 2004b), prior to the early needle response observed in September (Hoff et al. 1973). Tests in British Columbia could only duplicate an early needle-shedding response in stressed seedlings (Hunt 2004a) and, similarly to observations in Idaho (Hoff et al. 1973), this occurred in both resistant and susceptible genotypes. The reported high incidence of canker-free seedlings by Hoff et al. (1973) could not be duplicated (Rust 1998; Hunt 2004b), and Rust (1998) attributed the apparent high incidence of resistance in the data of Hoff et al. (1973) to a poor inoculation. In British Columbia, selection of resistant seedlings is mainly based on choosing seedlings with slow-canker-growth response, that is those showing only small active canker margins (<10% of normal), many of which will eventually heal entirely (Hunt 1997b; Hunt 2004c).

There is no apparent difference in virulence between British Columbia coastal and interior *C. ribicola* inocula (Meagher and Hunt 1999; Hunt 2004a), and there are no epidemiological differences attributable to rain or temperature between some interior and some coastal sites that could explain the lack of resistance in the Idaho seed source at low elevations of the British Columbia coast. The shorter growing season of interior sites appears to be the only environmental parameter differing between coastal and interior sites. Thus, instead of an early needle-shed response to explain the good field resistance of the Idaho stock growing in the British Columbia interior as compared with the lack of resistance in the same stock growing on the coast, it is proposed that the shorter interior growing season hinders growth of *C. ribicola* down the needle into the stem and within the stem, but only in particular, resistant genotypes, such as those found in the original Idaho seed orchard. Therefore, where the growing season is short (about 5 months), many resistant seedlings may naturally shed infected needles 3 years after inoculation and prior to *C. ribicola* reaching the stem. Where the growing season is longer (about 6 months), *C. ribicola* may reach the stem in additional resistant seedlings, and slow-canker-growth resistance responses (Hunt 1997b) may occur. Where the growing season is the longest (about 7 months), normal cankers may occur in resistant seedlings (Hunt 2004b, 2004c). This pattern of canker development is consistent with observations between low- and high-elevation coastal sites (Hunt and Meagher 1989) and among southern and northern Idaho (or interior British Columbia) sites (Schwandt and Ferguson 2003; Hunt 2004a). It is further proposed that these resistance responses increase with age (Hunt 2004c). Age-related resistance to blister rust is already known for eastern white pine (*Pinus strobus* L.) (Patton 1961).

Several studies are under way to determine the effect of growing season or age on resistance to blister rust in white pine. The objective of this study was to see if there was a difference in cankering incidence, when inoculated, mature white pine clones were incubated at a site with a short growing season (high-elevation coastal site) versus a site with a long growing season (low-elevation coastal site).

Materials and methods

In 1995, scions were taken from 18 parent trees phenotypically selected for resistance to blister rust (Hunt 2004c), and these were grafted to make clones. Previously, most of these parent trees (mean age 49 years, in 1995) were given a percentage value for slow-canker-growth responses (Table 1) based on inoculating their 2-year-old offspring (Hunt 2003, 2004c). In the results section, these values for slow canker growth of the ortets (parents) are related to canker incidence and canker type on inoculated ramets. By early September 1998, 43 ramets (2–3 per clone) were well established in 11-L pots and inoculated by placement for several weeks under *Ribes* spp. infected with *C. ribicola* at Saanichton (near Victoria, British Columbia), using methods reported by Hunt (2004a). Ninety 2-year-old seedlings originating from the Idaho seed orchard (resistant) and 275 seedlings from British Columbia sources (susceptible) were inoculated at the same time. When inoculation was complete, the ramets and seedlings were transported to a short-growing-season site (a high-elevation site at about 1000 m) north of Victoria, at a latitude approximately 48° N and a longitude 124° W. Pots were heeled in, and seedlings were planted. These were examined for canker development once or twice a year during the subsequent 3 years. In September 2001, there were 38 surviving ramets (17 clones), and none of the foliage exposed to the 1998 inoculation remained. These ramets were removed from the high-elevation site (100 m), reinoculated in the *Ribes* spp. garden at Saanichton, and held at the Pacific Forestry Centre, a long-growing-season site at about 50-m elevation. Again, the clones were examined for canker development twice annually during the next 3 years.

Results

Short-growing-season site

Many seedlings and three ramets died of drought within the first year of incubation at the short-growing-season site. All established seedlings had needle-infection spots, and most were severely cankered after 2 years. On the 40 ramets that survived, no needle-infection spots were observed, and there was no cankering after 2 years. After 3 years, 97% of 68 established Idaho seedlings and 99% of 214 established British Columbia seedlings were cankered, but none of the 40 ramets were cankered. When the ramets were moved to the Pacific Forestry Centre, two (5%) became cankered from the 1998 inoculation by 2002 (Table 1). Slow-canker-growth responses for the Idaho seedlings, for the British

Table 1. Incidence of blister rust cankers in clones of western white pine, 3 years after inoculation of the ramets with *Cronartium ribicola* and incubation at a high-elevation short-growing-season site in British Columbia, and 3 years after reinoculation of the same ramets and incubation at a low-elevation long-growing-season coastal site in British Columbia.

Clone No.	Number of ramets	Slow canker growth (%)[a]	Cankering (%)	
			Short growing season	Long growing season
1062	1	4	0	100
1108	3	5	0	332
1115	2	9	0	0
1116	1	4	0[b]	NT[c]
1119	3	8	0	67[d]
1121	2	14	0	100[d]
1124	3	9	0	0
1135	3	24	0	0
1147	3	27	0	0
1151	2	0	0	50
1155	2	10	0	50[d]
1158	2	0	0	50[d]
1186	2	23	0	0
1278	3	46	0	0
9022	2	10	0[b]	50[d]
9269	2	NT	0	0
9272	2	NT	0	100
9300	2	NT	0	100

[a]Ortet rating for slow-canker-growth response, based on previously inoculated seedlings (Hunt 2003, 2004c).
[b]No cankers developed at the high-elevation incubation site, however, cankers developed on one ramet, 1 year after ramets were moved to a low-elevation site.
[c]No testing.
[d]Slow-canker-growth response in the inoculated ramets.

Columbia seedlings, and for the clones were 7%, 1%, and 0%, respectively.

Long-growing-season site

Needle-infection spots were apparent on many ramets after 1 year of incubation, and 59% of the 17 clones (27% of the ramets) were cankered after 3 years (Table 1). Four inoculated clones had normal cankers, with all the ramets having one to several normal cankers, except for one canker-free ramet. All ramets of one clone had slow-canker-growth responses. Five clones had ramets with one or two slow-canker-growth responses and ramets that were canker free. Seven clones had only canker-free ramets; four of these (Nos. 1135, 1147, 1186, and 1278) had previously been classified with the greatest incidence of slow-canker-growth response in the parent (ortet) (Table 1). The rating for mean percentage of slow-canker-growth incidence in the ortets, if their seedlings had previously been inoculated, was 2 for the ramet group with normal cankers, 7.8 for the ramet group with slow canker growth, and 23 for the ramet group that was canker free (Table 1).

Discussion

Needle spotting was not observed on inoculated parent tree ramets, but was commonly observed on inoculated seedlings at the short-growing-season site. Thus, there was a marked reduction in cankering incidence on ramets (0%–5%) compared with seedlings (97%–99%). Cankers appeared earlier on seedlings than on ramets, thus suggesting that pathogen development slowed in older ramets. These observations are consistent with the hypothesis that resistance can increase with age.

At the short-growing-season site, resistance was greater [all clones with canker-free ramets (100%)] than it was on the same ramets at the long-growing-season site [seven clones with canker-free ramets (41%)]. This is consistent with the hypothesis that resistance increases where growing season is short. These results also agree with a previous study in which Idaho seedlings lacked infection spots and subsequently developed fewer cankers at a short-growing-season site, while a site with a longer growing season produced seedlings with more cankering and more slow-canker-growth responses (Hunt 2004a).

Data from this study support the hypothesis that the length of the growing season and the plant age explain some of the resistance responses to blister rust in western white pine. These data are also consistent with our observation that certain binding domains (reactive sites) on developmental and seasonally active proteins might have been selected for a general resistance response to disease, while maintaining their developmental and seasonal functions (Liu and Ekramoddoullah 2004; Liu et al. 2005). Consequently, in certain genotypes, for physiological reasons, these proteins may be more active with age and (or) where the growing season is short, thus causing collateral damage

to *C. ribicola*. For instance, one pathogenesis-related protein group (PR 10) increases in the presence of the pathogen and primarily increases with cold temperature (Liu et al. 2005). This protein group is thus more active when the growing season is short, however, to date, there is no direct evidence that it is responsible for the observations reported here.

The preliminary evidence, based on the long-growing-season site, that the three clonal groups (normal cankers, slow canker growth, and canker-free ramets) indicated a hierarchal response (based on the mean percentage of slow-canker-growth incidence of the ortets being 2, 7.6, and 23, respectively) warrants further investigation, as it appears that a superior parental orchard could be obtained by restricting it to parents with a high incidence of slow-canker-growth response. This may suggest that defense resistance proteins maybe in greater abundance and (or) at an earlier age in these parents.

The results here suggest that western white pine plantations at high elevations suffer less blister rust damage than those at low elevations. This would be especially true for certain genotypes selected for resistance, such as those having a slow-canker-growth resistance response. In rating sites for blister rust hazard, the length of the growing season (elevation and latitude) should be factored in, particularly when certain resistant genotypes are planted. In British Columbia, test plantations of these resistant genotypes, such as slow-canker-growth and resistant Idaho stock, should be established at contrasting elevations to find cut-off points where gene–environment interactions favour low blister rust hazard. On some sites, the hazard may be too severe to grow these genotypes, or deer protectors (Hunt 2002) or pruning (Hunt 1998) may be needed to protect the trees until they develop sufficient age-related resistance, or until nearby alternate host populations are reduced.

In summary, the present and previous data (Hunt 2004a) are consistent with the hypothesis that the length of the growing season and (or) the plant age are (is) related to increased resistance to blister rust. This resistance is manifested in at least three responses: (1) less infection spotting and subsequently less cankering; (2) more needle infections that fail to reach the stem, which results in less cankering; and (3) when cankering occurs, an increased slow-canker-growth response. These responses appear to be hierarchical, but consistency of the responses among host genotypes requires further investigation. The lack of cankering in any of the clones from ortets that produced seedlings with high frequency of slow canker growth is encouraging.

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