CROP/CULTURE: White pine (*Pinus monticola* D. Don) **LOCATION/RÉGION:** British Columbia

NAMES AND AGENCIES/ NOMS ET ÉTABLISSEMENTS:

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TITLE/TITRE: DOTHISTROMA NEEDLE BLIGHT ON WESTERN WHITE PINE IN BRITISH COLUMBIA

INTRODUCTION: Dothistroma needle blight (*Dothistroma septosporum* (Dorog.) Morelet), also known as red band needle blight, can be a devastating disease of pines (Barnes et al. 2005; Woods et al. 2005). Needles of all ages are affected, usually in the lower portion of the crown, but in more severe cases the disease can cause total defoliation and mortality. This is the case with the current *D. septosporum* outbreak in northwestern British Columbia (BC) where it is causing unprecedented mortality in plantations and mature stands of lodgepole pine (*P. contorta* var. *latifolia* Dougl. *ex.* Loud.) (Woods et al. 2005). Severe defoliation caused by *D. septosporum* has been reported in western white pine (*P. monticola* D. Don) in Idaho (Shaw and Leaphart 1960). However, in BC the disease was not reported to cause severe defoliation on *P. monticola* until 1982 when it caused up to 80% discoloration and defoliation in 15-30 year-old stands of nearly pure white pine in southeastern BC [Forest Insect and Disease Conditions (FIDS) 1982-1990]. In 1982, at the most severely affected location near Nakusp, a permanent sample plot was established by FIDS and examined for 8 years. Although severity varied annually (between 56 and 82% of needles infected), the disease was nonetheless present each year and caused substantial reduction in height and diameter (Unger and Vallentgoed 1990). Mortality resulting from repeated severe infections was reported for only one tree during the 8-year assessment (FIDS 1982-1990).

Western white pine is highly susceptible to the introduced blister rust Cronartium ribicola J.C. Fisch. in Rabh., which causes branch and stem cankers. There are several tree improvement programs to screen for rust resistance in western North America (Eramian 1999; Hunt 1999; Kitzmiller and Samman 1999; Sniezko 1999). In BC, provenance trials support broad seed transfer rules for maintaining adaptability and productivity of western white pine, such that coastal trees originating north of the Columbia River in Washington State plus interior trees can be planted on the coast. However, coastal trees are not cold hardy enough to be recommended for plantations in the BC Interior (Thomas and Lester 1992; Meagher and Hunt 1999). Thus, there are two western white pine-seed zones for BC: coastal and interior. Although a single dominant gene or major gene resistance (MGR) against blister rust is known from southern Oregon (Kinloch et al. 1999), much of the resistance seems to be ontogenetic (Sniezko et al. 2000: Hunt 2005). Mature plant resistance appears to be a common phenomenon in several host-pathogen interactions (Punithalingam and Gibson 1973; Liu and Harder 1996; Ficke et al. 2003), including against C. ribicola in both P. strobus L. (Patton 1961) and P. monticola (Hunt 2005). Selecting pines for increased ontogenetic resistance to C. ribicola may require evaluation in longer term field trials rather than inoculating and screening the more susceptible young seedlings. In BC such long term trials have recently been established (Carlson et al. 2010; King et al. 2010). A few older trials already existed and have been examined for rust incidence several times. Dothistroma needle blight has been noted in two of these trials and in other white pine plantations throughout the southern BC Interior.

Dothistroma septosporum sporulates early in the season during cool, moist weather and produces spores for a longer time than *C. ribicola* produces basidiospores. It is important to survey for *D. septosporum* because it may be: 1) able to out-compete *C. ribicola* for suitable needle infection sites, thus interfering with results of field evaluations for longer-term resistance to *C. ribicola*; 2) be so severe on trees of particular provenances that seed transfer rules will need to be modified; and 3) be severe on certain blister rust-selected trees to the point that they should be culled from orchards. The objective of the present study was to observe the symptoms and general impact of *D. septosporum* on *P. monticola* in BC and specifically on stocks selected for blister rust resistance.

METHODS: All trials examined were originally established to test field resistance to blister rust, or to demonstrate resistance, or to be used operationally to establish western white pine plantations. They were established by the Canadian Forestry Service or different agencies within the BC Forest Service. Since they were evaluated for *D. septosporum* incidence and damage by different agencies different evaluation methods were used.

Two older trials where *D. septosporum* was noted were at Pye Lake on Vancouver Island and on Texada Island, both coastal locations (Fig. 1). The Pye Lake site, planted in 1984, had two types of planting blocks: sub-block and mixed block. The sub-block type had 40 Idaho trees (rust resistant seed from the R.T. Bingham Arboretum) and 60 nonselected local trees (Sayward provenance) as adjacent sub-blocks. The mixed block type had *P. monticola* trees alternated with Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco. Nearly every other *P. monticola* originated from Idaho or was from the Sayward provenance (for more details see Hunt 1994 and 2002). *Dothistroma septosporum* was first observed in 1995. A survey for white pine blister rust and *D. septosporum* was conducted the following year. The most severely blighted trees (trees exhibiting discolouration of needles, defoliation and stem or branch lesions) were growing near a swamp and were confined to one of the sub-blocks and one of the mixed blocks. Trees with greater than 50% defoliation were recorded as severely blighted and these same blocks were re-evaluated for similar defoliation and mortality in 2001 and 2007. The two white pine sources were compared by a Chi-square test (testhttp://www.graphpad.com/quickcalcs/chisquared1.cfm).

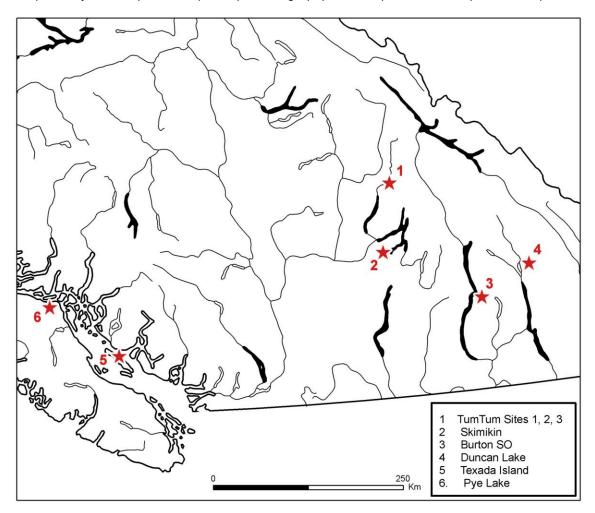


Figure 1. Map of southern British Columbia showing the location of the study sites.

The Texada Island site was planted in 1994 and consisted of four-tree-row plots replicated seven times for several coastal families, an interior family, an Idaho family, a blister rust-resistant MGR family (Washington State X Southern Oregon), and a Macedonian pine (*Pinus peuce* Grisebach) provenance from Bulgaria. Trees were evaluated for *D. septosporum* infection in 2003 by measuring the height of live crown defoliation with a tape measure and estimating the percentage of foliage discolouration as compared to noninfected foliage to create a disease index. Only samples sizes of 12 or greater were compared using the Student-Newman-Keuls multiple range test (Statistica 6.1, StatSoft Inc., 2325 E. 13th St., Tulsa, OK, 74104, USA).

Dothistroma septosporum was noted in a blister rust demonstration trial at Duncan Lake in south-eastern BC (Fig. 1). The Duncan Lake trial was established in 1998 and consists of one interior provenance in four replicate blocks (500 individuals) and six Idaho seedlots (100 to 700 individuals each) planted in one to six blocks for a total of 17 blocks of Idaho stock. The trees were evaluated in 2007 for presence or absence of stem and branch die-back and again in 2008 for defoliation by scoring each tree on the following scale: 0 = no defoliation, 1 = small amount of defoliation (<50%), and 3 = >50% defoliation. Scores were added and then averaged by block, which were in turn averaged to yield weighed scores by white pine source for the entire plantation.

Plantings using the blister rust-resistant source from Idaho (R.T. Bingham Arboretum) were established as follows: 1) Three interior plantations (Tum Tum 1, 2, and 3) in 1999 at North Adams Lake, 2) an abandoned interior seed orchard in Burton in 1988, and 3) the Skimikin seed orchard in 1980. All five sites were examined in 2007 and *D. septosporum* was identified by the characteristic black and erumpent stromata on moribund or necrotic needles bearing distinct dark brown or red banding. If found, spores were collected.

RESULTS AND COMMENTS: Where spores found (Table 1), measurements of their width matched those for *D. septosporum* (Barnes et al. 2005). Symptoms on stems and branches included fingernail-sized sunken lesions and resinosis in the bark at the base of infected needles (Fig. 2), similar to those attributed to a resistant reaction to *C. ribicola* (Bingham 1983). Frequently, the host formed a necrophylactic periderm (Mullick 1977) in the bark around the base of diseased needles that appeared as a small circular raised area on the branch or stem (Fig. 3). At some sites these small circular lesions amalgamated into a stem canker (Fig. 4) causing die-back above the canker. No fruiting was observed on any circular depressions or larger cankers and although attempts were made, no pathogens were isolated. Table 1 shows signs and symptom development on *P. monticola* caused by *D. septosporum* across several geographically distant locations in southern BC.

Table 1. Presence/absence of signs and symptoms of Dothistroma septosporum on Pinus monticola atseveral locations in British Columbia.

Site	BEC ¹ zone	Stand age (yrs)		•	Red	•	ns and sig Cankers	gns Mortality ³
Pye Lake	CWHxm2	24	yes	2.5 ⁴	yes	yes	yes	yes
Texada Island	CWHxm1	24	yes	2-2.5	yes	yes	no	no
Skimikin	IDFmw2	27	yes	2-2.5	yes	yes	no	no
Tum Tum 1	ICHdw3	9	yes	no	yes	yes	yes	no
Tum Tum 2	ICHdw3	9	yes	no	yes	yes	yes	no
Tum Tum 3	ICHdw3	9	yes	no	yes	yes	yes	no
Duncan Lake	ICHmw2	10	yes	2-2.5	yes	yes	yes	yes
Burton SO	ICHmw2	20	yes	no	yes	yes	no	no

¹Ecological zone. See Meidinger and Pojar (1991); Braumandl and Curran (1992).

²Circular depressions. ³Mortality was caused by cankering and severe defoliation.

⁴ Spore width in µm.



Figure 2. Circular stem lesion at the base of a needle cluster. Needles bear stromata of *Dothistroma* septosporum



Figure 3. (left) Small, older stem lesion caused by *Dothistroma septosporum* showing sloughing of rhytidome tissue following formation of a necrophylactic periderm.

Figure 4. (right) Small lesions of *Dothistroma septosporum* amalgamating to form a larger stem canker.

Initially, at Pye Lake (in 1996) it appeared that both *P. monticola* sources (Sayward and Idaho) were similarly infected because in the mixed planting 68% and 70% of trees exhibited severe defoliation (>50% foliage loss) among a total of 34 local (Sayward) and 29 Idaho trees, respectively. However, in the subblock type only 32% of the local provenance showed infection compared to 71% infection in the Idaho trees. Between 1996 and 2007 trees died from both blister rust and *D. septosporum*. When the blister rust-killed trees were excluded from the samples there was considerably more *D. septosporum* mortality in the Idaho source (40% mixed block and 82% sub-block) than in the local provenance (10% mixed block and 46% sub-block) (Table 2). By 2007, in the mixed block, there was significantly more mortality and defoliation among 21 Idaho trees than in 30 Sayward trees (χ 2 = 4.94; p = 0.026). More Sayward trees recovered from blighting than Idaho trees (Table 2).

At Texada Island there was considerable variation in height of live crown defoliation even in adjacent trees from the same family. The interior and Idaho sources were significantly more damaged than only one or two coastal families (Fig. 5); however, the MGR family (Oregon X Washington) was significantly more damaged than all coastal BC families (p = 0.05) (Fig. 5). Interestingly, *P. peuce* was not attacked.

At Duncan Lake in 2007, the six Idaho seedlots ranged from having 15 to 19% of the trees with branch or stem die-back compared to the BC interior population which averaged 16% of the trees with branch or stem die-back. In 2008 the defoliation score for the Idaho seedlots ranged from 1.52 to 1.99 with an average of 1.84 compared to a range of 1.36 to 1.91 and a mean of 1.67 for the BC population.

At the three Tum Tum sites near North Adams Lake, the severity of defoliation and cankering on 8-yearold Idaho-sourced P. monticola was the worst we observed (Table 1). Here, and at both Burton and Skimikin, no formal surveys were done because all trees were from Idaho sources and could not be directly compared to local stock. Only defoliation and circular lesions were observed at the latter two sites (Table 1).

Table 2. The percent *Dothistroma septosporum* infection of *Pinus monticola* trees growing at Pye Lake, British Columbia in 1996 and of these the percent infection, mortality and trees recovered in 2007 as recorded from two plantation blocks (Mixed and Sub blocked) for two sources (Idaho and Sayward)

Mixed Block				
	1996		2007	
Source	% Infection	<u>% Infection¹</u>	% Mortality	% Trees recovered ²
Idaho	70	7	40	53
Sayward	68	14	10	76
Sub Blocked				
	1996		2007	
Source	% Infection	<u>% Infection¹</u>	% Mortality	<u>% Trees recovered²</u>
Idaho	71	12	82	6
Sayward	32	38	46	15

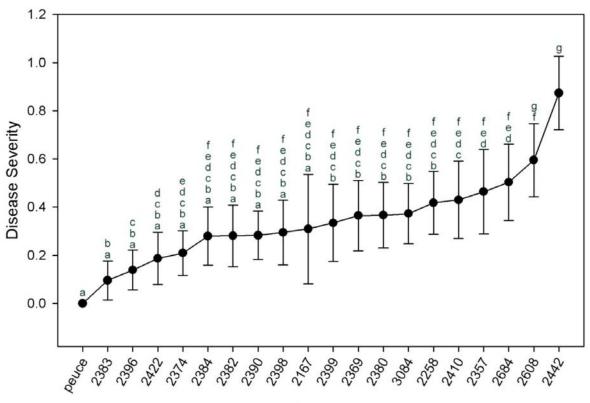
¹None of the trees that lacked initial severe infection in the mixed block suffered future *D. septosporum* mortality whereas, in the sub block, three additional Idaho trees and one additional Sayward tree died *from D. septosporum*.

²This is the % of trees that had no blighting in 2007, but were blighted in 1996.

Of the two known species of Dothistroma affecting pines, only D. septosporum is known from BC. In this study the spore widths (Table 1) confirm the designation for the pathogen of dothistroma needle blight of western white pine as *D. septosporum* (Barnes et al. 2005). For more than 10 years circular lesions, cankering and die-back associated with *D. septosporum* were noted on western white pine in BC. Significant mortality was observed at the plantation with the longest infestation (Pve Lake). However, at the three younger Tum-Tum plantations cankering was much more severe, such that considerable mortality is anticipated. Mortality caused by D. septosporum will reduce the stocking and increase risk that these stands will not achieve 'free-growing' status. This status is a licensee obligation for ensuring effective reforestation under the Forest and Range Practices Act administered by the BC Ministry of Forests and Range. The damage is in sharp contrast to earlier observations in a permanent plot established in the B.C. Interior (FIDS, 1982–1990) and in Idaho (Shaw and Leaphart 1960), where only defoliation was reported. No pathogens have been observed to fruit on the lesions or cankers, nor have they been isolated from the lesions or cankers. Small circular lesions on branches have formed around a single needle cluster, and sometimes these needles bear D. septosporum stromata. The first criterion of Koch's postulates is to have consistent association of the pathogen with the symptoms. Because this close association has been observed many times over several years, we believe the lesions and their amalgamation into cankers are caused by D. septosporum. Moreover, the authors are unaware of any example of an air-borne pathogen that meets this first criterion and where subsequent fulfilment of Koch's postulates has revealed a different causal agent. Consequently, we believe the probability that the lesions are caused by an overlooked or undescribed pathogen is remote; however, stem cankering has not been previously been attributed to D. septosporum.

Defoliation of BC Interior and Idaho stocks of western white pine at the Texada plantation ranked high, but this was significant for only a few coastal families. Obvious variation from tree to tree in the four- row plots suggests that trees displaying significantly less defoliation could be selected. It was surprising that at Pye Lake the Idaho source did not recover from severe infection as well as the local Sayward provenance, and to date, mortality has been greater among the Idaho source (Table 1). This result suggests that Idaho stocks should be used with caution on the coast, and in particular they should not be

transferred into environments conducive to *D. septosporum* infection, as even local stocks may prove unsatisfactory for restocking. The importance of local environmental conditions to mortality was apparent at Pye Lake as only two of six blocks, those near the swamp, had copious needle blighting and barks lesions. Cankering was a result of an amalgamation of adjacent bark lesions, which could occur only under intense infection. Severe infection may be reduced in high hazard environments (McCulloch and Woods 2009) by minimizing the planting of susceptible hosts. Unfortunately, the blister rust-resistant MGR trees at Texada were significantly more infected than all other provenances. The tree-to-tree variation within families in the Texada plantation suggests there is an opportunity to select for resistance to dothistroma needle blight.



Disease Severity with Multiple Range Confidence Limits

Seedlot

Figure 5. Dothistroma disease severity index with SNK multiple range test of provenances. All provenances are from coastal British Columbia except 2684 (Idaho), 2608 (British Columbia interior) and 2442 (Oregon X Washington).

Historically, dothistroma needle blight has been considered to have a relatively minor impact on endemic hosts. More recently however, an outbreak of this disease in northwestern British Columbia has decimated entire plantations of lodgepole pine and in some cases has killed even mature trees. Local increases in summer precipitation, an indirect effect of climate change, appear to be the driver behind the current outbreak (Woods et al. 2005). If precipitation increases in future, as climate models suggest for certain biogeoclimatic zones (Meidinger and Pojar 1991) across southern BC, the climate may then become more conducive for spread and severity of foliar diseases like dothistroma blight. This will have serious implications for the health of lodgepole and western white pine in BC. There is an apparent lack of infection on *P. peuce* in contrast to the severe infections observed on pines from western North

America: limber (P. flexis James), white bark (P. albicaulis Engelm.) (Taylor and Walla 1999), lodgepole (Woods et al. 2005), Monterey (P. radiata D. Don) (Punithalingam and Gibson 1973), ponderosa (P. ponderosa Laws.) (Peterson 1967) and western white. Thus, one could argue that the pathogen has been introduced from Europe. However, P. nigrum Arnold., native to Europe, is also highly susceptible (Peterson 1967; Bassett 1969) and even P. peuce has been listed as a host in Austria (Barnes et al. 2008). Genetically, D. septosporum appears to exhibit plasticity in pathogenesis because, on occasion, it has attacked conifers other than pines (Bassett 1969; Allen et al. 1996, Punithalingam and Gibson 1973), so a recent change to being a canker pathogen is a possibility. On the other hand, it is also possible that cankers have been overlooked, but were observed in our study because copious inoculum that developed on highly susceptible pines produced an abundance of symptoms. In BC, western white pine primarily exists as a minor component in mixed conifer stands (Hunt 2009). Most of the other conifers are nonhosts for D. septosporum. Mixtures with non-hosts are well-known to reduce disease damage dramatically (Wolfe 1985). However, because western white pine is fast growing, a high-value timber species (Muir and Hunt 2000), and has known resistance to Phellinus sulphurascens Pilát (formerly P. weirii (Murr.) Gilb) (Theis and Sturrock 1995), it is sometimes a favoured planting choice (Sorensen 2009). This will increase its relative abundance in a stand mixture, which could potentially augment dothistroma needle blight. Additionally, compared to many other conifers, western white pine appears to be adaptable to a wide range of biogeoclimatic zones/subzones. In the face of global warming this may favour a wider use of the species for reforestation (Hunt et al. 2009). It would also increase the relative abundance of white pine in the landscape. Our observations were that cankering caused by D. septosporum occurred only on imported western white, or local sources growing amongst these imported sources. The local sources were less damaged than the imported sources. Consequently, we encourage British Columbia tree breeding programs that use imported gene sources for blister rust resistance to test selections for resistance to *D. septosporum* too.

REFERENCES:

Allen, E.A., Morrison, D.J., and Wallis, G.W. 1996. Common tree diseases of British Columbia. Natural Resources Canada, Canadian Forest Service. Victoria, BC. 178 pp.

Barnes, I., Kirisits, T., Akulov, A., Chhetri, D.B,. Wingfield, B.D., Bulgakov, T.S., and Wingfield, M.J. 2008. New host and country records of the dothistroma needle blight pathogens from Europe and Asia. For. Path. 38:178-195.

Barnes, I., Crous, P.W., Wingfield, B.D., and Wingfield, M.J. 2005. Multigene phylogenies reveal that red band needle blight of *Pinus* is caused by two distinct species of *Dothistroma, D. septosporum* and *D. pini*. Studies in Mycology 50:551-565.

Bassett, C. 1969. Larix decidua a new host for Dothistroma pini. Plant Dis. Reptr. 53: 706.

Bingham, R.T. 1983. Blister rust resistant western white pine for the Inland Empire. USDA For. Serv. Gen. Tech. Rept. INT-146.

Braumandl, T.F., and Curran, M.P. 1992. A field guide for site identification and interpretation for the Nelson Forest Region. Land Manage. Handb. 20. B.C. Min. For., Victoria, B.C.

Carlson, M., Berger, V., and Ukrainetz, N. 2010. Production of blister rust resistant white pine seed and realized genetic gain trials in the B.C. Interior, pp 96-102. *In*: Cleary, M.R. Proceedings of the 3rd Western White Pine Management Conference; 2008 June 17-18; Vernon BC. Ministry of Forests and Range, Southern Interior Forest Region, Kamloops, BC.

Eramian, A. 1999. Blister rust resistance program for the Inland Empire. The Genetic Forest. 6:1 & 4.

Ficke, A., Gadoury, D.M., Seem, R.C., and Dry, I.B. 2003. Effects of ontogenic resistance upon establishment and growth of *Uncinula necator* on grape berries. Phytopathology 93:556-563.

Forest Insect and Disease Conditions-Nelson Forest Region 1982-1990. Can. For. Serv. Pacific For. Centre, Victoria, BC.

Hunt, R.S. 1994. Transferability of western white pine within and to British Columbia – blister rust resistance. Can. J. Plant Pathol. 16:273-278.

Hunt, R.S. 1999. Blister rust resistance program for British Columbia. The Genetic Forest: 6:8.

Hunt, R.S. 2002. Blister rust resistance in western white pine – my view. Proceedings of the 50th Western International Forest Disease Work Conference, Oct. 7-11, 2002. Powell River, BC pp. 106-110.

Hunt, R.S. 2005. Effect of plant age and length of growing season on the development of blister rust cankers in western white pine. Can. J. Plant Pathol. 27:453-457.

Hunt, R.S. 2009. History of western white pine and blister rust in British Columbia. For. Chronicle. 85: 516-520.

Hunt, R.S., Geils, B.W., and Hummer, K.E. 2010. White pines, *Ribes*, and blister rust: Integration and action. For. Pathol. 40:402-417.

King, J., Noshad, D., David, A., and Smith, J. 2010. A review of genetic approaches to the management of blister rust in white pines. For. Pathol. 40:292-313.

Kinloch, B.B., Sneizko, R.A., Barnes, G.D., and 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: 861-867.

Kitzmiller, J., and Samman, S. 1999. Blister rust resistance program for California. The Genetic Forest: 6:7-8.

Liu, J.Q., and Harder, D.E. 1996. Components of resistance in barley to stem rust: pre- and post-penetration development in seedling and adult plant leaves. Can. J. Bot. 74:1305-1312.

McCulloch, L., and Woods, A. 2009. British Columbia's northern interior forests: Dothistroma Stand Establishment Decision Aid. BC JEM. 10:1-3.

Meagher, M.D., and Hunt, R.S. 1999. The transferability of western white pine to and within British Columbia based on early survival, environmental damage, and juvenile height. West. J. Appl. For. 14: 41-47.

Meidinger, D., and Pojar, J. 1991. Ecosystems of British Columbia. B.C. Min. For., Res. Br., Victoria, B.C. Spec. Rep. Ser. No. 6.

Mullick, D.B. 1977. The non-specific nature of defense in bark and wood during wounding, insect and pathogen attack (Studies of periderm, IX). Recent advances in Phytochemistry. 11:395-441.

Muir, J.A., and Hunt R.S. 2000. Assessing potential risks of white pine blister rust on western white pine from increased cultivation of currants. HorTechnology 10:523-527.

Patton, R.F. 1961. The effect of age upon susceptibility of eastern white pine to infection by Cronartium ribicola. Phytopathology 51:429-434.

Peterson, G.W. 1967. Dothistroma needle blight of Austrian and ponderosa pines: epidemiology and control. Phytopathology 57:437-441.

Punithalingam, E., and Gibson, I.A.S. 1973. *Scirrhia pini*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 368.

Shaw, C.G., and Leaphart, C.D. 1960. Two serious foliage diseases of western white pine in the Inland Empire. Plant Dis. Reptr. 44:655-659.

Sniezko, R. 1999. Blister rust resistance program for Oregon & Washington. The Genetic Forest: 6:4 & 7.

Sniezko, R., Bower, A., and Danielson, J. 2000. A comparison of early field results of western white pine blister rust resistance in sugar pine and western white pine. HorTechnology 10:519-522.

Sorensen, J. 2009. Western white pine shaking off the rust. Truck Logger. Spring issue: 23-27.

Taylor, J.E., and Walla, J.A. 1999. First report of *Dothistroma septospora* on native limber and whitebark pine in Montana. Plant Disease. 83:590.

Thies, W.G., and Sturrock, R.N. 1995. Laminated root rot in western North America. Gen. Tech. Rep. PNW-GTR-349. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station. 32 pp.

Thomas, B.R., and Lester, D. 1992. An examination of regional, provenance, and family variation in cold hardiness of *Pinus monticola*. Can. J. For. Res. 22:1917-1922.

Woods, A., Coates, K.D., Hamann, A. 2005. Is an unprecedented dothistroma needle blight epidemic related to climate change? BioScience 55:761-769.

Unger, L., and Vallentgoed, J. 1990. Forest Insect and Disease Conditions – Nelson Forest Region. Forestry Canada, Pac. For. Centre, Victoria, BC.

Wolfe, M.S., 1985. The current status and prospects of multiline cultivars and variety mixtures for disease resistance. Annu. Rev. Phytopathol. 23:251-273.