24691

Transworld Research Network
37/661 (2), Fort P.O., Trivandrum-695 023, Kerala, India

Recent Res. Devel. Mycol., 1(2003): 73-85 ISBN: 81-7895-117-7

6 White pine blister rust

R.S. Hunt
Canadian Forestry Service, Pacific Forestry Centre, 506 W. Burnside Rd.
Victoria BC V8Z 1M5, Canada

Abstract

White pine blister rust, a fungal disease caused by Cronartium ribicola, attacks white pines and alternates to Ribes and Pedicularis spp. The pathogen originated somewhere in Asia, perhaps in association with Korean (Pinus koraiensis) and Japanese stone (P. pumila) pines. It was first collected in 1846 from the Crimea on R. nigrum, not in 1854 from Estonia, as frequently cited. C. ribicola took nearly 50 years to spread throughout Europe and for Europeans to abandon growing eastern white pine (Pinus strobus). The pathogen was accidentally exported to eastern and western North America from Europe on eastern white pine seedlings. In the early 1900s hundreds of thousands of infected seedlings were shipped to many locations throughout New England, Ontario, and Quebec. Few are aware that parts of these lots were transshipped to yet other nurseries, including some in the western United States, despite a quarantine being in place. In 1910 about 100 infected seedlings were

Correspondence/Reprint request: Dr. R.S. Hunt, Canadian Forestry Service, Pacific Forestry Centre, 506 W. Burnside Rd. Victoria BC V8Z 1M5, Canada

sent in a 1000-tree shipment from France to Vancouver, Canada. This batch of 100 trees likely formed the bulk of the C. ribicola gene pool in western North America. However, anomalies in the early spread of blister rust in the western United States can probably be attributed to lots transshipped to the west from the eastern United States.

Blister rust is continuing to spread in western North America, and it is encountering new pine hosts. Likely, several of these species will need programs to develop blister rust resistant stock if their associated ecosystems are to remain stable. This can be expedited by co-operation with established programs. However, to breed for durable resistance, the considerable variation in C. ribicola and C. ribicola-like fungi that attack white pines in Asia must be considered.

Introduction

Eastern white pine, *Pinus strobus* L., was introduced from eastern North American to Europe in 1553, but it was not widely disseminated to European gardens until after 1705, and forest plantations did not become widely established until after about 1750 (64, 92). After about 100 years of plantation forestry using eastern white pine a devastating disease started in Eastern Europe and spread westward. This was white pine blister rust caused by *Cronartium ribicola* J.C. Fisch., a fungus that required two hosts to complete its life cycle – white pines (Fig. 1) and currants (*Ribes* spp.) (Fig. 2) (Details on the life cycle can be found in Appendix 1). The mortality caused by *C. ribicola* was so severe that eastern white pine is now rarely planted in Europe. Similar devastation occurred in North America, particularly in western white pine, *P. monticola* D. Don (5).

Figure 1. Blister rust cankers result from the orange coloured mycelium of *Cronartium ribicola* growing in white pine branches.

Figure 2. Telial columns of *Cronartium ribicola* germinating to produce basidiospores (white areas) on the underside of a *Ribes* leaf.

For many years western white pine was considered commercially non-viable due to this disease. However, because of the initial high demand for white pine wood for ship's masts, and because it was easily planed and took paint uniformly in woodworking, it commanded premium prices (61, 67). Also, western white pine is silviculturally desirable because it is resistant to root diseases (67). Furthermore, it is now recognized that eastern white pine is necessary for both the protection of black bear cubs and as perching sites for birds of prey (85). Another severely impacted western species, whitebark pine, *P. albicaulis* Engelm., is an important food source for nut crackers and grizzly bears (44). Because of the economic and ecological importance of white pines, foresters, pathologists, mycologists, geneticists, and ecologists have studied blister rust with the hope of saving, or restoring, white pine ecosystems. This manuscript is a synthesis of the historical work on this species, leading up to the present where it appears that resistance programs will indeed save white pines.

Origin and spread of *Cronartium ribicola*

Finding the pathogen

Frequently, *C. ribicola* is cited as being first reported from western Russia (1856) or Estonia (1854) (28, 77), but Peterson (76) documents the first report from the Ukraine (Crimea) in 1846. Perhaps from there it first moved northward to Estonia, then it progressed towards the Atlantic as follows: Prussia (1865), Denmark (1883), and Britain (1892) (64, 77, 91). Klebahn (50) in the 1880s first took aeciospores of this pine pathogen, then named *Peridermium strobi* Kleb., and inoculated them to *R. nigrum* L. to obtain Fischer's leaf pathogen (*Cronartium ribicola* Fisch.) of currant bushes. The connection of these two previously separately named fungi was reconfirmed in the 1890s by Plowright (79). In the early 1900s Klebahn (51) made the reverse inoculation by taking currant leaves bearing basidiospores to obtain pine infection, which was

confirmed by Spaulding (89). There are several taxonomists named Fischer and there is some doubt as to which one originally described the pathogen on *R. aureum* Pursh, but the taxon is generally considered to be *Cronartium ribicola* J.C. Fischer in Rabenhost, 1872 (25). At this time *R. aureum* was recently introduced from North America, and so Europeans initially thought that the rust had been carried with it, i.e., from North American to Europe (91).

Where did the pathogen come from?

Discovering the native home of *C. ribicola* should indicate where it is genetically most variable and where its pine host has stable resistance. Mimicking this resistance in other white pine species and testing with *C. ribicola* isolates from *C. ribicola*'s native home should lead to the development of durable resistance in white pine species.

In the early 1900s four white pine species (Swiss stone pine, *P. cembra* L., Siberian pine, *P.sibirica* du Tour., Korean pine, *P. koraiensis* Siebold et Zuccarini, and Japanese stone pine, *P. pumila* von Regel) were treated as a single taxon stretching from Europe across Asia to the Pacific Ocean. Thus, Swiss stone pine, endemic to the Swiss Alps, was regarded as a disjunct population of an extensive species. Since at that time the rust was known in Siberia and Japan (91, 92), it was easy for Schellenberg (86) and others to hypothesize that the rust was native to the Alps (91). A few researchers still support this notion (59, 88). However, it is difficult to believe that plantations of *P. strobus* would remain uninfected for over 100 years when the highly susceptible black currant was planted nearby and in valleys below Swiss stone pine. It thus seems unlikely that *C. ribicola* is native to Switzerland. Moreover, surveys in1915 by Eduard Fischer (12) suggested that the rust was still spreading into northern Switzerland as if it had been introduced.

Since the first European report of *C. ribicola* was from currants in the Crimea (76), could these have become infected from spores blowing east across the Black Sea? Macedonian pine, *P. peuce* Grisebach, occurs in isolated pockets in the mountains west of the Black Sea, and black currants were cultivated in the valleys below, but *P. strobus* was not planted in southern Europe (91). Thus, it may have taken time for favourable environment conditions to occur and for *C. ribicola* to spread to northern European white pine plantations from infected Macedonian pines. Although this hypothesis for the origin of *C. ribicola* seems more plausible, it is still difficult to believe that planted black currants would not have become widely infected, thus transmitting the pathogen to eastern white pines much sooner. On the other hand, it is difficult to understand why the two European white pines, Swiss stone pine and Macedonian pine, are generally highly resistant to blister rust (88), apparently without having had selection pressure from *C. ribicola*. This certainly is in contrast to western conifer diseases including the *P. contorta* Loud. – *C. comptoniae* Arth. pathosystem in western North America where resistance appears quickly eroded without selection pressure from the pathogen (38, 39).

The closest documented source of *C. ribicola* to Europe was Siberia (41, 91), and thus it seemed possible that the rust naturally spread from there to Europe (90). Leppik (57) perpetuated the Siberian hypothesis for the origin of *C. ribicola*, but this hypothesis ignores collections that were made in India perhaps as early as 1854 (90); certainly Bagchee (1) documented *C. ribicola* in India. Carl Heimburger, formally in-charge of white pine improvement in the province of Ontario, (per comm.) postulated that Tibetan

monks could have carried a bonsai Himalayan pine, *P. wallichiana* A.B. Jackson, into Europe. The bonsai could have resulted from rust, and this may have been the initial source of infection in Europe.

Distributed somewhat between India and the three northern Asian white pines are several species of white pines in Southeast Asia. These could potentially harbour *C. ribicola* or *C. ribicola*-like fungi that attack white pines, and one of these may have been the original inoculum source for European white pines. One of the more northern of these in Sichuan, China is *P. armandi* Franchet. Aeciospores collected of a *C. ribicola*-like pathogen from this host infected only one *Ribes* sp. of six inoculated, and the local *Pedicularis* sp. was not attacked (98). Hei et al. (24) examined the nucleotide sequences of the internal transcribed spacers (ITS) of the ribosomal RNA of this rust and concluded that this was an undescribed species distinct from *C. ribicola*. This then is not the source of inoculum for *C. ribicola* in Europe. However, ITS work with the *C. ribicola* from Korean pine growing in China was concordant with North American *C. ribicola* (24). Isoenzyme patterns were also similar (7). This is interesting because the Korean pine pathogen has a pathotype that alternates to both *Ribes* spp. and *Pedicularis* spp. (99, 101), and another pathotype alternating just to *Pedicularis* spp. (93), while the European and North American pathotype of *C. ribicola* attacks *Ribes* spp., but not *Pedicularis* spp. (34, 73, 93). Thus, the *C. ribicola* found on Korean pine is not a perfect match for the initial inoculum that infected white pines in Europe. However, *C. ribicola* from Japan endemic on *P. pumila* and found on planted *P. strobus* also alternates to both *Ribes* spp. and *Pedicularis* spp., except that one collection of aeciospores from a *P. strobus* plantation could only attack *Ribes* spp. (100). DNA work has yet to be done to confirm a strong genetic relationship among Japanese collections of *C. ribicola* to those found on Korean, Siberian or European pines. Additionally, *Endocronartium sahoanum* Imazu et Kakishima, a pine to pine rust on *P. pumila*, appears morphologically related to *C. ribicola* in illustrations by Imazu and Kakishima, while *E. yamabense* (Saho et I. Takahashi) Paclt appears morphologically distinct (40).

In summary, stem rusts attacking white pines in northern Eurasia are very diverse with *Cronartium* and *Endocronartium* each having more than one species. At least one of the species, *C. ribicola*, has unique pathotypes specific for alternate hosts. Relationships of these rusts with more southern Asian rusts, such as that reported from India (1), remain to be investigated. The genetic situation with *C. ribicola* may be analogous to that of *Melampsora epitea* Kunze et Schm. where there is a patchwork of pathotypes with particular host preferences (83). The *C. ribicola* originally introduced to Europe appears to be a narrow gene pool compared to the over-all variation within the species, suggesting that *C. ribicola* went through a genetic bottleneck when it went from Asia to Europe.

Introduction of *Cronartium ribicola* to North America

Starting just before 1900 and until the establishment of the 1912 quarantine (17, 91) to prevent importation of white pines into the United States and Canada, millions of eastern white pine seedlings were imported and several hundred thousand of these were infected (90). These arrived in several eastern United States, Ontario and Quebec from Belgium, France, Germany, and the Netherlands. Importers frequently sold bulk lots to other nurseries, in adjacent states and counties, and some of these in turn sold bulk lots

further west (43). Frequently, the initial and secondary destinations of many lots were not traceable, so inspections could not be made (91). When imported lots were located, many inspected seedlings appeared free from rust and often only visibly infected pines were destroyed (91). Unseen incipient cankers could then have been disseminated on stock released for sale. These shortcomings permitted the pathogen to become well established from several different European sources. Blister rust was detected first in New York in 1906 (90) and in Canada in 1914 (17). Spaulding (91) documented its subsequent spread in the eastern United States and Haddow (20) documented its spread in Ontario.

The origin of blister rust in western North America is frequently attributed to a single importation of 1000 eastern white pines from Ussy, France to Vancouver, British Columbia in 1910 (28, 92). Based on infection levels reported by Spaulding (90), only about 100 of them may have been cankered. This limited introduction may have led to a secondary genetic bottleneck resulting in less variation in the western populations of *C. ribicola* compared to eastern North American populations (23). However, the history of rust importation into the west was more complex than is generally known. Although official records for white pine importations prior to 1910 for western Canada do not exist (19), importations did occur, for Pennington (75) reports an importation into Victoria, British Columbia prior to 1910. Because British Columbia had only a sparse population (47) imports prior to 1910 were probably very few in number. However, between 1910 and 1914 an additional 350 seedlings were imported, but their European origin and Canadian disposition are unknown (19). The earliest native pine infections were dated to 1913 (53, 75) at Thurston Bay and near the present-day ski resort of Whistler. These two sites are about 200 km and 80 km north of the original 1910 importation, respectively. Based on canker aging the next expansion in distribution occurred in about 1917 after the cankers initiated in 1913 produced spores. This was followed by another expansion in 1921 (63, 75). Eastham (11) surveyed black currants before British Columbia enacted a provincial quarantine in 1919 (10) and he did not find any *C. ribicola*. When he did find *C. ribicola* for the first time in September 1921 on black currants, it was already widespread (18), including into Washington State (43). This record correlates well with the distribution expansion recorded by Pennington (75) from dating pine cankers. *Cronartium ribicola* has been common on black currants in British Columbia ever since.

Pennington (75) rightly suggested that the wind patterns favoured spread of the rust rapidly northwestward and east from Vancouver, but not southward. Thus, an infection over 200 km south of Vancouver in the Cascade Mountains of Washington State dated to 1913 (63) seems to be an anomaly. Since dated cankers come with the proviso that their date is "the maximum possible age" (53) (i.e., the earliest possible initiation date), cankers dated to 1913 may have been initiated in 1914 or 1915. An alternate explanation for this southern infection can be found in Joy (43) who compiled records for a nursery about 90 km southwest of this site. These records demonstrate that the 1912 quarantine to prevent shipments of *P. strobus* from eastern United States to the west (91) was not working. For example, 50 seedlings came into the nursery from Illinois in 1915 and several of these died from unknown causes. A few others appear to have been imported to the same nursery in 1913 from Ohio. This information suggests that this nursery and other western nurseries could have imported infected *P. strobus* from eastern nurseries for resale in the west. In fact, this is the only logical way to explain the origin of a pine

infection dated to 1917 or 1918 in the Cascade mountains of Oregon over 500 km south of Vancouver; while at the same time the spread with the prevailing winds was only 425 km eastward (63). Moreover, 4 to 5 years later there was considerable extension of the rust in British Columbia, but still "No infection was found south of Blaine (a Washington State border town), although much more intensive scouting was carried on in Washington than in British Columbia" (75). Likely these other *C. ribicola* imports mixed with a larger gene pool of the pathogen slowly moving south from British Columbia. On the other hand, it could be argued that the paucity of southward winds from British Columbia would have permitted local populations to flourish. Regardless, about 95 years after its introduction to western North America, the rust continues to spread (13). It has not yet been documented in Mexico.

Controls

The first blister rust control was to try and prevent dissemination of infested pine stock within Europe (92). In North America the first infected *Ribes* that were found were destroyed, and later the first infected pines that were found were also destroyed (90). *Ribes* eradication grew in popularity particularly through the depression of the 1930s when cheap labour was available. On a large scale (61) it worked in only a few areas such as Maine (68) and parts of the lake states (95). However, eradication of *Ribes* (54) or *Pedicularis* (52) near and within plantations was successful because basidiospores that infect pines are frequently concentrated near their alternate hosts (46). In areas where eradication failed, dormant long-lived *Ribes* spp. seeds were stimulated to germinate by the removal of existing plants, so new plants just replaced the old. In other cases spores travelled with prevailing winds to cause infection of pines many kilometres away (35, 97). In some areas the terrain precluded *Ribes* eradication (80). Another management strategy, promoting the growth of a non-susceptible barrier between *Ribes* spp. and white pines, reduced the movement of spores between the two (46, 92, 96). Also, rapid crown closure shaded out *Ribes*, while timber harvesting in some areas made *Ribes* spp. flourish (9, 31). Cultivated black currants were found to produce a particularly high load of inoculum (94) to the point that some states in the USA have forbidden their cultivation (58, 63). There is concern that increased cultivation of currants highly susceptible to blister rust resistance could bridge eastern and western North American populations of *C. ribicola* and perhaps create new virulent genotypes from population mixing, or through increased chances for mutations, thereby threatening the production of pines already selected for blister rust resistance (23, 67).

Blister rust surveys in North America confirmed that rust incidence varies on a broad geographical scale, so areas were delineated into zones of varying hazard (16, 55, 96). It was suggested that different stock types, silvicultural treatments, and *Ribes* control would be more applicable in particular zones (21, 27, 96).

In the late 1940s the USDA Forest Service started screening chemicals for blister rust control. Two, cycloheximide and phytoactin, appeared promising (65, 66). The shared enthusiasm of one Forest Service employee and the company manufacturing the chemicals resulted in popular articles and publicity. As a result, many trees were treated, even aerially (15). Later trials by other forest pathologists could not duplicate the original results, and it was suggested that secondary organisms naturally invading the cankers had confounded the original data (56). No promising fungicides were found in

trials conducted in the 1960s and 1970s (37, 82). Currently, triadimefon is the most promising fungicide to control blister rust (2, 42).

Silvicultural controls included culling of cankered white pines, pruning cankers, removing lower branches, scribing cankers, scribing the stem at the bases of branches when branch cankers were close to the trunk, and maintaining high stocking density to exclude basidiospores (21, 22, 32, 45, 62). On some sites trees were attacked when young and they needed to be pruned shortly after establishment, and frequently they needed to be pruned two or three times. On sites where cankers were initiated in the stem or were too close to the stem for effective pruning and scribing, solid plastic deer protectors showed promise in protecting trees until they were large enough to be successfully pruned (30).

Two approaches have been used to develop blister rust resistance in white pines: 1) inter-specific hybridization, and 2) selection within a pine species for resistant families or individuals. The first approach has been used for Macedonia (4) and eastern (8, 70) pines, and the second for western (3), sugar, (*P. lambertiana* Doug.) (48), Korean (52), white-barked (60) and eastern white pines (72). In the latter approach, the first resistant trees were chosen from among grafted clones of phenotypically resistant forest selections that had been inoculated (81, 84). Unfortunately, many of these selections produced seedlings that were susceptible to rust because the parents had become resistant with age (69, 71). Bingham (3) and Patton (72) initiated programs to screen seedlings for blister rust resistance, programs that were copied in Oregon (87), California (48) and British Columbia (29). These programs have incorporated two types of resistance: 1) ontogenetic, which is not race specific, increases with host age, is modified by the environment, and may be dosage dependent (Fig. 3) (29); and 2) R-genes, or single gene resistance traits, which are race specific (49). Single gene resistance to *C. ribicola* is manifested in the needles (Fig. 4) during initial infection (74).

The future

Surveys will need to be carried out to determine the range extensions of *C. ribicola* into new geographic regions (13) and to determine the incidence of infection on new pine hosts. Surveys where the pathogen causes cankers on a high proportion of pines will be needed to document potential losses in order to justify resistance selection programs. These programs are expensive, so a co-operative effort will be necessary. Networking to take advantage of the experience and facilities of existing resistance programs should be encouraged. If resistance programs are not supported, biodiversity and valuable timber species will be lost.

White pines need to be bred and selected for durable resistance, perhaps by pyramiding single R-genes into individuals with ontogenetic resistance. Since a single R-gene may mask other resistance traits, one approach is to develop panels of *C. ribicola* pathotypes (races) for testing against white pine selections. Another is to develop probes that identify masked traits. These approaches are compatible, so both should be pursued. Environmental limitations and inheritance patterns of ontogenetic resistance need to be established. More information on the variability of *C. ribicola* throughout Asia is required. This information is necessary to strengthen quarantines and to indicate where resistant white pine selections should be tested for their stability to resist attack. Pines

Figure 3. A slowly growing *Cronartium ribicola* canker in an inoculated western white pine seedling. The orange margin of fungus growth is small to non-existent. "Slow-canker growth" is a form of ontogenetic resistance.

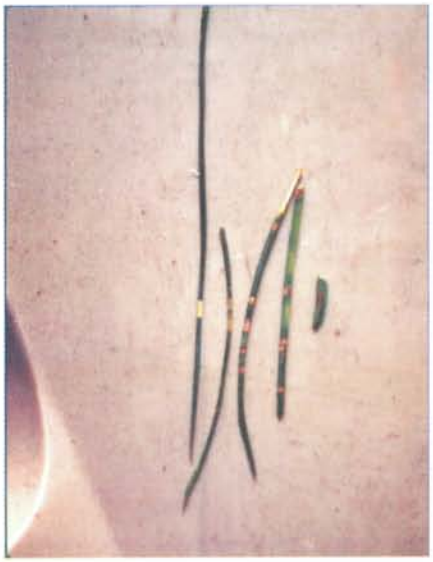


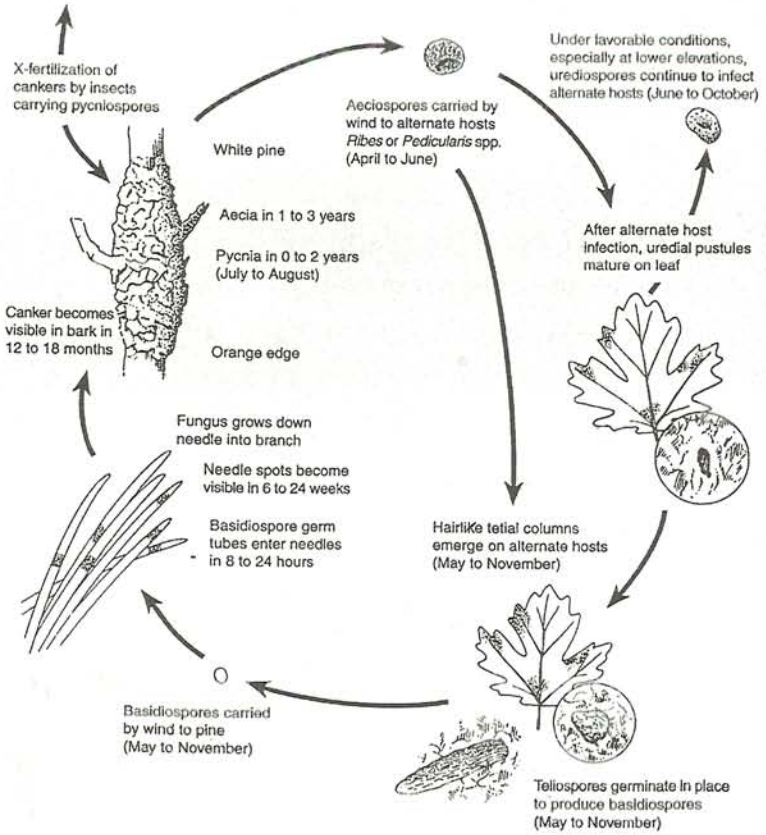
Figure 4. A contrast between normal blister rust infection spotting (yellow and orange banded spots on left two needles) and hypersensitive spots (non-banded spots with marginal necrosis on right three needles) caused by a single dominant gene for resistance in western white pine.

native to the natural range of *C. ribicola* could be potential sources of resistant genes and their resistance mechanisms could suggest new strategies for developing durable resistance.

Appendix I

The life cycle of *Cronartium ribicola*

Basidiospores are haploid and cause a perennial infection in pines. Spermatia (pycniospores) are produced annually on the periphery of pine cankers and insects, particularly flies, transfer these among cankers (6, 33). This results in an n + n, or a dikaryon state (78), but only within basal "2-legged cells" (26). The remainder of the infection remains haploid. Markers indicate that outcrossing is random (14). In the spring the 2-legged dikaryotic cells produce dikaryotic aeciospores. Dikaryotic spores are wind-borne and can only infect alternate hosts, such as *Ribes* spp. Alternate hosts produce dikaryotic urediniospores and/or diploid teliosopores (36, 63). Urediniospores intensify the infection on alternate hosts; teliospores are produced *en masse* in hair-like strands (Fig. 2) and germinate *in situ* followed by meiosis to produce four basidiospores. Basidiospores are carried by wind to pine hosts. Infection of alternate hosts ceases with leaf fall.



References

1. Bagchee, K.D. 1950, Indian Forest Record (NS) Botany , Gov. Press, New Delhi.
2. Berube, J.A. 1996, For. Chron. 72, 637-638.
3. Bingham, R.T. 1983, USDA, For. Serv. Gen. Tech. Rept. INT-146.
4. Blada, I., and Popescu, F. 2004, IUFRO White Pines (Medford OR), USDA FS RMRS-P-32, 230-242.
5. Buchahan, T.S. 1938, J. For., 36, 321-328.
6. Buller, A.H.R. 1950, Researches on fungi, vol. 7, Univ. of Toronto Press.
7. Cheng, D.S., Xue, Y., and Shao, L.P. 1995, IUFRO Rusts of pines (Tsukuba, Japan), S. Kaneko, K., Katsuya, M, Kakishima and Y. Ono (Eds.), Proc. 4: 71-75.
8. Daoust, G., and Beaulieu, J. 2004, IUFRO White Pines (Medford OR), USDA FS RMRS-P-32, 3-11.
9. Davis, K.P., and Moss, V.D. 1940, Nor. Rocky Mt. Exp. Stat., Stat. Pap. No. 3.
10. Eastham, J.W. 1922/3, The Agric. J. (B.C.), 7, 57,64.
11. Eastham, J.W. 1950, Can. Phytopath., News 2(2), 1-6.
12. Fischer, E. 1916, Mitt. Naturf. Gesell. Bern, 1915, p. 33.*
13. Geils, B., Conklin, D., Frank, K., Guyon, J., Harris, J.L., Hoffman, J., Jacobi, W., Kearns, H., Newcomb, M., Smith, E., Van Arsdel, E., and Vogler, D. 2003, West. Intern. For. Dis. Wk. Conf. 50, 94-99.
14. Gitzendanner, M.A., White, E.E., Foord, B.M., Dupper, G.E., Hodgskiss, P.D., and Kinloch, B.B. 1966, Can. J. Bot. 74,1852-1859.
15. Glew, D.R., and Macleod, R.T. 1963, BC For. Serv. For. Man. Div. No. 2.
16. Gross, H.L. 1985, White pine sym. Suppl, Proc. Ent. Soc. ON., 116, 73-79.
17. Güssow, H.T. 1916, Bull. Can. Foresrty Assoc., 7 pp.
18. Güssow, H.T. 1922, Dept. Agric. Dom. Expt. Farms, Ottawa Canada, p 6-7.
19. Güssow, H.T. 1923, Dept. Agric.Dom. Expt. Farms, Ottawa Canada, p 6-7.
20. Haddow, W.R. 1969, ON Dept. Lands & For. Res., Rept. No. 86.
21. Hagle, S.K., McDonald, G.I., and Norby, E.A. 1989, USDA For. Serv. Gen. Tech. Rept. INT-261.
22. Hayes, G.L., and Stein, W.I. 1957, USDA, For. Serv., PNW For. & Range Exp. Stat. Res Note No. 151.
23. Hamelin, R.C., Hunt, R.S., Geils, B.W., Jensen, G.D., Jacobi, V., and Lecours, N. 2000, Phytopathology, 90, 1073-1078.
24. Hei, W., Hou, L-B., Liu, X-Y., and Yang, Z-Z. 2004, IUFRO Rusts of For. Trees (Yangling, China) For. Res. (Suppl.)16, 146-152.
25. Hirasuka, Y. 1995, IUFRO Rusts of pines (Tsukuba, Japan), S. Kaneko, K. Katsuya, M. Kakishima and Y. Ono (Eds.), Proc. 4, 77-80.
26. Hirt, R.R. 1964, NY Coll. For. Tech. Pub. 86.
27. Hodge, J.C., Juzwik, J., Gross, H.L., and Retnakaran, A. 1989, Min. Nat. Resources, ON.
28. Hummer, K.E. 2000, HorTechnology, 10, 515-517.
29. Hunt, R.S. 2004, Can. For. Serv. BC-X-397.
30. Hunt, R.S. 2002, Can. J. Plant Pathol., 24, 74-76.
31. Hunt, RS. 2000, West. J. Appl. For. 15:38-39.
32. Hunt, R.S. 1998, West. J. Appl. Forestry, 13, 60-63.
33. Hunt, R.S. 1985, Can. J. Bot. 63, 086-1088.
34. Hunt, R.S. 1984, Can. J. Bot., 62, 2523-2524.
35. Hunt, R.S. 1983, For. Chron. 59, 30-33.
36. Hunt, R.S., and Jensen, G.D. 2000, HorTechnology 10, 530-532.
37. Hunt, R.S., and Ilnytzky, S. 1979, Fungicide and Nematicide Tests, 34, 130.
38. Hunt, R.S., and Van Sickle, G.A. 1984, Can. J. For. Res. 14, 672-675.

39. Hunt, R.S., and Ying, C.C. 2004, Integrating scales of forest pathology, J.E. Lundquist and R.C. Hamelin (Eds.), APS Press. *In press.*
40. Imazu, M., and Kakishima, M. 1995, IUFRO Rusts of pines (Tsukuba Japan), S. Kaneko, K. Katsuya, M. Kakishima and Y. Ono (Eds.), Proc. 4, 27-36.
41. Jing, Y., Li, W.H., and Zhao, S.G. 1995, IUFRO Rusts of pines (Tsukuba, Japan), S. Kaneko, K. Katsuya, M. Kakishima and Y. Ono (Eds.), Proc. 4, 37-41.
42. Johnson, D.R., Kinloch, B.B., and McCain, A.H. 1992, Tree Planters Notes, 34, 7-10.
43. Joy, E.L. 1939, Typewritten, USDA, Forest Service, Spokane, WA.
44. Kendall, K.A., and Arno, S.F. 1990, USDA, For. Serv. Gen. Tech. Rept. INT-270, 264-272.
45. Kim, H.J., and Yi, C.K. 1988, Res. Rept. Res. Inst. (Seoul) No. 36.
46. Kimmey, J.W., and Wagener, W.W. 1961, USDA For. Serv. Tech. Bull. No. 1251.
47. King, J.N., and Hunt, R.S. 2004, IUFRO White Pines (Medford OR), USDA FS RMRS-P-32, 12-19.
48. Kinloch, B.B., and Byler, J.W. 1981, Phytopathology 71, 386-391.
49. Kinloch, B.B., and Dupper, G.E. 2002, Phytopathology 92, 278-280.
50. Klebahn, H. 1888, Ber. Deut.Bot. Gesell., 6, 45-55.*
51. Klebahn, H. 1905, Pflanzenkrank Z., 15, 65-108.*
52. La, Y.J., and Yi, C.K. 1995, IUFRO Rusts of pines (Tsukuba, Japan), S. Kaneko, K. Katsuya, M. Kakishima and Y. Ono (Eds.), Proc. 4, 171-175.
53. Lachmund, H.G. 1933, J. Agric. Res., 46, 675-693.
54. Laflamme, G., Blais, R., and Desgagne, D. 1998, IUFRO Rusts of For. Trees, (Saariselkä, Finland), Jalkanen et al. (Eds.) Proc. 1, 123-130.
55. Lavallee, A. 1986, Can. For. Serv. Info. Lflet LFC 23F.
56. Leaphart, C.D., and Wicker, E.F. 1968, Plant Dis. Reptr., 52, 6-10.
57. Leppik, E.E. 1970, Ann. Rev. Phytopath., 8, 323-344.
58. McKay, S. 2000, HorTechnology 10, 562-564.
59. Macek, J. Z. 1976, Gozdarski Vestnik, 34, 33-39.
60. Mahalovich, M.F. 2004, IUFRO White Pines (Medford OR), USDA FS RMRS-P-32, 181-187.
61. Maloy, O.C. 1997, Ann. Rev. Phytopath. 35, 87-109.
62. Martin, J.F., and Gravatt, G.F. 1942, USDA, Farm. Bull. No. 1885.
63. Mielke, J.L. 1943, Yale School of For. Bull. 52.
64. Moir, W.S. 1924, USDA Bull. 1186.
65. Moss, V.D. 1961, For. Sci. 7, 380-396.
66. Moss, V.D., Viche, H.J., and Klomparens, W. 1960, , J. For., 58, 691-695.
67. Muir, J.A., and Hunt, R.S. 2000, HorTechnology 10, 523-527.
68. Ostrofsky, W.D., Rumpf, T., and Struble, D. 1987, Phytopathology 77,1617.
69. Patton, R.F. 1961, Phytopathology 51, 429-434.
70. Patton, R.F. 1966, Breeding pest-resistant trees, Gerhold, H.D. et al. (Ed.), Pergamon Press, Oxford.
71. Patton, R.F. 1967, IUFRO XIV Congress (Section 22), Munich, Proc. 876-890.
72. Patton, R.F. 1972, USDA For. Serv. Misc. Publ. 1221, 373-385.
73. Patton, R.F., and Spear, R.N. 1989, Phytopathology 79, 539-547.
74. Patton, R.F., and Johnson, D.W. 1970, Phytopathology 60, 977-982.
75. Pennington, L.H. 1925, J. Agric. Res., 30, 598-607.
76. Peterson, R.S. 1973, Rept. Tottori Mycol. Inst. Japan, 10, 203-223.
77. Phillips, D. H., and D.A. Burdekin. 1982, Diseases of Forest and Ornamental Trees. MacMillian, London, 435 pp.
78. Pierson, R. K. 1933, Nature 131(165), 728-729.
79. Plowright, C.B. 1899, Gard. Chron., 26, 94.*
80. Porter, W.A. 1948, Report on the condition of white pine in the Mount Revelstoke area, B.C., Can. Dept. Agric., Lab. For. Path., Victoria, BC (typed).

81. Porter, W.A. 1960, Can. Dep. Agr., Lab. For. Pathol., Victoria, BC, Canada.
82. Quick, C.P. 1967, USDA, For. Serv. Res. Note PSW-149.
83. Ramstedt, M. 1998, IUFRO Rusts of For. Trees, (Saariselkä, Finland), Jalkanen et al. (Eds.) Proc. 1, 123-130.
84. Riker, A.J., Kouba, T.F., Brener, W.H., and Byam, L.E. 1943, J. For. 41, 753-760.
85. Rogers, L.L., and Linquist, E.L. 1992, White pine symposium, Duluth MN R.A.Stine, and M. J. Baughman (Eds.) Proc. 39-43.
86. Schellenberg, D.H.C. 1904, Naturw. Z., 2, 233-241.*
87. Sniezko, R.A. 2003, IUFRO Rusts of For. Trees (Yangling, China) For. Res. (Suppl.)16, 101-112.
88. Søegaard, B.F. 1972, USDA For. Serv. Misc. Publ. 1221, 233-239.
89. Spaulding, P. 1912, Science, 35, 146-147.
90. Spaulding, P. 1914, USDA Bull. 116.
91. Spaulding, P. 1922, USDA Bull. 957.
92. Spaulding, P. 1929, USDA, Tech. Bull. No. 87.
93. Stephan, B.R., and Hyun, S.K. 1983, J. Plant Dis. Prot., 90, 670-678.
94. Taylor, M.W. 1922, Phytopathology, 12, 298-300.
95. Van Arsdel, E.P. 1968, Intn. Congr. Plant Pathol., London, 1, 207.
96. Van Arsdel, E.P. 1961, USDA For. Serv. Lake States For. Exp. Stat. Pap. No. 82.
97. Van Arsdel, E.P., Riker, A.J., Kouba, T.F., Suomi V.E., and Bryson, R.A. 1961, USDA For. Serv. Lake States Stat. Pap. No. 87.
98. Yang, Z-Z. 2003, IUFRO Rusts of For. Trees (Yangling, China) For. Res. (Suppl.)16, 99-100.
99. Yi, C.K., and La, Y.J. 1974, For. Res. Inst., Seoul S. Korea Res. Rept., 21, 207-213.
100. Yokota, S., and Uozumi, T. 1976, World For. Congr., Oslo, Norway, IUFRO, 16, 2, 330-343.
101. Yokota, S., Uozumi, T., Edo, K., and Matsuzaki, S. 1975, Plant Dis. Reptr., 59, 419-422.

* original not seen.