Proceedings of the 51st Annual Western International Forest Disease Work Conference

Riverside Inn Conference Center
Grants Pass, Oregon
August 18 to 22, 2003

Compiled by:

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Papers are formatted and with minor editing for language and style, but otherwise printed as submitted. Authors are responsible for content.

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WIFDWC 2003 Program

Western International Forest Disease Work Conference
August 18–22, 2003; Grants Pass, Oregon

Monday, August 18

12:30–5:30 **Nursery Pathology Workshop and Tour**
J. Herbert Stone Nursery, Central Point, OR. Katy Marshall, Chair

2:00–4:00 **Root Disease Model Workshop/Demonstration**
Riverside Inn Conference Center, Blue Heron Room. Fred Peet, CFS PFC, Victoria, BC.

4:00–7:00 **Registration. Riverside Inn Conference Center, Lobby**
7:00–10:00 **No-host Social, Riverside Inn Conference Center, Blue Heron Room**

Tuesday, August 19

7:00–8:30 **Rust Committee Breakfast (Otter Room). Brian Geils, Acting Chair**
Registration
8:30–9:00 **Conference Opening Ceremony (Blue Heron Room)**
Welcome to Rogue-Siskiyou National Forests. Greg Clevenger, Resources Staff Officer

Chair's opening remarks. Everett Hansen

9:00–9:30 **Keynote Presentation: Nature Notes for the State of Jefferson**
Dr. Frank Lang, Emeritus Professor of Biology, Southern Oregon University

9:30–10:00 **Refreshment Break**
10:00–1130 **Regional Reports**
11:30–1:00 **Hazard Tree Committee Lunch (Otter Room)**
Riverside Inn Conference Center, Otter Room. John Pronos, Chair

1:00–3:00 **Panel: A new tradition, What is timely in forest pathology, 2003. Bart van der Kamp, Chair**
A new home for *Cryptococcus neoformans* var. *gattii* in a Canadian temperate climatic zone. Karen Bartlett, School of Occupational and Environmental Hygiene, University of British Columbia, Vancouver, BC.

An update on Swiss needle cast in western Oregon. Alan Kanaskie, Oregon, Department of Forestry, Salem, OR.

Dothistroma in northwest British Columbia: Why there? Why now? Alex Woods, BC Ministry of Forests, Smithers, BC.

The latest of SOD. David Rizzo, University of California, Davis, Department of Plant Biology, Davis, CA.

3:00–3:30 **Refreshment Break**

3:30–5:00 **Special Papers. James Worrall, Moderator**

Early thinning in mixed-species plantations of Douglas-fir, hemlock, true fir and ponderosa pine affected by Armillaria root disease in central Oregon and Washington: 20 to 30 year results. Gregory M. Filip (presenter), Pacific Northwest Region, Portland, OR; Lisa Ganio, Oregon State University, Corvallis; and Stephen Fitzgerald, Oregon State University, Redmond.

Shore pine dwarf mistletoe: Should it be classified as a subspecies or a race of hemlock dwarf mistletoe? Ed F. Wass, Pacific Forestry Centre, Victoria, BC and Robert L. Mathiasen (presenter), Northern Arizona University, Flagstaff, AZ.

Long-term monitoring of tree damage caused by porcupine feeding in the Khutzeymateen Inlet. Stefan Zeglen (presenter) and Alex Woods, BC Ministry of Forests, respectively Nanaimo and Smithers, BC.

Fire and dwarf mistletoe. Robert Tinnin, Portland State University, Portland, OR.

Armillaria root disease in campgrounds of southern Colorado. Jim Worrall (presenter), Kelly Sullivan, Rocky Mountain Region, respectively Gunnison and Lakewood, CO; Tom Harrington and Joe Steimel, Iowa State University, Ames, IA.

5:00–7:00 **Poster Session Setup**
7:00+ **Poster Session and Ice Cream Social. Kelly Sullivan, Moderator**

Wednesday, August 20

7:00–8:30 **Dwarf Mistletoe Committee Breakfast (Otter Room). Katy Marshall, Chair**
8:30–5:30 **Field Trip: Rogue Rive National Forest, Prospect District and Crater Lake National Park**
6:30+ **A gathering at the Goheens' Place**

Thursday, August 21

8:00–10:00 **Panel: Quarantines and problematic genetic exchange. Susan Frankel, Moderator**

History of forest disease quarantines in the United States. Borys Tkacz, National Program Manager, Forest Health Monitoring, Arlington, VA.

The regulatory system for forest pathogens in North America, the European Union, and other parts of the world: a pop quiz. Susan Frankel, Plant Pathologist, Pacific Southwest Region, Forest Health Protection, Vallejo, CA.

Phytophthora ramorum quarantine: Challenges of regulating a new organism with a wide host range. David Rizzo, Associate Professor, UC Davis, CA.

Preventing exotic pathogen threats to forests—a sideways scientific look. Clive Brasier, Pathology Branch, Forest Research, Farnham, Surrey, UK.

Current challenges in forest pathogen protection. Faith T. Campbell, Director, Invasive Species Program, American Lands Alliance, Washington, DC.

10:00–10:30 **Refreshment Break**
10:30–12:00 **Business Meeting. Bart van der Kamp, Acting Chair for Everett Hansen**
12:00–12:30 **Lunch and load buses for field trip**
12:30–6:00 **Field Trip: Siskiyou National Forest, Two River District**
6:00–7:00 **No-host Social Hour. Weasku Inn**

7:00–9:30 **Banquet**
9:30+ **Buses return to Riverside Inn**

Friday August 22

7:00–8:30 **Root Disease Committee Breakfast. Katy Marshall, Acting Chair**
8:30–10:20 **Panel: Mechanisms and inheritance of disease resistance in forest trees. Det Vogler, Moderator**

Resistance to white pine blister rust in North American five-needle pines and Ribes and its implications. Paul J. Zambino (presenter) and Geral I. McDonald, Rocky Mountain Research Station, Moscow, ID.

Genetic resistance in Port-Orford-Cedar to the non-native root rot pathogen *Phytophthora lateralis*—2003 update. Richard Sniezko, Dorena Genetics Resource Center, Cottage Grove, OR.

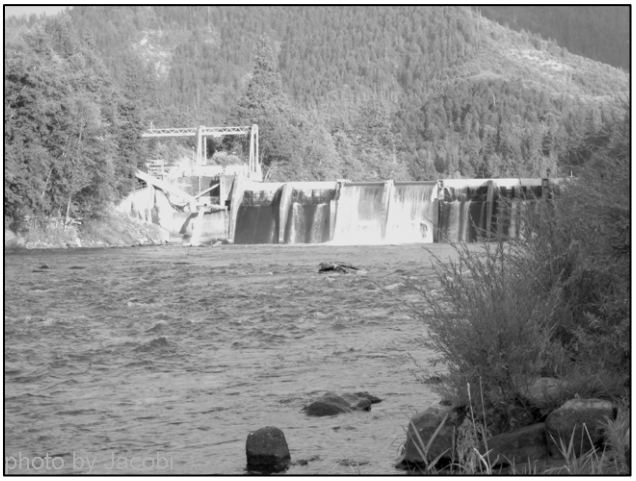
Need for studying both host and pathogen in gene-for-gene systems. Thomas L. Kubisiak (presenter), C. Dana Nelson, Southern Institute of Forest Genetics, Saucier MS and Henry V. Amerson, North Carolina State University, Raleigh, NC.

Inherent and induced resistance to pitch canker in *Pinus radiata*. Thomas R. Gordon (presenter) and Christopher J. Friel, University of California, Davis, CA.

Resistance of pines to dwarf mistletoe. Robert F. Scharpf (retired) Pacific Southwest Research Station, Placerville, CA.

10:40–11:10 **Endnote Presentation: Climate change and vegetation response modeling**
Ron Neilson, USDA Forest Service, Pacific Northwest Research Station, Corvallis, OR

11:10–12:00 **Discussion and closing ceremony**



Mistletoes of the State of Jefferson

The forests of southern Oregon and northern California contain a large and diverse flora with many plants endemic to the region. Before the workshop, Pete Angwin, Don Goheen, Ellen Goheen, Katy Marshall, Bob Mathiasen, Dave Schultz, and Bob Schroeter collected all 12 taxa of *Arceuthobium*, a *Phoradendron*, a *Gymnosporangium*, and 17 of the 24 conifers native to the State of Jefferson. These specimens were presented at the workshop socials as an identification quiz. Eun Sung Oh took third place (a myrtlewood cutting board—good work); Bob Scharpf got second place and a bottle of Druid Fluid from Troon Vineyards of Applegate, Oregon. The combined talents of Jim Worrall, Diane Hildebrand, Mary Lou Fairweather, Bill Woodruff, Dave Schultz, and Angel Saavedra were required to improve on Bob Scharpf's score; the super team won a Tower of Treats from Harry and David.

Between the papers, posters, and other sections of the proceedings are scattered a series of sidebars with descriptions of the Jefferson mistletoes adapted from information published in the Mistletoes of North American Conifers (General Technical Report RMRS-GTR-98.)

Chair's Opening Remarks

Everett Hansen

Welcome to the 51st annual WIFDWC. A very special welcome to very emeritus WIFDWCers Lew Roth, Don Graham, and Gene Van Arsdel, as well as our guests from Britain, Clive Brasier (barely emeritus), Joan Webber, and Christina, as well as Alina Greslebin visiting from Argentina.

I'm not high on the "State of Jefferson" bit, but this part of the west is different. It is lots warmer than BC, but certainly not California; and not really wanting to be Oregon either. Apart from an historical chip on their shoulder, however, it is hard to pinpoint what holds the place together. Surely it is more than a few plastic bears on the street corners. This area stands out for its diversity as much as anything. Prof Frank Lang has given us a convincing introduction to the geological and botanical uniqueness of the Klamath-Siskiyou region. Last night we were introduced to the pathological diversity of the region as well. Thanks to Katy Marshall, Don and Ellen Goheen, and the continuing tutelage of Bob Mathiason for the dwarf mistletoe quiz.

The area is also known for its human diversity. Long-haired diehard Deadheads mix with short-haired bear baiters living the past in log stockades, curly-haired "let's get down to business" women, and the matron* that keeps them in line. And that's just the Forest Service Port-Orford-cedar root disease management team.

With this diversity of people comes a diverse array of public opinion, much of it focused on forest management. Actually, you only hear two opinions down here: "cut it all, now" and "don't you dare cut a thing." The many other views are drowned out in the acrimony. The public dialogue between these two world views is very civilized—in the courtroom. In the woods or the Supervisor's office, it is considerably more shrill. Here as elsewhere, the federal foresters are caught in the political middle, always one President behind in their management direction. Those that took in the tour of the Forest Service J. Herbert Stone nursery yesterday saw this first hand. Could you find any trees in the nursery, among the wild grasses, native brush seedlings, and sugar beets grown to pay the bills? What a change.

Despite the political and environmental quagmire, a remarkable team of forest pathologists is asserting itself in this part of the west. Across the decades of my career, I have watched the center of the forest pathological universe shift around the west (it has always stayed in the West!). I was by turns jealous of the fun-filled teamwork of the California group from the University, the state, and the branches of the Forest Service, then the hugely productive collaboration that grew in British Columbia. More recently, the Montana crowd seemed to have everything going for it. Now, however, I propose that the pathological center of the universe is here in Oregon. I cite Sudden Oak Death, Swiss needle cast, and cedar root rot for evidence.

The Oregon Invasive Species Council bestowed their "Ten Fingers in the Dike" award on the Oregon SOD team this year, recognizing the massive effort that the pathologists from the Oregon Departments of Forestry and Agriculture, Oregon State University, and the Forest Service are making to contain *Phytophthora ramorum* in this state. We are proving, again and again, that eradication is a process, not an end point. To paraphrase the words of Commander-In-Chief Bush speaking of Iraq, "We will not be bound by an artificial timetable in achieving our goals." Biological frustrations aside, this teamwork has been one of the most satisfying parts of my career. Another highlight is the Port-Orford-cedar root disease management program of the Forest Service and the BLM. This is my nominee for the most successful, against the longest odds, sustained forest disease management program on the continent. The Swiss Needle Cast Research Cooperative is my third exhibit. Although this program is based at Oregon State University, (Greg Filip is Director and Jeff Stone is principle mycologist) and the Oregon Department of Forestry provides most of the legwork and plane time, the heart of the Coop lies in industry. It was initiated and continues to be funded by the forest industry, and the industry continues to set its direction. You will hear more of each of these programs through the course of this meeting.

The chairmanship of WIFDWC has always been an honorary and ceremonial position, with the real work done by the other officers. I, however, have set a new standard of insignificance. From my absence when the electoral railroad stopped in Powell River, through my invisibility during the planning stages of this meeting, and culminating in my early departure from Grants Pass, I have made little impact on the dramatic success of the 51st WIFDWC. Thank you, thank you, thank you, to all those that did the real work: Hadrian Merler program chair, Brian Geils secretary, John Schwandt treasurer for life, and of course the local arrangements team headed by Ellen Goheen. Once more, thank you.

*It was rather vigorously suggested to me that "matron" was perhaps not the most sensitive word choice. I was thinking of parallels with "prison warden" or "head nurse," a necessary authority figure. Certainly, no sexist or ageist connotations were seriously intended.

Is it mistletoe?

Witches' brooms are not only caused by mistletoes and not all mistletoes cause brooms. A witches' broom of incense-cedar is caused by the rust fungus *Gymnosporangium librocedi*. These brooms are bushy and somewhat erect; the telia are borne on the scale-like leaves of incense-cedar (smells like a pencil) as small red cushions which mature to yellow gelatinous globs. (see Diseases of Pacific Coast Conifers, Agriculture Handbook 521).

Panel: A New Tradition, What is Timely in Forest Pathology, 2003

Bart van der Kamp, Moderator

Welcome to this afternoon's panel

This is the 15th WIFDWC meeting I have attended over a span of some 35 years, and I dare say that in almost everyone of them there have been panels on root diseases, rusts, and/or dwarf mistletoes. A veritable tradition indeed. A look at today's titles might, at first glance, suggest that you have stumbled into the wrong room. Is this a panel? What is the common theme? Well, some months ago Hadrian Merler phoned me and asked me to organize a new sort of panel, not focused on a particular group of diseases, but rather a panel designed to deal with 'rapidly breaking news', in our case either about new diseases, outbreaks of old diseases in new situations, or updates on rapidly changing situations.

Program

A new home for *Cryptococcus neoformans* var. *gattii* in a Canadian temperate climatic zone. Karen Bartlett, School of Occupational and Environmental Hygiene, University of British Columbia, Vancouver, BC.

An update on Swiss needle cast in western Oregon. Alan Kanaskie, Oregon, Department of Forestry, Salem, OR.

***Dothistroma* in northwest British Columbia: Why there? Why now?** Alex Woods, BC Ministry of Forests, Smithers, BC.

The latest of SOD. David Rizzo, University of California, Davis, Department of Plant Biology, Davis, CA.

Comments

Now, while this may be a new way of putting topics together, perhaps starting a new tradition, I also hope we can maintain and even enhance an old tradition at WIFDWC: frank, vigorous, and, dare I say it, even vehement discussion in which speakers are challenged, alternate interpretations of data explored, and the truth is worth some hurt feelings (the latter can always be cured in the bar afterwards). And so I have invited one of our established members, one who has been steeped in this WIFDWC tradition from birth, and one who embraced it wholeheartedly and with great skill from the very first meeting he attended, to take special care that this great tradition is maintained and exercised also this afternoon. Terry, thanks for taking this on.

photo by Jacobi

Not all “leafy mistletoes” have “leaves”

Mistletoes of genus *Phoradendron* usually have obvious leaves, but the leaves of *Phoradendron juniperinum* and *Phoradendron librocedri* are reduced to minute scales. The fleshy, sticky, berry-like fruits similar to those of the European mistletoe (*Viscum album*) clearly indicate this is not a dwarf mistletoe (that is, a species of *Arceuthobium*). In North America, the native hosts of dwarf mistletoes are all in the family Pinaceae; any mistletoe on a juniper or incense-cedar in Jefferson, is a *Phoradendron*. The incense-cedar mistletoe and juniper mistletoe are very similar in appearance and have variously been recognized as different species, subspecies, varieties, or not. *Phoradendron libocedri* is very rarely reported for hosts other than incense-cedar; it is not common but is widely distributed throughout its host range.

A New Home for *Cryptococcus neoformans* var. g*attii*, in a Canadian Temperate Climate Zone

Karen H. Bartlett, Murray Fyfe, Laura MacDougall, Sunny Mak, Craig Stephen, and Sarah Kidd

Abstract—Beginning in 1999 and continuing to the present, a new infectious disease has been documented in British Columbia, Canada. The causative organism is *Cryptococcus neoformans* var. *gattii*, a basidiomycetous yeast which normally has a limited habitat in trees of tropical or subtropical areas of the world. Cases of cryptococcosis in humans and animals living in BC reported no travel history to endemic areas. Therefore, environmental sources of the organism were sought, particularly along the east coast of Vancouver Island where the majority of cases were clustered. In BC, *C. neoformans* var. *gattii* was found to colonize a wide variety of tree hosts. The organism was recovered from air and from soil. Isolates from clinical and environmental origins were found to be identical by PCR fingerprinting. This paper reports the first environmental isolation of *C. neoformans* var. *gatti* in a temperate climate zone.

Introduction

Cryptococcosis is an infection caused by an encapsulated, basidiomycetous yeast, *Cryptococcus neoformans*. The route of entry for infection for this organism is through the lungs, with possible systemic spread via the circulatory system to the brain and meninges. There are four cryptococcal serogroups associated with disease in humans and animals, distinguished by capsular polysaccharide antigens. *C. neoformans* is grouped into three varieties: *grubii* (serogroup A), *neoformans* (serogroup D) and *gattii* (serogroups B and C) (Franzot and others 1999; Kwon-Chung and Bennet 1992).

Cryptococci of serogroups A and D have a world-wide distribution and are particularly associated with soil and weathered bird droppings, although the birds themselves are not affected by the organism (Kwon-Chung and Bennet 1992; Hubalek 1975; Kwon-Chung and Bennet 1984). *C. neoformans* serogroups A and D have become the leading cause of life threatening infection in immunocompromised persons (Kwon-Chung and Bennet 1992).

In contrast, *C.* var. *gattii* (serogroup B) may infect a variety of mammalian hosts with intact immune systems. *C.* var. *gattii* has been reported to have a limited habitat in the tropics or sub tropics, and to colonize specific tree hosts, the river gum eucalypts (*Eucalyptus camaldulensis* and *E. tereticornis*) (Ellis and Pfeiffer 1990). However, beginning in 1999, increasing numbers of cases of human and animal disease associated with *C.* var. *gattii* were referred to the Central Laboratory for Veterinarians and the British Columbia Centre for Disease Control. Cases of cryptococcosis caused by *C.* var. *gattii* were confirmed in hosts with no other travel history than to have lived or to have visited the eastern coast of Vancouver Island, Canada.

From January 1999 to June 2003, the number of confirmed human cases was 77 and 5 deaths were directly attributable to *C.* var. *gattii* infection. Globally, the annual incidence rate of cryptococcosis is 1.5 per million population. The cases in British Columbia (BC) represent an average incidence rate of 19.5 cases per million, which is twice the rate of 8.5 cases per million population in tropical northern Australia where cryptococcosis associated with *C.* var *gattii* is endemic (Sorrell 2001).

Since January 2003, there has been a sudden increase in the number of cases of cryptococcosis reported in companion animals, with some 40 new cases this year in cats, dogs, ferrets and one bird. A number of these human and animal cases, particularly in cats, confirmed as *C.* var. *gattii* cryptococcosis, have no travel history to tropical or sub-tropical areas, no exposure to eucalypts, and very limited exposure to the outdoor environment.

Objective

A search was initiated in BC for sources of exposure to *C. neoformans* var. *gattii*, with particular emphasis on airborne exposures to account for the human and animal cases of cryptococcosis reporting limited exposure to any outdoor or wooded environments.

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Bartlett, Karen H. is an Assistant Professor in the School of Occupational and Environmental Hygiene, University of British Columbia, 2206 East Mall, Vancouver, BC, Canada V6T 1Z3.
Fyfe, Murray, MacDougall, Laura and Mak, Sunny are with the Epidemiology Service of the British Columbia Centre for Disease Control, 655 West 12th Ave. Vancouver, BC, V5Z 4R4.
Stephen, Craig is a veterinary epidemiologist with the Centre for Coastal Health, 900 5th St. Nanaimo, BC V9R 5S5.
Kidd, Sarah is a doctoral student at the University of Sydney, Australia.

Methods and Materials

Sample sites—Samples were taken from the homes and environs of cases (human and animal). All trees or homes were mapped using a GPS unit (eTrex). Sampling locations that were positive for the recovery of *C. neoformans* were tagged and repeatedly sampled throughout a calendar year. The abodes of animal and human cases of cryptococcosis were mapped using GIS coordinates, with the majority of cases resident on the eastern coast of Vancouver Island

Swab samples—Sterile, cotton tipped swabs with accompanying Amies transport media (Starswab II™) were used to recover cryptococci from trees. In the laboratory, the swabs were streaked onto a differential agar medium (niger seed agar). Ground niger seed in the agar provided caffeic acid as a substrate from which phenoloxidase-positive organisms form melanin, allowing cryptococci belonging to serogroups A, D, B or C to be visualized. Plates were incubated at 30° C for 48 hours to 7 days. Cryptococci which were brown on niger seed agar were purified and transferred to Canavanine-Glycine-Bromothymol Blue (CGB) agar (Kwon-Chung and others 1982). Growth of cryptococci belonging to serogroups B and C turn the medium blue in contrast to serogroups A and D which do not. Cultures conforming to cultural and microscopic characteristics were serotyped using monoclonal antibodies to capsular antigen (Crypto Check, Iatron Laboratories).

Air samples—(1) An Andersen six-stage sampling head was fitted with 100 mm plates containing 45 mL of niger seed agar. Air was drawn through the sampling head at 28.3 lm^{-1} for 15 min using a portable AirCon II pump. (2) A Reuter Centrifugal Sampler (RCS) was fitted with a 34 compartment strip filled with niger seed agar. RCS samples were taken for 4 minutes at an effective flow rate of 40 lm^{-1}. Agar media were returned to the lab and incubated as described.

Soil samples—Zip lock bags were used to collect surface soil samples. In the laboratory, approximately 2 g of soil was distributed into sterile 50 mL centrifuge tubes and 10 mL sterile water added. The tubes were vortexed, then the contents allowed to settle for 10 minutes. Duplicate plates of niger seed agar were inoculated with 0.1 mL each and processed as described above.

PCR—Molecular fingerprinting was performed on human, animal, and environmental isolates using polymerase chain reaction (PCR)-URA5 restriction fragment length polymorphism (RFLP) (Meyer and others 1999).

Results

Cryptococcus neoformans was isolated from swab, air and soil samples. Table 1 summarizes the number of initial, investigative swab samples that were positive for *C. neoformans* var. *gattii* culture.

Table 1—Results of initial swab samples of 1528 trees.

Tree species	Number examined	Number positive	Percent positive
Alder (*Alnus*)	174	22	13
Arbutus (*Arbutus*)	82	9	10
Ash (*Fraxinus*)	2	1	50
Birch (*Betula*)	5	0	0
Cherry (*Prunus*)	19	2	11
Cedar (*Thuja*)	166	17	10
Dead stump	8	1	13
Douglas fir (*Pseudotsuga*)	499	69	14
Eucalyptus (*Eucalyptus*)	23	0	0
Fir (*Abies*)	65	10	15
Fruit trees (various)	13	0	0
Garry Oak (*Quercus*)	65	14	22
Hemlock (*Tsuga*)	27	0	0
Maple (*Acer*)	122	9	7
Pine (*Pinus*)	38	2	5
Poplar (*Populus*)	16	0	0
Spruce (*Picea*)	23	1	4
Willow (*Salix*)	10	0	0
Other (or unknown)	171	4	2

Cryptococcus neoformans var. *gattii* was isolated from air in the environs of culture positive trees. Although there were seasonal differences in concentration, the organism was airborne in all seasons. Table 2 summarizes the geometric mean concentrations recovered from air.

Table 2—Airborne concentration of *C. neoformans* (Serogroup B) near culture positive trees in BC.

Season	n	GM[a]	GSD[b]	Range	p-value[c]
		CFU/m^3			
Summer (June–August)	27	30.5	11.6	0–2692	*
Fall (September–November)	23	6.7	5.5	0–436	* †
Winter (December–February	25	1.2	1.8	0–7	* † ‡
Spring (March–May)	21	5.3	6.2	0–550	*

[a] geometric mean
[b] geometric standard deviation
[c] legend: *,†,‡ group mean indicated significantly different from other means ($p < 0.001$) by Scheffe post hoc test.

About 12 percent of airborne *C. neoformans* particulate was $\leq$ 3.3 µm under quiescent conditions. However, during

chain sawing or chipping of infected wood, aerosols of cryptococci were present in higher concentration of respirable particulate as summarized in Tables 3 and 4.

Table 3—Airborne cryptococcal particulate under quiescent conditions.

Stage	Size (µm)	Mean (SD) CFU/m^3	Range CFU/m^3	Percent total
1	> 7.0	58 (164.4)	0–941	
2	4.7–7.0	51 (159.6)	0–582	87.9
3	3.3–4.7	57 (164.8)	0–707	
4	2.1–3.3	16 (58.2)	0–306	
5	1.1–2.1	1 (2.8)	0–14	12.1
6	0.65– 1.1	1 (2.4)	0–12	

n = 41 samples

Table 4— Aerosolized cryptococcal particulate.

Stage	Size (µm)	Mean (SD) CFU/m^3	Range CFU/m^3	Percent total
1	> 7.0	1019 (711.6)	25–1968	
2	4.7–7.0	586 (517.5)	14–1413	76.6
3	3.3–4.7	620 (584.0)	21–1378	
4	2.1–3.3	443 (556.2)	4–1413	
5	1.1–2.1	88 (163.4)	0–417	23.4
6	0.65–1.1	9.3 (5.4)	2–18	

n = 6 chain sawing and chipping tasks.

High concentrations of *Cryptococcus neoformans* var. *gattii* were isolated from the top soil beneath the tree canopy, and up to 20 m distant from an infected tree. Table 5 summarizes the geographic locations of positive soil sites. Figure 1 illustrates the locations and numbers of swab samples taken, overlaid on a map of health regions on Vancouver Island.

Table 5—*C. neoformans* serogroup B concentration in soil by geographic location.

Location	n	GM	GSD	range
		CFU/gram soil		
(1) Victoria* (southern tip of Vancouver Island)	45	16.6	18.2	0–3236
(2) Cowichan† (50 km north of Victoria)	71	245	69.0	0–331131
(3) Nanaimo (100 km north of Victoria)	56	0	0	0
(4) Parksville‡ (125 km north of Victoria)	271	43.7	1.6	0–338844
(5) Courtenay (200 km north of Victoria)	58	3.3	12.0	0–4365
(6) Port Alberni (40 km west of Parksville)	15	4.1	6.3	0–71
(7) Gulf Islands (11 to 15 km west of Cowichan)	43	0	0	0
(8) Mainland BC (50 km east of Vancouver Island)	41	0	0	0

* significantly different than (2), (4), (7) and (8) $p < 0.025$
† significantly different than (1) and (3–7) $p < 0.025$
‡ significantly different than (2), (3), (5), (7) and (8) $p < 0.025$

Epidemiology—Demographics: Mean case age at diagnosis was 59.7 years (range 20–82; SD 13.5). Fifty-eight percent of cases were male. Sixty percent were retired or unemployed at the time of their illness. Thirty matched case-control sets were interviewed. Risk factors for a diagnosis of cryptococcosis included: prior diagnoses of pneumonia (OR 2.71; 95% CI 1.05–6.98) or other lung problems (OR 3.21; 95% CI 1.08–9.52), or use of systemic corticosteroids (OR 8.11; 95% CI 1.74–37.8).

Animal cases had a wide distribution of ages from 9 months to 13 years. Risk factors have not been identified for animal cases.

PCR—Clinical cultures from immunocompetent humans and animals, and environmental isolates belonged to serogroup B. PCR-RFLP analysis of clinical and environmental isolates revealed two genetic variants, VGII (93 percent) and VGI (7 percent).

Discussion and Conclusions

This is the first description of recovery of the pathogenic basidiomycetous yeast, *Cryptococcus neoformans* from the environment in a temperate climate zone.

Although the prevalence of cryptococcosis is low (19.5 per million), the disease is debilitating and requires many months of antifungal therapy to resolve. The organism is not infectious from human to human or from animal to human contacts. The ecological niche of basidiomyceteous fungi in decaying wood has led researchers to theorize that *C.* var. *gattii* requires a tree host to become pathogenic to mammals, probably through the formation of basidiospores (Ellis and Pfeiffer 1990).

Cases of animal or human cryptococcosis caused in BC by *C. neoformans* var. *gattii* have been recognized since 1999. However, searches through stored cultures have not found any serogroup B organisms prior to this outbreak, suggesting this is an emerging infectious disease for this area. To date, no environmental isolates have been found in this temperate zone other than on the east coast of Vancouver Island. The east coast of the island is characterized by soil and vegetation of the Douglas-fir Biogeoclimatic Zone. The east coast of Vancouver Island has lower average rainfall and milder winters than the remainder of the east coast of the island or the west coast of

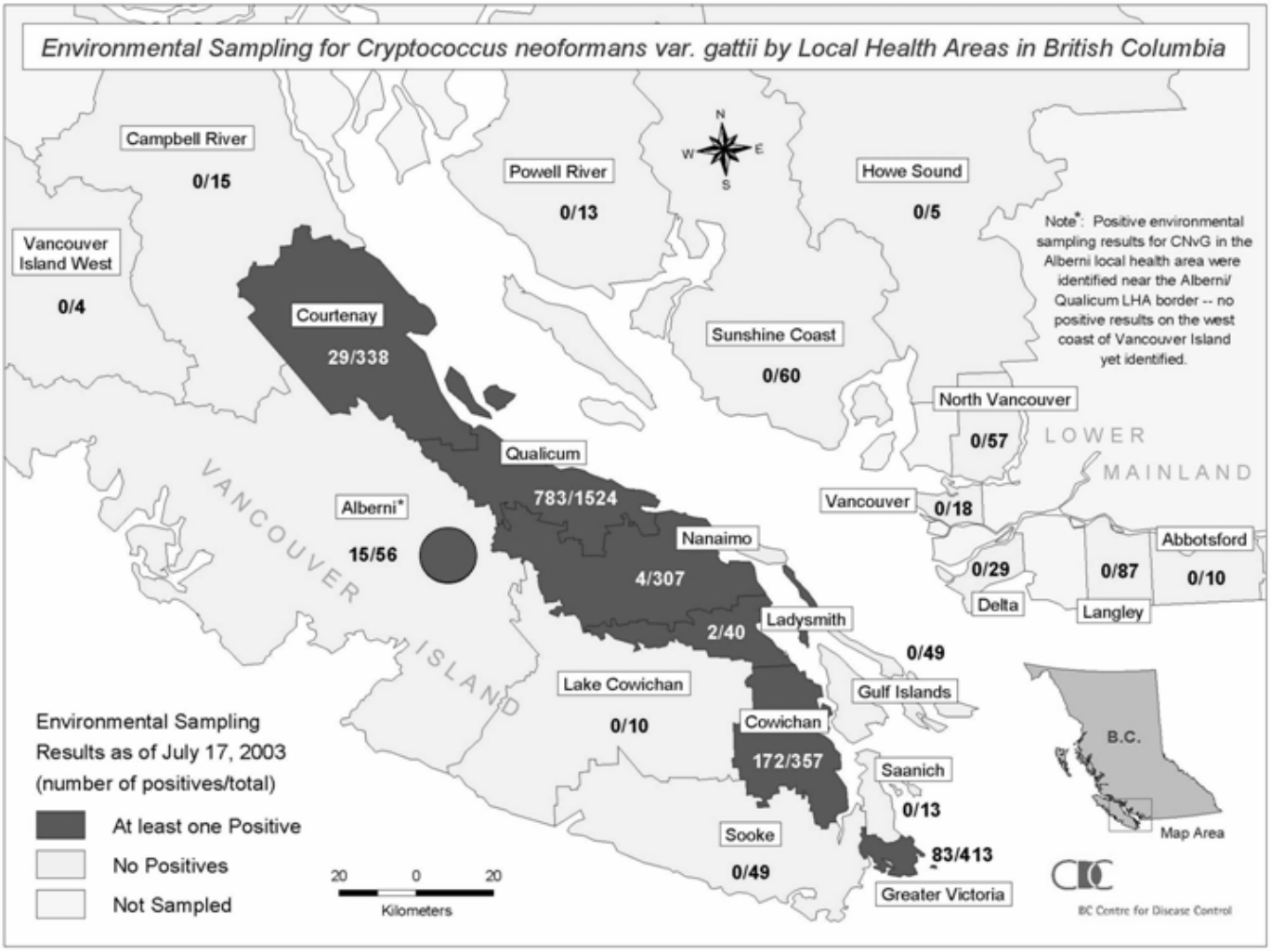

Figure 1–GIS mapping of tree location sampled for *C. neoformans var. gattii.*

the Mainland, which is characterized primarily by soil and vegetation of the Mountain Hemlock Zone.

In tropical and sub-tropical zones *C. neoformans* var. *gattii* colonizes unique tree hosts (for example, eucalypts in Australia, India, Brazil, Columbia). In contrast, *C. neoformans* var. *gattii* in BC has colonized a wide variety of trees, both deciduous and coniferous. The organism has consistently been recovered from swab cultures taken over a period of a year. In the tropics the organism is airborne only when the colonized trees are blooming (or pollinating). Although the concentration in air is higher in the summer than other seasons on the east coast of Vancouver Island, we have recovered airborne organisms in all months of the year.

The primary ecological niche of this organism is most likely the mixture of soil and tree debris. High concentrations of the organism have been consistently recovered from top soil. The high concentration of cryptococci indicate extremely successful competition with the rich diversity of microflora that colonize the rhizome spheres of vegetation. This suggests that it is less likely that the organism is a transient visitor to the Island. Studies are underway to more thoroughly characterize the soil chemistry for comparison with soil from the Mountain Hemlock zone.

The organism has been recovered from the soles of shoes of the research staff, and on car tires after sampling trips into culture positive areas suggesting that the organism can be carried by fomites. However, to date, no positive sites have been identified on the Gulf Islands or the Mainland, BC.

In conclusion, a new ecological niche for *C. neoformans* var. *gattii* has been described. Research is continuing to characterize other determinants of exposure to this organism (for example, climate, soil or wood product movement) in order to reduce the risk of environmentally acquired cryptococcosis for susceptible populations.

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An Update on Swiss Needle Cast in Western Oregon

Alan Kanaskie, Mike McWilliams, Keith Sprengel, Douglas Maguire, Doug Mainwaring

Abstract—Since the early 1990s, Swiss needle cast (SNC) damage has increased dramatically in the Coast Range of western Oregon. Aerial surveys estimate an increase in acres with severe damage from 130,000 acres in 1996 to nearly 390,000 acres in 2002. Aerial surveys also show that most damage is within approximately 18 miles of the coast. On permanent monitoring plots, needle retention ranges from 1.3 to 3.6 annual foliage compliments (mean = 2.3). Reduction in volume growth averages 23 percent across the study area, with the most severely damaged stands experiencing more than 50 percent growth reduction due to SNC, as compared to plantations with the lowest level of damage. Thinning studies indicate that trees respond positively to thinning across a range of SNC damage, with the degree of growth response decreasing as foliage retention decreases. At the stand level, the ratio of 3-year post thinning to 3-year pre-thinning basal area growth was less than 1.0 when foliage retention was less than 2.3 years. Aerial application of chlorothalonil over a 5-year period resulted in dramatic increases in stand volume growth compared to untreated stands. Ongoing studies focus on evaluating the economics of fungicide applications, and understanding the interaction between SNC severity and silvicultural treatments, especially thinning.

Background and History

In the early 1980s, several Douglas-fir plantations in coastal northwest Oregon appeared unusually chlorotic in late winter and early spring. Most trees had lost all but the current-year needles, and trees appeared to be growing more slowly than expected. Only Douglas-fir was affected.

By the mid-1980s, additional plantations displayed similar symptoms. The disorder at that time was referred to as Douglas-fir decline or the Tillamook crud, and was often attributed to "off-site" planting. Swiss needle cast (SNC) caused by *Phaeocryptopus gaeumannii* was associated with the symptoms on all sites, but abundance of fruiting of *P. gaeumannii* varied considerably from site to site.

Applications of the fungicide chlorothalonil (Bravo 500) to paired trees (one treated, one untreated) in 1988 showed that needle retention and color improved dramatically and consistently when new foliage was protected. Although this and subsequent fungicide-based studies have consistently implicated Swiss needle cast, other factors were suspected as contributing to the dramatic increase in damage caused by this historically benign pathogen.

In the early 1990s, Swiss needle cast damage slowly increased in the Oregon Coast Range. In the spring of 1994, an alarmingly large area of Douglas-fir forest with SNC symptoms was visible, and the first aerial survey to map the extent of damage was launched. Damage continued to increase during subsequent years, and in 1997, the Oregon State University Swiss Needle Cast Cooperative (SNCC) was founded by concerned land managers to conduct research on the disease and its management. Several aspects of SNC research and monitoring were presented at the 2001 WIFDWC in Carmel, CA. This paper provides an update on SNC in western Oregon.

Swiss Needle Cast Aerial Surveys, 1996 to 2003

Survey Procedures

Aerial surveys for SNC have been conducted in April and May each year since 1996. The observation plane flew at 1,500 to 2,000 feet above the terrain, following north–south lines separated by 2 miles. Observers looked for areas of Douglas-fir forest with obvious yellow to yellow-brown foliage, a symptom of Swiss needle cast. Patches of forest with these symptoms (patches are referred to as polygons) were sketched onto computer touch screens displaying topographic maps or ortho-photos and the position of the aircraft (surveys prior to 2000 used paper sketch maps).

Each polygon was classified for degree of discoloration as either "S" (severe) or "M" (moderate). Polygons classified as "S" for discoloration had very sparse crowns and brownish foliage, while those classified as "M" had

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predominantly yellow to yellow-brown foliage with slightly denser crowns than those classified as “S”.

The Coast Range was surveyed on May 19 to May 28, 2003. The area surveyed extended from the coastline eastward until obvious symptoms were no longer visible, and from the Columbia River south to Brookings. We did not survey the Cascades in 2003, but Swiss needle cast does occur at damaging levels in some areas.

Results and Discussion

Figure 1 shows the approximate size and location of areas of Coast Range Douglas-fir forest with symptoms of Swiss needle cast detected during the survey conducted in May 2003 and in May 1996. Figures 2, 3, and 4 show the trend in damage from 1996 through 2003.

Figure 1—Areas of Douglas-fir forest in western Oregon with symptoms of Swiss needle cast detected during an aerial survey in May 1996 and May 2003.

The 2003 Coast Range survey covered about 3 million acres of forest. Approximately 268,000 acres of Douglas-fir forest had obvious symptoms of Swiss needle cast; 196,000 north of the Lincoln–Lane county line, and 72,000 acres south of the Lincoln–Lane county line (figure 1). The easternmost area with obvious SNC symptoms was approximately 25 miles inland from the coast. Most areas with symptoms that could be detected from the air occurred within 18 miles of the coast.

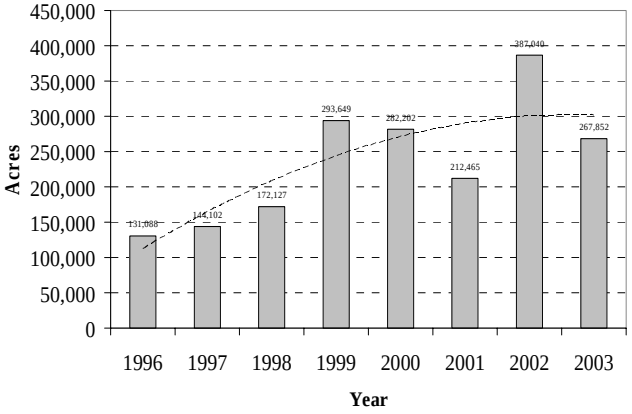

Figure 2—Trend in area of Douglas-fir forest in western Oregon with symptoms of Swiss needle cast detected during aerial surveys in April and May, 1996 to 2003.

Some year-to-year variation in survey results is due to timing of the flights relative to the development of SNC symptoms. Because symptoms develop rapidly during April and May, later surveys usually detect more areas with symptoms than those conducted earlier. The 2003 survey was flown from May 19 to May 28, which is relatively late (the 2001 survey was flown from May 3 to May 10; the 2002 survey was flown from May 1 to May 31). This difference in timing of the survey, coupled with weather-driven tree phenological development, could account for much of the yearly variation in survey results. Of possible significance is the fact that most acres mapped in 2002 and 2003 were classified by observers as "moderate", and very few acres were classified as "severe". Although this distinction in degrees of discoloration often is fickle, the difference between the 2002–2003 surveys and previous surveys is notable.

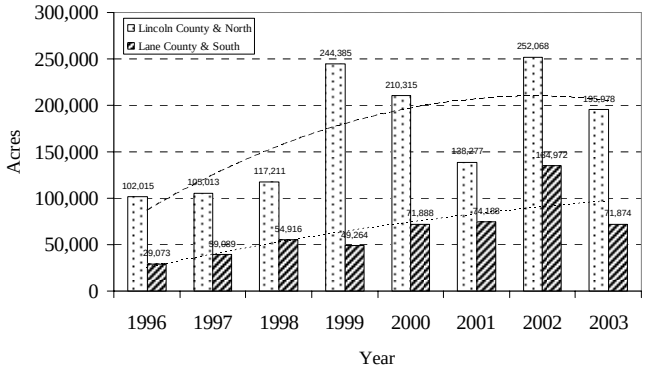

Figure 3—Area of Douglas-fir forest in western Oregon with symptoms of Swiss needle cast detected during aerial surveys in April and May, by zone, 1996 to 2003.

Aerial survey results are conservative estimates of damage because observers map only those areas where disease symptoms have developed enough to be visible from the air. Permanent monitoring plots and ground checks have shown that Swiss needle cast occurs throughout the survey area, but that symptoms often are not developed enough to enable aerial detection. Because the survey detects discoloration and does not describe needle retention (which is correlated with growth loss), estimates of disease impact on tree growth should not be made from the aerial survey alone. However, the aerial survey does provide a broad picture of the area significantly impacted by Swiss needle cast, which from a practical point of view establishes a zone in which forest management should take into account the effects of the disease.

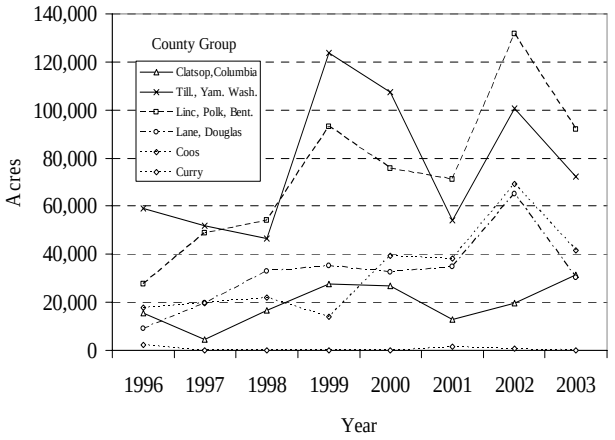

Figure 4––Area of Douglas-fir forest in western Oregon with symptoms of Swiss needle cast detected during aerial surveys in April and May, by county group, 1996 to 2003.

Monitoring SNC on Permanent Plots in 10- to 30-year-old Douglas-fir Plantations

A permanent plot network was established in 1997 to provide a basis for monitoring Swiss needle cast damage and for quantifying impacts of SNC on tree growth. This paper describes only the results of monitoring various indicators of SNC damage. A discussion of growth impacts can be found in Maguire and others (2002).

Objectives

The objectives of the ongoing study are: 1) to describe the trends in the severity of damage from Swiss needle cast in randomly chosen 10- to 30-year-old Douglas-fir plantations in the Coast Range of western Oregon; 2) to estimate the area (acres) affected by SNC, and; 3) to ground-truth areas mapped by aerial survey.

Methods

In 1997, 77 Douglas-fir plantations in the northern Coast Range of Oregon were randomly chosen for monitoring trends in damage from Swiss needle cast. The target population was all Douglas-fir plantations between 10 and 30 years total age (age in 1996) and located within 18 miles of the coast, north of Newport and south of Astoria. With much cooperation from landowners, a list of plantations meeting these criteria was assembled. Plantations were selected from this list with probability proportional to size (area). The target population included 4,504 plantations covering 187,545 acres. The initial sample included 77 plantations covering 6,873 acres (figure 5). One plantation was lost from the study in 1999 due to cutting.

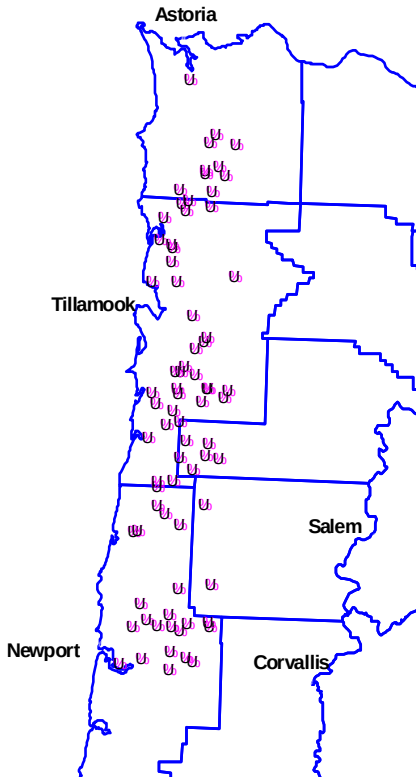

Figure 5—Location of 76 permanent plots for monitoring Swiss needle cast and tree growth in 10- to 30-year-old Douglas-fir plantations, Coast Range, Oregon.

Swiss needle cast damage was assessed in April and May of each year since 1997. The 1997 assessments were based on a sample of ten trees per stand (two trees at each of five points along a transect). Beginning in 1998, assessments were made on ten trees per stand located in the 1/5-acre permanent growth monitoring plots (Phase III) located at point 5 of the 1997 transects. The same ten trees in each plot were assessed each year unless mortality or breakage necessitated substitution.

Stand Ratings—Stand ratings were designed to provide a quick method of estimating Swiss needle cast severity by making a general assessment of average stand condition during a brief walk-through of the stand. All ratings refer to the Douglas-fir component of the stand in the vicinity of the permanent plots. Overall stand discoloration was rated on a scale of 1 to 4 for as follows: 1 = normal green color, with Douglas-fir similar in color to healthy hemlock; 2 = slight yellowing; 3 = moderate yellowing, and; 4 = severe yellowing and/or browning.

The Swiss Needle Cast Severity Rating for the stand was described according to the following 6-class system (needle retention was assessed on unshaded secondary laterals in the upper middle crown, usually whorl 5 to 7 from the tree top):

1 = Healthy, normal-appearing Douglas-fir stand. Typical of the east-slope Coast Range stands that are dark green, growing normally, and with normal needle retention (3.5 years or more mid-crown). Douglas-fir and hemlock of the same size will not differ appreciably in color. Swiss needle cast may be present, but causing symptoms only on 3-year-old and older needles.

2 = Almost normal, but showing slight yellowing. Needle retention normal (3.5 or more years present on most trees) Douglas-fir will appear slightly more yellow than hemlock or spruce. Crown still appears full and dense. No reduction in height growth increment.

3 = Yellowing obvious. Most trees retaining 2.5 to 3 years of needles. No obvious height increment reduction.

4 = Yellowing obvious. Most trees retaining 1.5 to 2 years of needles. Reduction in height growth increment by 25 percent of normal for one or more of the last three years will not be obvious, but may occur on a few trees only.

5 = Very yellow stand. Most trees retaining 1 to 1.5 years of needles. Height growth increment is reduced by at least 25 percent of normal for one or more of the last 3 years on at least 50 percent of trees, but not as much as described for rating 6.

6 = Stand is extremely yellow to yellow-brown, with very sparse foliage. Most trees retaining 1 year of needles or less in upper crown. Obvious height growth reduction for 4 or more years. These are the most severely damaged stands, typical of the Juno Hill, Beaver, and Hebo areas.

Individual tree assessments on plots—Ten co-dominant or dominant trees in each 1/5-acre permanent plot were assessed for damage from Swiss needle cast. Sample trees were permanently tagged so the same trees could be assessed each year.

Needle retention was estimated for the middle of each third of the live crown (upper, middle, lower) by examining secondary lateral branches and estimating the average number of annual needle compliments present (a secondary lateral is a branch that originates on the side of the main lateral branch). Sample branches were chosen to represent the average condition in the part of the crown being examined. The number of annual needle compliments present for each third of the live crown was estimated to the nearest 0.1 as follows:

0.5 = 50 percent of one-year-old needles remain, all older needles gone

1.0 = All one-year-old needles remain, older needles gone

1.2 = One-year-old needles plus 20 percent of two-year-old needles remain

1.6 = One-year-old needles plus 60 percent of two-year-old needles remain

2.0 = One- and two-year-old needles remain, older needles gone

2.5 = One- and two-year-old needles remain, plus 50 percent three- year-old needles remain

3.0 = All one-, two-, and three-year-old needles remain....

...and so on up to 6.0.

Whorl-5 needle retention was estimated by examining branches in the fifth whorl down (occasionally the sixth or seventh whorl) from the top of the tree. Needle retention, that is, percentage of the full compliment of needles remaining on the branch at the time of the assessment, was estimated for each of the four most recent internodes of shoot growth on secondary laterals according to the following 0 to 9 scale:

0 = 0 to 10 percent of full compliment present;

1 = 11 to 20 percent of full compliment present;

2 = 21 to 30 percent of full compliment present;…

9 = 90 to 100 percent of full compliment present.

Crown indicators—From 1998 to 2000 inclusive, observers estimated the following four indicators, based in part on the US Forest Service Forest Health Monitoring protocols:

1) Crown Color—discoloration of the upper 1/2 to 1/3 of crown, near whorls 5 to 7 from the top, using the 1 to 4 scale previously described for stand color;

2) Crown Density—estimate the percentage of sunlight being blocked by all parts of the crown, in 5-percent classes;

3) Foliage Transparency—estimate the percentage of sunlight being transmitted through the foliage, in 5-percent classes;

4) Crown Dieback—estimate the percentage of the total crown area that has dieback, in 5-percent classes. For the 2001 and subsequent assessments, crown density and transparency variables were dropped because they had not proven very useful in previous analyses.

Results and Discussion

The mean SNC stand rating (1 to 6 scale) increased gradually by nearly one rating class between 1997 and 2003 (figure 6). This trend suggests a general increase in SNC damage over the period based on subjective overview ratings. Although the SNC rating for 2003 was not significantly different from the 2002 rating, the ratings for these two years were significantly greater than in previous years.

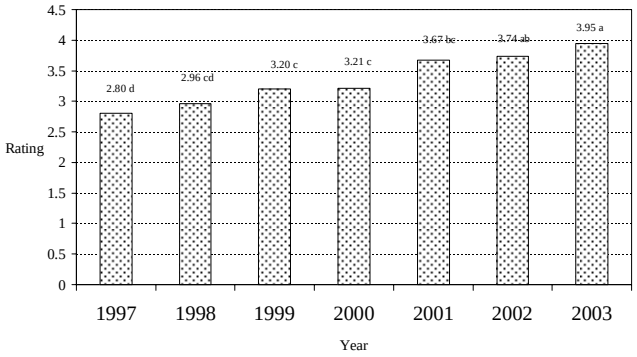

Figure 6—Mean SNC Stand Rating for 76 permanent plots in 10- to 30-year-old (1996 age) Douglas-fir plantations. Swiss needle cast severity increases as SNC stand rating increases. Means with same letter within a data series are not significantly different (analysis of variance, Fisher's LSD, α=.05).

In contrast, the mean stand discoloration rating did not differ consistently during the same period, suggesting a trend of slightly improving stand color between 1997 and 2001, then an increase in 2002 and 2003 to 1997 levels (figure 7). One explanation for the discrepancy between these two stand ratings is that the SNC rating incorporates both needle retention and color into the rating. A stand that is very yellow but with good needle retention could lead to different relative ratings on each scale. The SNC rating (1 to 6) is the preferred method for overview rating stands because it incorporates many indicators of Swiss needle cast damage. The stand color rating was originally conceived as a link to aerial survey and remote sensing applications. In practice, the stand discoloration rating has

proven very difficult to determine with consistency because of the influence of sunlight, cloud cover, and observer subjectivity. The SNC rating often is not consistent with needle retention ratings, largely because the SNC rating tends to focus on condition of the upper crown, which is the only part visible from outside of the stand.

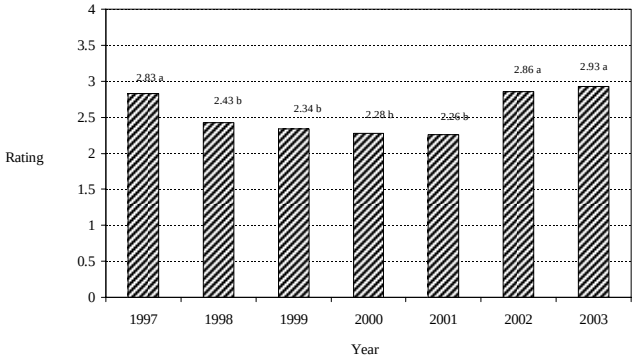

Figure 7—Mean SNC Stand Discoloration Rating for 76 permanent plots in 10- to 30-year old (1996 age) Douglas-fir plantations. Stands become more discolored (more yellow) as Stand Discoloration Rating increases (1=no discoloration, 4=most discoloration). Means with same letter within a data series are not significantly different (analysis of variance, Fisher's LSD, α=.05).

Mean needle retention (whole crown) for all plots increased slightly from 1997 to 1999, decreased from 1999 to 2001, then increased gradually to the highest level (2.54 annual needle compliments) since measurements began. Although the differences were significant (analysis of variance, .05 significance level), they were very small (figure 8).

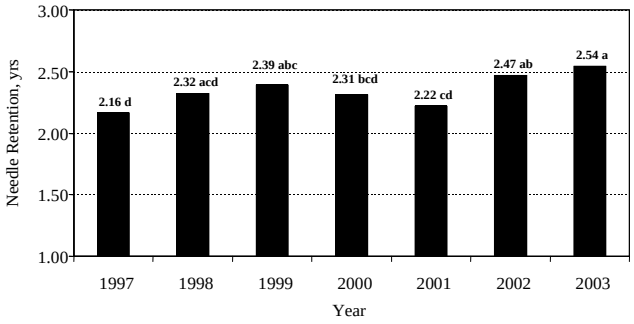

Figure 8—Mean needle retention for 76 permanent plots in 10- to 30-year old (1996 age) Douglas-fir plantations. Means with same letter are not significantly different (analysis of variance, Fisher's LSD, α=.05).

Analysis by crown thirds consistently has shown that mean needle retention is lowest in the upper third of the tree crown and greatest in the lower third of the tree crown. Mean needle retention in the upper third of the crown showed a noticeable but slight decrease from 1998 to 1999, probably reflecting the interaction of Swiss needle cast with the high frequency of severe windstorms and a period of very cold weather that occurred during the winter of 1998–1999 (Figure 9). Slight improvements in needle retention in 2001–2003 could be due to recent relatively mild winters, as well as increasing tree size and subsequent crown sheltering, both of which could reduce foliage loss.

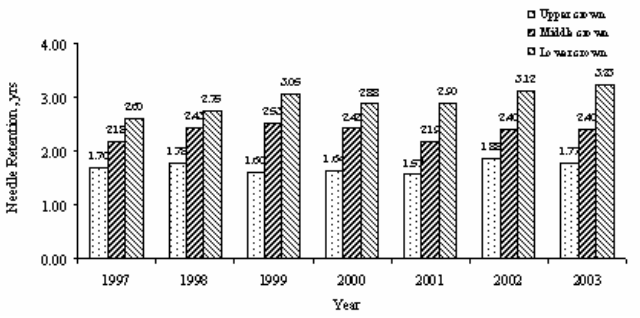

Figure 9—Mean needle retention for 76 permanent plots in 10- to 30-year-old (1996 age) Douglas-fir plantations, by crown thirds. Means with same letter are not significantly different (analysis of variance, Fisher's LSD, α=.05).

Mean needle retention for each permanent plot appears in figure 10. Mean needle retention (whole crown) differed significantly (analysis of variance, paired t-tests, α=.05) between 1998 and 2003 on 13 (17 percent) of the plots. During this period, mean needle retention increased on 4 of the plots, and decreased on 9 of the plots (figure 11). We chose 1998 as the reference year rather than 1997 because the 1997 data were from transect trees, while all subsequent data were from permanent plot trees, with the same trees being measured each year. The largest improvement in needle retention for an individual stand during this period was 0.71 annual needle compliments; the largest decrease in retention was 1.14 annual needle compliments. We did not observe any geographic pattern to the changes in needle retention.

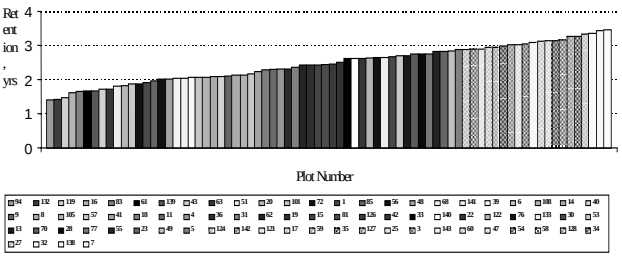

Figure 10—Mean needle retention (whole crown) in May 2003 for each of the 76 permanent plots in 10- to 30-year-old (1996 age) Douglas-fir plantations.

Mean needle retention ratings were expanded to estimate the number of acres in each needle retention class for the 187,545-acre population. Since 1997, there has been a general increase in the estimated number of acres with needle retention of at least 2.25 annual compliments, and a general decrease in estimated acres with less than 2.25 annual compliments (figure 12).

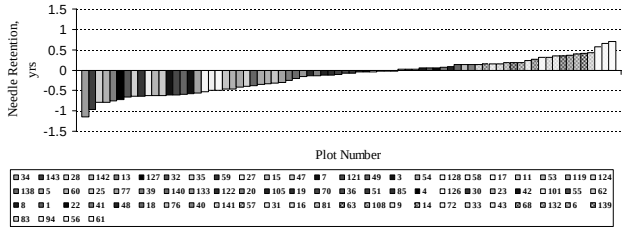

Figure 11—Increase or decrease in mean needle retention between 1998 and 2003 in 76 permanent monitoring plots in 10- to 30-year-old (1996 age) Douglas-fir plantations. Mean needle retention increased significantly on four of the plots, and decreased significantly on nine of the plots (analysis of variance, Fisher's LSD α=. 05). Horizontal line above or below vertical bars indicates stands with significant differences.

Conclusions

Based primarily on needle retention ratings, these results show little evidence of a significant change in damage from Swiss needle cast since 1997. The slight but significant increase in mean needle retention from 2001 to 2002, and the lack of a consistent trend of worsening damage from SNC are encouraging, and are consistent with casual observations in the north coast area. However, the overall poor needle retention in the sample population suggests a continuing severe growth reduction from Swiss needle cast.

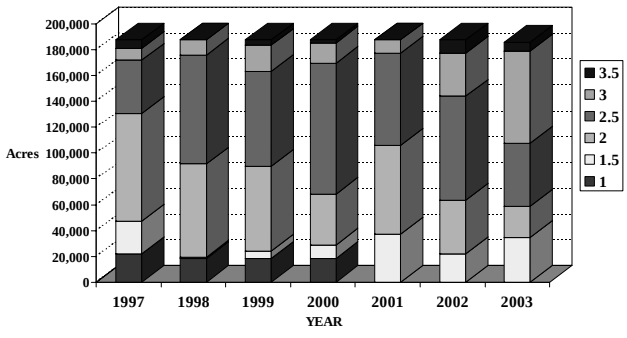

Figure 12—Distribution of Douglas-fir plantation acreage by needle retention class for the 187,545-acre population from which sample plantations were chosen.

Pre-commercial Thinning of Douglas-fir Stands with Varying Intensity of Swiss Needle Cast_____

Background

Many young Douglas-fir plantations in coastal Oregon exhibit extreme symptoms of Swiss needle cast, and these symptoms are associated with reduction in tree growth. Observations suggest that thinning stands with severe Swiss needle cast may increase foliage loss and discoloration, and exacerbate thinning shock. Other observations indicate that early thinning to maintain deep crowns may mitigate some of the growth loss attributed to Swiss needle cast. The response of stands to thinning is expected to vary according to the initial severity of Swiss needle cast at time of thinning.

Objectives

The objectives of the study were: 1) to monitor concurrently on permanent plots the course of Swiss needle cast symptoms and the effect of the disease on the growth of individual trees; 2) to measure shifts in SNC infection severity and associated tree growth responses over time, and; 3) to measure differences in disease severity and tree growth in thinned and unthinned plots. This reports focuses on trends in the various indices of disease severity between 1998 and 2003. Tree growth responses to pre-commercial thinning and Swiss needle cast appear in Maguire and others (2001).

Methods

In April and May of 1998, twenty-three paired 0.2 acre square plots were installed in 10- to 16-year-old Douglas-fir plantations (1997 age) in northwest Oregon. Plot locations were selected across a range of Swiss needle cast severity classes and distributed across different topographic aspects (figure 13). One plot in each pair was pre-commercially thinned to approximately 200 trees per acre in May 1998 (because of initial stocking levels, at two sites the target residual was 100 trees per acre). At five of the 23 locations, an additional plot was thinned to approximately 100 trees per acre. During thinning, tree spacing was given priority over tree quality. All crop trees were measured for d.b.h., total height, and height to crown. Swiss needle cast severity (needle retention and discoloration) was assessed annually during April and May each year since plot establishment. Growth measurements are taken every two years. All stand and tree ratings were as described for the permanent plot study.

Results and Discussion

Analysis of data for all 23 sites revealed few trends. Four growing seasons after thinning, mean needle retention did not differ significantly between thinned and unthinned plots (analysis of variance, Fisher's LSD, α=05). Mean needle retention also did not differ significantly among the five annual measurements for any of the treatments (analysis of variance, .05 significance level) (figure 14). There was no significant difference in Stand Swiss Needle Cast Severity or Discoloration Ratings among years or between thinning treatments (figure 15).

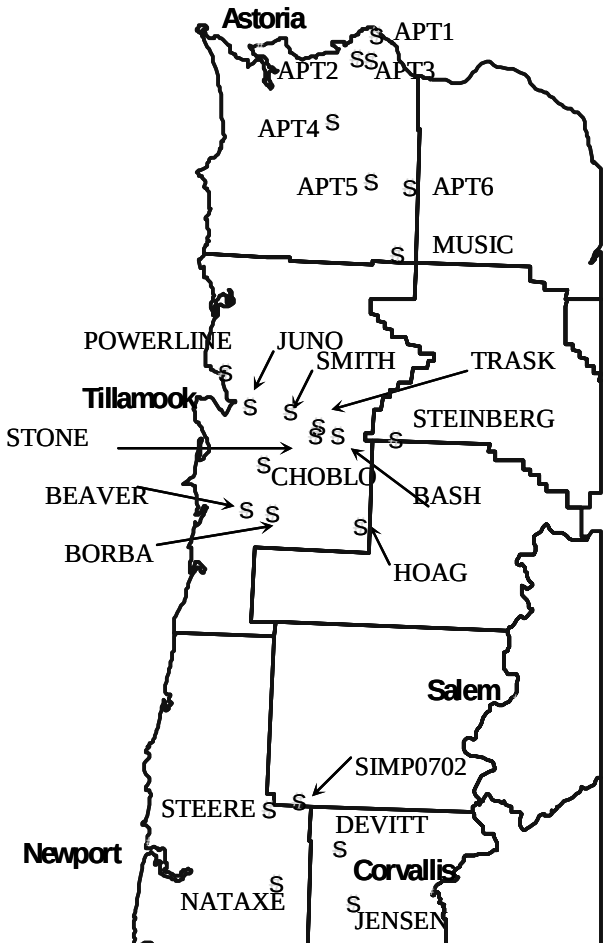

Figure 13—Location of 23 permanent plot sets to monitor disease symptoms and evaluate growth response to pre-commercial thinning in Douglas-fir plantations with varying intensity of Swiss needle cast in the Coast Range of Oregon.

Analysis of data from each installation separately revealed that mean needle retention in 2002 differed significantly between thinned and unthinned plots at 7 of the 23 sites (T100 and T200 treatments were combined for this analysis). At six of these sites (APT4, APT5,APT6, Nataxe, Axe, Steere, and Steinburg), trees in the thinned plots had greater needle retention than trees in the unthinned plots. At the other site (Powerline, Tillamook County), mean needle retention of trees in the unthinned plot was greater than needle retention trees in the thinned plot (analysis of variance, .05 significance level). The magnitude of difference in needle retention between thinned and unthinned plots on these seven sites ranged from 0.5 to 1.0 annual needle compliments (figure 16).

Five of the sites received two levels of thinning; 100 (T100) and 200 (T200) residual trees per acre. A comparison of mean mid-crown needle retention among treatments at these sites five growing seasons after thinning showed significant differences among thinning treatments at three of the five sites (figure 17). At two sites, APT5 and APT6, mean needle retention was greater in the thinned plots than in the unthinned plots, but did not differ between the T100 and T200 treatments. At the other site (Devitt), mean needle retention in the T100 plot was significantly less than in the T200 plot and the unthinned plot.

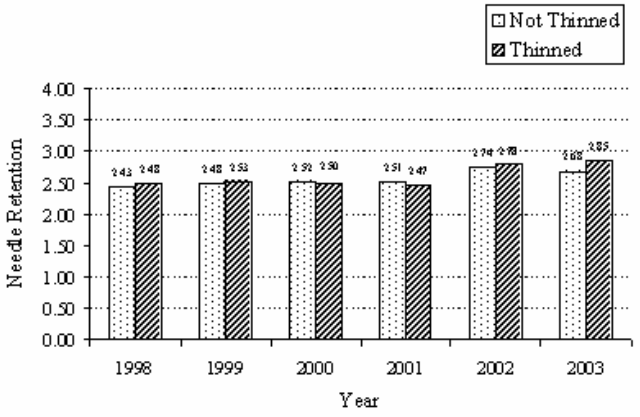

Figure 14—Mean needle retention (whole-crown) in paired thinned and unthinned plots in Douglas-fir plantations affected by Swiss needle cast in the Coast Range of northwest Oregon, 1998 to 2003. Plots were thinned in May 1998. Needle retention was evaluated in May of each year. Mean needle retention did not differ significantly (analysis of variance, α=.05) between thinned and unthinned plots, or among years.

Casual observations have lead to speculation that Douglas-fir plantations with severe Swiss needle cast and poor needle retention will experience more needle loss following pre-commercial thinning than unthinned plantations. Analysis of the six stands with the lowest needle retention at the time of thinning and the six stands with the greatest needle retention at the time of thinning showed little difference in the effects of thinning on needle retention four growing seasons after thinning. Needle retention was

significantly lower in the thinned plot at only one of the six sites with the poorest initial needle retention (Figure 18), and at none of the six sites with the highest initial needle retention (figure 19).

Needle retention is not the only measure of the effects of thinning on tree damaged by Swiss needle cast. A small amount of tree fall, top breakage, and branch dieback occurred at low levels in a few of the thinned plots, but not in the unthinned plots, and especially in plantations with the most severe Swiss needle cast.

A trend appears to be developing for improved needle retention in thinned plots. The expected high loss of foliage following pre-commercial thinning of stands damaged by Swiss needle cast has not occurred five growing seasons after thinning. Even sites with severe disease (such as Juno Hill and Beaver) showed little difference in mean needle retention between thinned and unthinned plots. Needle retention ratings, although correlated with tree volume growth, likely do not capture the entire the impact of the Swiss needle cast on tree growth. Retention ratings do not account for shoot length, needle size and quality, crown length, or the absolute amount of foliage present, all of which vary considerably in stands affected by Swiss needle cast. A differential tree growth response to thinning across a range of Swiss needle cast damage still is quite possible, despite the inconclusive needle retention results.

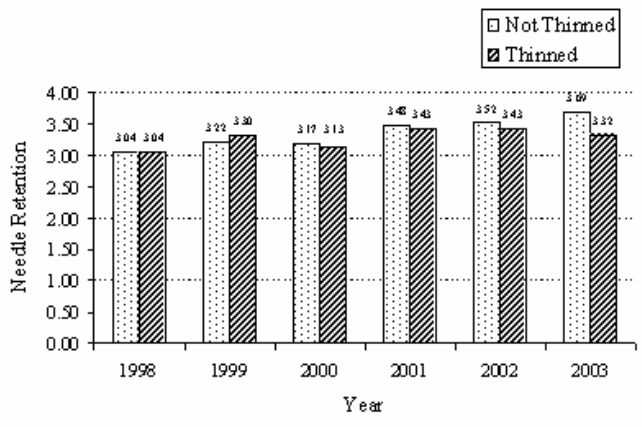

Figure 15—Swiss Needle Cast Stand Severity Rating for paired thinned and unthinned plots in Douglas-fir plantations affected by Swiss needle cast in the Coast Range of northwest Oregon, 1998 to 2003. Plots were thinned in May 1998. The SNC severity rating did not differ significantly (analysis of variance, α=.05) between thinned and unthinned plots, or among years.

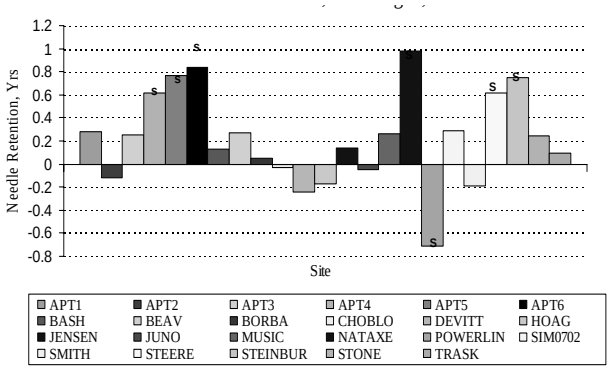

Figure 16—Mean difference in mid-crown needle retention in 2003 for 23 paired thinned and unthinned plots of Douglas-fir affected by Swiss needle cast in the Coast Range of northwest Oregon. Plots were thinned in May 1998. Needle retention was evaluated in May of each year. A vertical bar that extends below the zero line indicates that needle retention in the thinned plot was less than in the unthinned plot; if above the line, needle retention in the thinned plot was greater than in the unthinned plot. Significant differences are indicated by "s" (t-test, α=.05). T100 and T200 treatments were combined for this analysis.

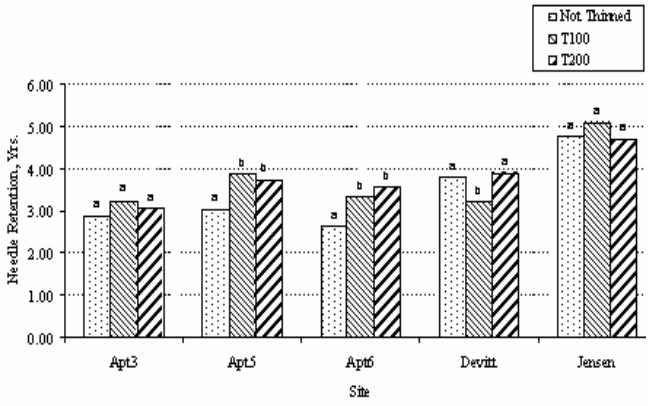

Figure 17—Mean needle retention (mid-crown) in 2003 for sites with three treatments (not thinned; T100 = 100 trees per acre after thinning; T200 = 200 trees per acre after thinning). Means within a site with the same letter are not significantly different (analysis of variance, Fisher's LSD, α= .05).

The stands in this study were approximate 12- to 16-years old at the time of thinning. Trees of this age typically have deep crowns and are growing vigorously. Older overstocked stands with relatively low crown ratios could respond quite differently to thinning than the stands in this study.

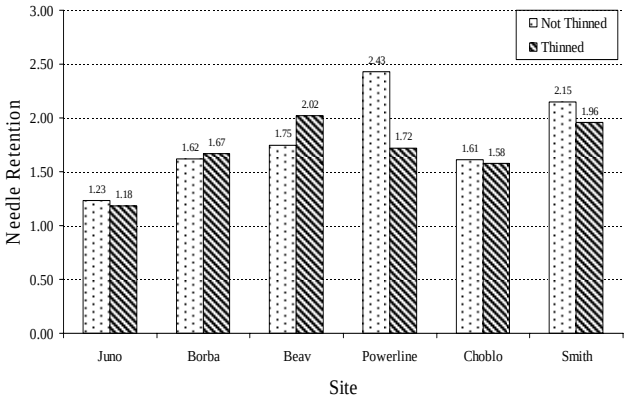

Figure 18—Mean needle retention (mid-crown) in 2003 for paired thinned and unthinned plots with the lowest initial (1998) needle retention.

Conclusions

These results suggest that pre-commercial thinning does not have an obvious detrimental effect on Douglas-fir plantations affected by Swiss needle cast in the Coast Range of Oregon. The 2003 data suggest a slight improvement in needle retention in the thinned plots. Pre-commercial thinning remains a viable stand management tool in the Coast Range in all but the most severely damaged stands.

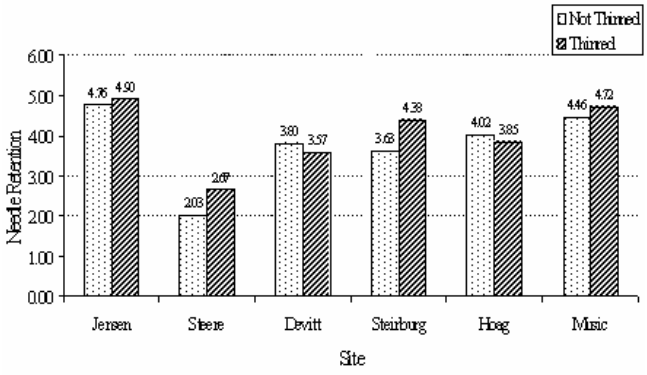

Figure 19—Mean needle retention (mid-crown) in 2003 for paired thinned and unthinned plots of Douglas-fir with the greatest initial (1998) needle retention.

Response of Douglas-fir to Aerial Applications of Chlorothalonil to Control Swiss Needle Cast

Methods

The study sites were located near the town of Hemlock, Oregon, on the west flank of the Coast Range. The study was designed as a complete randomized block experiment with three separate blocks or sites, all in 16-year-old Douglas-fir plantations. Each 10-acre study site was divided into two 5-acre experimental units, one of which served as the control and the other of which was treated with fungicide. On the treated units, chlorothalonil (Bravo 720) was applied by helicopter two times each year at a rate of 5.5 pints per acre, with treatment areas double-flown to ensure thorough coverage. The first application occurred when new shoots on approximately 40 percent of the trees were 1 to 5 cm in length, and the second when at least 90 percent of the trees had new shoots of this length. Applications occurred in May and early June, and were separated by two to three weeks. The treated units were sprayed each year for five years (1996 to 2000).

Measurement plots were established in each of the experimental units during the dormant season of 2000–2001. Each plot was 0.1 acre in area (37.2 ft. radius). All trees on the plots were tagged at breast height, and live Douglas-fir trees were measured for d.b.h., height, and height to lowest live branch. The largest five trees on each plot were felled, and a stem disk was removed at breast height and at crown base. Radial growth and sapwood width was measured on four radii corresponding to the longest axis and the axis perpendicular to the longest on the disk. All standing live Douglas-fir trees were cored to the pith, and radial growth for the last ten years and sapwood width were likewise recorded. Annual height growth for the last 11 years was also measured on the felled trees, and each was rated for foliage retention as an index of Swiss needle cast severity. Needle retention was estimated at the tree level by dividing the crown into thirds and estimating the average number of years foliage was retained. In addition, one branch was sampled from the south side of whorls five through ten from the tip of the tree. On each of these branches, the percentage of each annual age class retained on the primary and secondary laterals was estimated and summed for the total number of years retained. All needle retention ratings were done in April or May, prior to bud break. Annual longitudinal branch increments were recorded for each of these sample branches, and the number of whorl and interwhorl secondary branches on the primary branch axis were recorded.

As would be expected, plot initial conditions varied with respect to Douglas-fir basal area, top height, foliage retention, crown sparseness (CL:SA), and basal area in other species, underscoring the need to consider covariates in the analysis. Various stand attributes were computed from the plot data, and five-year growth responses were tested for treatment effects by analysis of covariance performed as a regression model. Traditional covariates influencing stand growth were included, such as initial Douglas-fir basal area, total basal area, and site index. In addition, indicator variables were introduced for treatment and block effects. Because the treatment effect was expected to be exerted by its effect on foliage retention, both foliage retention and CL:SA were initially treated as response variables in randomized block ANOVA (with no covariates), but were then introduced as predictor variables in the model that accounted for fungicidal treatment effects.

Longitudinal branch growth over the 5-year post-treatment period was also analyzed for treatment effects by ANOVA, with separate analyses run for each of the six sample whorls (whorls five through ten). Age class needle retentions and secondary branch counts similarly were tested for treatment effects to provide more detailed information on crown response. Secondary branch tallies were expressed as number of interwhorl or whorl branches per annual shoot.

In May 2003, two growing seasons after fungicide applications stopped, 12 codominant trees in each experimental unit were felled. Three branches from the fifth or sixth whorl from the top of the tree were collected, and needle retention and pseudothecia density were measured. A 1-inch thick disk was cut from each tree at breast height. Radial growth and sapwood width were measured on four radii corresponding to the longest axis and the axis perpendicular to the longest on the disk.

Results and Discussion

Plot-level needle retention and CL:SA—Analysis of variance (ANOVA) tests were run to test for treatment and block effects on both plot-level needle retention and crown sparseness. ANOVA indicated that needle retention was significantly enhanced by fungicide treatment ($p<0.0001$), though site was not an influence. The difference in average needle retention on treatment and control plots within a block averaged 0.88 years, ranging from 0.62 to 1.09 years; in other words, treated plots within a block retain, on average, an additional 0.88 years of needles. Treatment thus increased the average needle retention from 1.94 years in control plots to 2.82 years in treatment plots.

Crown sparseness (CL:SA, the ratio of crown length to sapwood area (Maguire and Kanaskie 2002)) was also reduced by treatment ($p=0.0081$), but a significant block effect was evident as well ($p= 0.0005$). Approximately 88 percent of the variation in crown sparseness was accounted for by the combined effects of block and treatment.

Stem volume growth—Cubic volume growth was also influenced significantly by both treatment and block effects. Plot volume growth was significantly greater after fungicide treatment, and growth increased with greater initial Douglas-fir growing stock . Very little of the basal area in these plots was contributed by species other than Douglas-fir. As a result, total basal area was not influential on Douglas-fir volume growth. The site indicator variables indicated that volume growth was also influenced by site factors; that is, there was a significant block effect. Growth of stands treated with chlorothalonil averaged 35 percent higher than the control stands over the 5-year response period (breast height ages 12 to 19). Growth of treated stands averaged 60 percent greater than control stands when the response was limited to the final three years of the study (1997 to 2000). This difference may be attributed to the time required for treated trees to build leaf area. Details of the statistical models can be found in Mainwaring and others (in press).

Longitudinal branch growth—Branches from whorls five and six grew significantly greater in response to fungicide treatments, with branch five exhibiting enhanced growth for the full 5-year growth period since treatment as well as for the most recent 3-year period. Branch six exhibited enhanced growth for only the most recent 3-year growth period. Other branches from whorls seven through ten showed no significant treatment response, with the exception of greater growth for branch eight over the 5-year growth period (but not for the most recent 3-year growth period). Branch growth in whorls six through eight were also significantly influences by site, but branches from whorls nine and ten showed neither treatment nor site effects.

The significance of site in explaining 5-year branch growth of some of the lower branches is likely the result of stand density differences. These lower branches interact with branches of other trees within the canopy, particularly at relatively high stand density. Their growth is therefore likely to be limited more by within-canopy shading and physical abrasion than from foliar loss caused by *P. gaeumannii*. Hence, any past differences in stand density or in timing of pre-commercial thinning among sites can produce significant site effects. In addition, with each successive year of the experiment, the lower branches are less likely to receive significant fungicidal application.

Branch needle retention—The treatment response of whole tree needle retention (average value estimated from the upper, middle and lower crown third needle retention)

and whole branch needle retention (average needle retention on secondary laterals connected to sample branches from whorls five through ten) were tested by analysis of variance. Fungicide treatment had a significant effect on both whole tree needle retention ($p<0.0001$) and branch needle retention ($p=0.0011$). If the needle retention estimate from each sample branch were tested individually, the strength of the treatment effect would increase with height in the crown. This trend would suggest that branch needle retention may be a better measure of needle retention if it is taken high in the crown, and/or that the fungicide penetrates only a limited distance into the crown. This response is consistent with the significant longitudinal branch growth in only the highest whorls.

Branch counts—Branch counts were generally unaffected by treatment. However, branch-8 did exhibit a significant treatment effect. Interwhorl counts on branch eight showed a significant treatment effect for the last three and five years, while the whorl count on branch eight was significant only over the last three years. Assuming the treatment effect on branch-8 is not a Type II error, it may be a result of interactions among self-shading, nutrition, and inter-tree competition.

In contrast to the limited treatment effects, all branch counts (except the whorl count on branch-10) exhibited a significant site effect over five years. This response may help explain the high correlation between crown sparseness (CL:SA) and site. Whereas needle retention was highly correlated with treatment, needle retention is a characteristic of individual branches. Total crown leaf area, and thus crown sparseness, depends both on the needle retention of individual branches as well as the total number of branches. The visually fuller and healthier crowns of trees treated with fungicide apparently result from increased needle retention on individual branches rather than an increase in branch numbers.

Douglas-fir growth and foliage retention two growing seasons after cessation of spraying—Two growing seasons after spraying ceased, mean basal area growth per tree was greater in treated compared to control plots (figure 20). Some of this continued growth difference can be explained by the greater foliage retention in the treated plots compared to untreated plots (figure 21). Even though operational fungicide treatments are not completely effective at eliminating the pathogen, the treatments do provide for increased foliage retention beyond the period of time during which fungicide is being applied. This could result from suppression of the pathogen population within the treated plots, and subsequent slow re-invasion after treatments stop, or the overall lowering of infection rates and needle colonization which would increase needle retention.

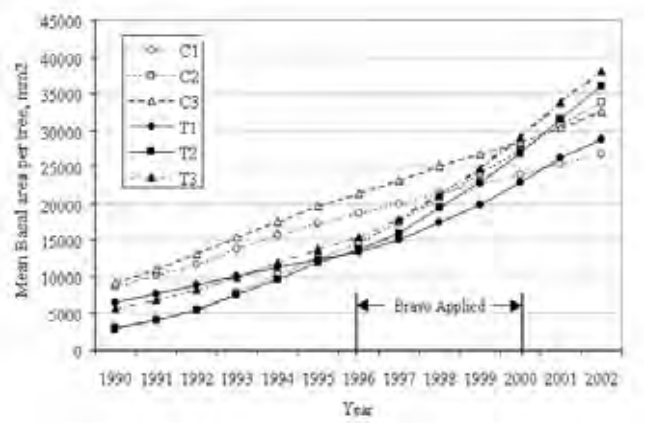

Figure 20—Mean basal area per tree (n=12 trees per plot) in plots treated with chlorothalonil for 5 years (1996 to 2000). The last fungicide treatment was applied just before the 2000 growing season.

Conclusions

The loss of volume growth associated with Swiss needle cast infection of coastal Douglas-fir is significant. Fungicidal suppression of the fungus with chlorothalonil has proven to diminish disease symptoms, and increase both foliage retention and volume growth. Sites used for this study showed a 35 percent volume growth increase after five years of treatment, but when confined to the final three years of the study, treated stands showed a 60 percent increase over control stands.

While the most obvious reason for decreased individual tree growth with severe SNC is accelerated foliage loss, this effect is compounded by delayed crown expansion, primarily the result of diminished longitudinal branch growth. On the stand level, the effects of diminished crown density and the diminished rate at which the crown fills available growing space further compounds the problem by enabling competitors in the understory to persist, or in some cases to re-initiate. This competitive pressure, focused below ground, is likely to be especially apparent on sites where below ground resources are limited such as the shallow soil of ridge tops or on drier, more exposed south slopes.

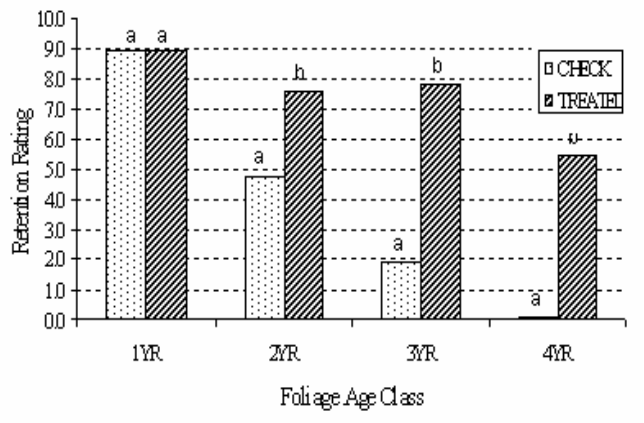

Figure 21—Mean needle retention per tree (n=12 trees per plot) in plots treated with chlorothalonil for 5 years (1996 to 2000). The last fungicide treatment was applied just before the 2000 growing season.

Application of the growth models to other stands is made possible by the association between treatment and crown characteristics. Because this model includes terms for both needle retention and CL:SA, it can be applied to similar, but untreated stands to assess their volume growth or the loss in volume growth resulting from their current condition. The utility of this model is enhanced by the fact that both foliage variables are relatively simple field measures.

Although the increase of volume growth with treatment is significant, widespread use of Bravo may not be operationally reasonable. The cost of two treatments per year was approximately $150 per acre, and subsequent field work suggests that treated stands have high infection rates in the year following suspension of treatment (J. Stone, personal communication).

Furthermore, Bravo has been found to be very toxic to fish (Davies and White 1985). Because of the economic, ecological, and aesthetic value of the fishery in the Coast Range, Oregon forest practice rules require that fungicides not be sprayed within 300 feet of fish-bearing streams or 60 feet of non-fish-bearing streams (Oregon Department of Forestry 1998). Because of the high stream density in the Coast Range, this would severely limit where this fungicide could be sprayed.

The significantly greater lateral branch growth in only the uppermost branches and the high correlation of branch needle retention with treatment in the same whorls would suggest that the size of the exposed crown is an important factor for efficient fungicide application. The latter result suggests that the fungicide does not reach deeply into the crown, yet crowns must be of sufficient size to produce merchantable timber. Because fungicide application would probably be limited to stands otherwise thought unable to reach merchantable size, thinning prior to a regeneration harvest would not be economically viable. Obviously, an efficient spray program would have to be coordinated closely with density control. A candidate stand would need to balance a closed canopy, desirable for maximized fungicide-foliage contact, with individual crowns of sufficient size to produce merchantable timber without the advantage of further thinning. The magnitude of the basal area growth increase and its persistence beyond the cessation of spraying suggests that fungicide applications might be economically feasible in certain situations.

Commercial Thinning Retrospective Study

A retrospective study was established in the fall of 2001 to assess the growth of Swiss needle cast (SNC)-infected Douglas-fir stands commercially thinned between four and nine years ago. The 24 study plots (aged 33 to 63) were distributed across six ODF districts (Tillamook, Forest Grove, Astoria, West Oregon, Coos, Clackamas–Marion), exhibiting site indices ranging from 96 to 144 feet (50 years), and foliage retention ranging from 1.8 to 4.38 years. Regression models were constructed to estimate periodic annual volume and basal area growth, annual percentage volume growth, and the post-thin/pre-thin basal area growth ratio.

Volume and basal area growth were found to decline with increasing intensity of SNC, as measured by current foliage retention and crown length to sapwood ratio (CL:SA). Compared with uninfected stands (represented by the Forest Grove district averages: 3.78 years foliage retention, 6.44 cm/cm2 CL:SA) thinned to a relative density of 35, plots with the lowest foliage retention (1.8 years) and highest CL:SA (13.5 cm/cm2) exhibited implied basal area growth losses as high as 55 percent, and volume growth losses as high as 38 percent.

Heavier thinning further decreased yield on the stand level, though at the lower densities, individual trees responded better.

The implied average annual percentage volume growth of the most heavily infected stands (foliage retention < 2.2 years) in the time since thinning was between 3.2 and 4.1 percent, depending on the value of CL:SA.

Stands thinned to a relative density of 35, and having a foliage retention below 2.3 years were found to have an implied post-thin/pre-thin basal area growth ratio of less than 1.0, indicating that basal area growth in the period prior to thinning was greater than that during the same

length period after thinning. Comparing the model-implied ratio from the lowest foliage retention (0.92, folret=1.8 years), to the average ratio calculated from two consecutive 3-year periods (both) just prior to thinning (0.88), suggests that at the lowest foliage retentions, there is no response; the growth of the residuals is only marginally better than the same trees would be in a higher density stand about to be thinned.

Results from this retrospective study make it possible to predict how previously thinned stands are currently growing. However, because foliage retention is known only for the end of the post-thin growth period, it can't be used to predict a future response at the time of thinning. Thinning has been observed to negatively effect foliage retention. If this is true, then the assumption that thinning doesn't affect foliage retention fails, and more intense thinning to enhance individual tree growth could actually reduce needle retention sufficiently to offset the expected growth acceleration. The second phase of the study, consisting of paired control and treatment plots, should rectify this problem. Details of the retrospective study can be found in Mainwaring and others (in press).

Acknowledgments

The aerial survey was conducted by the Oregon Department of Forestry Insect & Disease and Air Operations sections, and was funded by the Oregon State University Swiss Needle Cast Cooperative, the USDA Forest Service Forest Health Monitoring Program, and the Oregon Department of Forestry. Jim Baranek (ODF) piloted the plane. Mike McWilliams (ODF), Keith Sprengel (USFS), and Dave Overhulser (ODF) were the aerial observers.

Field work on permanent plots and thinning plots was completed and coordinated by Jon Laine (ODF Salem), John Beeson (ODF Salem), Steve Skinner (ODF Astoria), Matt Howard (ODF Astoria), Steve Dutton (ODF Tillamook), Jim Hines (ODF Tillamook), Dale Anders (ODF Forest Grove), Mike Totey (ODF Philomath), Tom O'Connor (ODF Philomath), Scott Malvitch (ODF Salem), Bryan Capitano (ODF Salem), Chet Smith (ODF Salem), Michael West (ODF Salem), Mark Gourley (Starker Forests), John Washburn (Simpson Timber), Rick Allen (Miller Timer Services), Ted Reiss (Miller Timber Services), Mark Montpas (Miller Timber Services).

The GIS data and a pdf file for the SNC aerial survey can be accessed via the ODF web page at: http://www.odf.state.or.us/fa/FH/maps.htm

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Dothistroma in Northwest British Columbia, Why There? Why Now?

Alex Woods

Abstract—Dothistroma needle blight caused by the fungus *Mycosphaerella pini* is causing severe damage to managed and natural stands of lodgepole pine in northwest BC. There appear to be two principle causes behind the damage occurring in this specific area, at this time. First, forest management policy and practice has lead to an unprecedented amount of young lodgepole pine hosts on the landscape. Second, the weather of the past decade appears to be changing resulting in more frequent consistent days of warm rain during summer months. The weather events coinciding with a previous outbreak of Dothistroma in the study area were investigated. The weather events that possibly lead to the decline in this previous Dothistroma, two consecutive years of dry conditions, have not reoccurred during the current epidemic.

Background

Dothistroma needle blight caused by the fungus *Mycosphaerella pini* is having a major impact on lodgepole pine stands, both natural and managed, in northwest British Columbia (BC). Over 90 percent of lodgepole-dominated managed stands have suffered some Dothistroma damage in the area. In almost all cases the disease situation appears to be worsening. I have yet to find a stand showing clear signs of recovery from this disease in the current epidemic. Instead, I am finding more examples of older stands, with trees aged 30 to 60 years, suffering severe defoliation and eventual death.

I believe there are two principle factors responsible for the current epidemic—1) forest management policy and practices which have resulted in species conversion to lodgepole pine stands over vast areas and 2) a suitable climate which has become even more favourable for foliar diseases such as Dothistroma.

Forest Management

I examined the species/age class distribution for the study area using the BC Ministry of Forest inventory database. I compared the species composition of all age classes combined, to the species composition of the managed stands (stands less than 20 years old in analysis) (figure 1). Lodgepole pine and interior spruce dominated stands comprise only about 10 percent each of the landbase when all age classes are combined. In the managed stands, however, these two species are the leading species in 80 percent of the landbase, 40 percent each. In other words, there is roughly four times the amount of lodgepole pine in the managed stands on a percentage basis than there was in the unmanaged forest area. This increase in host availability has undoubtedly had an influence on the creation of the current Dothistroma epidemic.

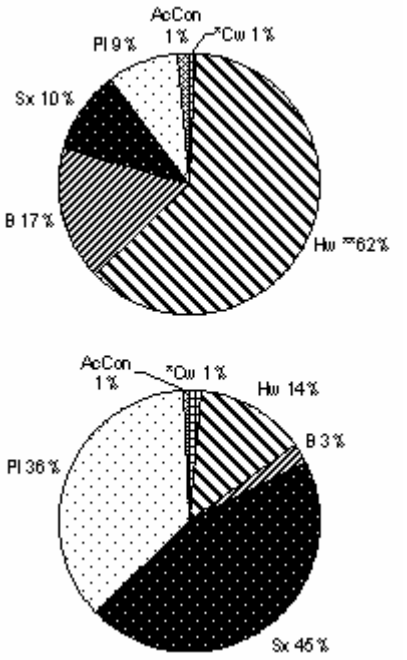

Figure 1—Species composition of all age classes combined (upper) compared to the species composition of managed stands <20 years old (lower).

Previous Disease Survey Reports of Dothistroma in Study Area

I reviewed past Canadian Forest Service, Forest Insect and Disease Survey (FIDs) annual survey reports for northwest BC for the period 1963–1995. The first record of Dothistroma in BC was in 1963, and the last survey conducted by FIDs was in 1995. The FIDs surveyors stated that they had identified a small but intense Dothistroma

In: Geils, B. W. comp. 2004. Proceedings of the 51st Western International Forest Disease Work Conference; 2003 August 18–22; Grants Pass, OR. Flagstaff, AZ: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station.

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infestation at Kisgegas Canyon within the study area, in their 1984 report. The report mentions that some mortality had occurred in the 8 m tall stand and that 20 percent of the trees were >80 percent defoliated. The fact that there was already mortality in the stand suggests that the disease was present for several years prior to their identification of the infestation. The FIDs survey report for 1986 stated that the Dothistroma infestation at Kisgegas Canyon appeared to be subsiding and that for the first time in four years the previous year's foliage was not being prematurely cast. There were no more records of Dothistroma in the area for the remainder of the period that FIDs surveys were conducted.

Growth Ring Analysis for Two Study Stands—Kisgegas Canyon and Sediesh Creek

I collected growth ring data from two study stands. Both sites contained a mixed stand of lodgepole pine and interior spruce. The Sedeish Creek stand was approx. 55 years old and the Kisgegas Canyon site contained some slightly older trees. At both sites pine growth rings showed a marked reduction in width in the early 1980s and a subsequent recovery until the early to mid 1990s at which point there has been a steady decline in increment (figure 2).

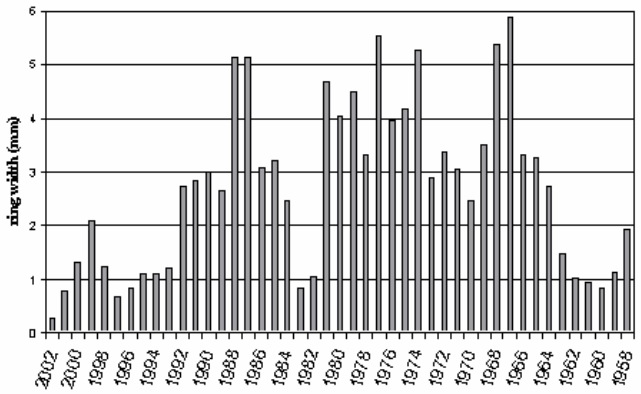


Figure 2—Annual growth ring width of lodgepole pine (Tree #6) at Sediesh Creek

The same growth reduction was not found in neighbouring spruce trees (figure 3) suggesting that the growth reduction was not likely due to abiotic factors.

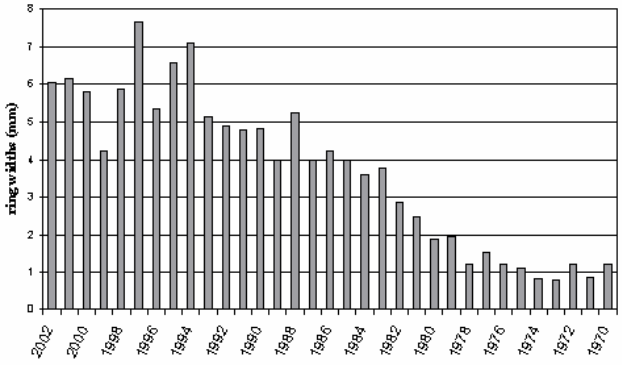


Figure 3—Annual growth ring widths of Interior spruce at Sediesh Creek

I believe the growth ring reduction in lodgepole pine was due to a previous Dothistroma blight infestation as was noted in the FIDs survey reports.

Favourable Climate

The second major factor that I believe is responsible for the current Dothistroma epidemic is climate. The climate in the study area was already favourable for foliar diseases as evidenced by the previous outbreak recorded by the FIDs survey. I obtained weather data from Environment Canada consisting of daily precipitation, maximum and minimum temperature and snow accumulation for the period 1950 to present for Smithers BC. Smithers was the closest weather station to the Kispiox study area. The climate in Smithers is slightly more continental and therefore somewhat drier and cooler than the Kispiox.

I analysed this data using Microsoft Access to look for trends in the weather that would fit the environmental requirements of Dothistroma. In particular I looked for consecutive days of warm rain that would be ideal for setting up an infestation. I also looked for weather conditions that may have contributed to the decline of the previous Dothistroma infestation in Kisgegas Canyon and Sediesh Creek.

The early 1980s were a time of weather extremes in the study area. In 1980, there were seven consecutive days of rain with average high temperatures over 16° C; only six such events have occurred over the past 53 years. In 1979 and twice in 1980, there were three consecutive nights of rain with temperatures averaging above 11° C. The only time in the past 53 years that such an event has occurred twice in one year is 1980.

The following year of 1981 was the driest year since 1950. Only four events consisting of eight consecutive days

without rain and average high temperatures above 27° C have occurred since 1950, and only in 1981 twice. The following year (1982) was also quite dry, and the period 1981–1982 is the only time that two consecutive years have had periods of eight consecutive days of no rain with highs over 26° C.

I believe the lodgepole pine trees at both Kisgegas Canyon and Sediesh Creek suffered severe damage from Dothistroma in 1980. Then the disease was subsequently held in check by two consecutive years of dry conditions. By the time the FIDs surveyors had identified the damage at Kisgegas Canyon, the infestation was likely already on the decline. The very poor radial growth in 1982 was very likely a direct reflection of the Dothistroma damage in 1980. The dry conditions in 1981–1982 were not sufficiently dry to cause any growth declines in neighbouring spruce trees, which if anything would have been more prone to drought stress than the lodgepole pine.

The weather during the growing season of 1997 was a major catalyst for the current Dothistroma epidemic. This year also marks the first time that I witnessed mortality due to Dothistroma in what were previously healthy vigorous lodgepole pine trees approximately 20-years old. Weather events consisting of five consecutive days of rain with average high temperatures above 18° C have occurred only four times since 1950, twice in 1997. The year 1997 had the third wettest June (91mm) and the second wettest July (99mm) in the past 53 years.

The weather since 1997 has continued to favour the spread and intensification of Dothistroma. The year 1999 included the wettest August since 1950; and the year 2002 included the third wettest September. Probably the best fit in terms of weather events between the initiation of the previous Dothistroma infestation and the current one is the occurrence of three consecutive nights of rain with temperatures not dropping below 11° C. The only time since 1979–1980, the time of the last outbreak, when such an event has occurred in successive years is the period 1998 to 2000.

This trend to wetter summers in the 1990s is most clearly shown when weather event consisting of three consecutive days of rain with high temperatures above 20° C are tallied by decade, since 1950 (figure 4).

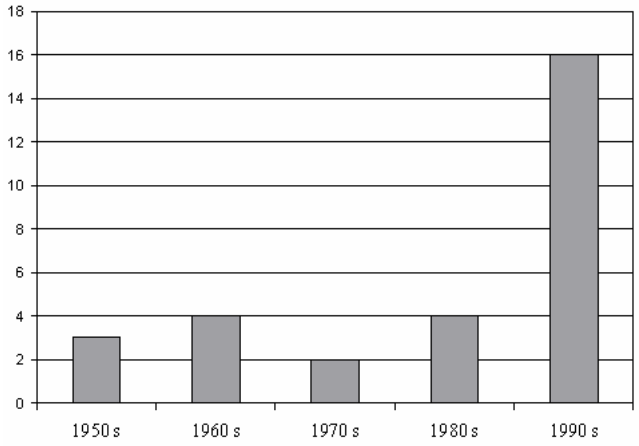


Figure 4—Frequency of weather events consisting of three consecutive days of rain with high temperatures averaging more than 20° C.

Conclusions

Whether this trend to wetter summer is climate change or simply a change in the weather is up for debate. The regional climate models for northwest BC do forecast wetter summers as part of their overall predictions of climate change. The weather does appear to have become more favourable for foliar diseases such as Dothistroma. This change in the weather coupled with a large increase in susceptible host, due to forest management policy and practice, provide a logical explanation for the current Dothistroma epidemic in northwest BC. The management of lodgepole pine as a timber species in the study area has been halted as a result of the current epidemic.

If it's a Doug-fir, it's probably a Doug-fir mistletoe.

The principle and only commonly infested host of *Arceuthobium douglasii* is Douglas-fir (Oregon Pine). Douglas-fir is rarely infected by *A. americanum* and *A. tsugense*. The shoots of Douglas-fir dwarf mistletoe are the smallest in North America (mean 2 cm) and branch in a flabellate or fan-shaped manner; systemic brooms (may be very large) are often evident whereas shoots are often obscure or absent. Shoots of *A. americanum* are about 5 to 9 cm tall (or more) and branch in a verticillate or whorled pattern. Shoots of *A. tsugense* are flabellate and 5 to 7 cm tall.

Special Papers

Jim Worrall, Moderator

Program

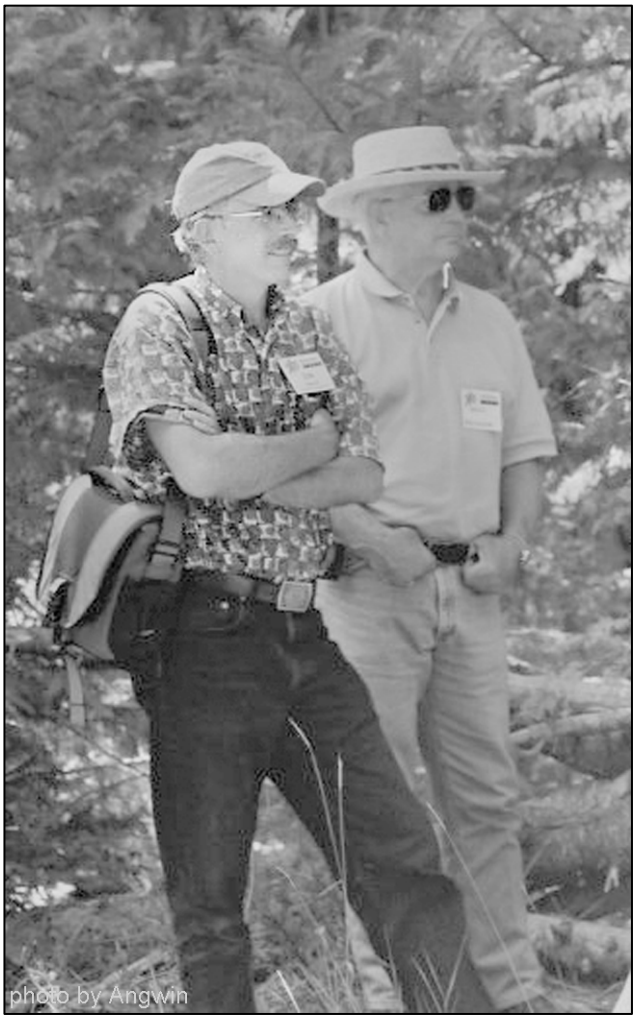
Early thinning in mixed-species plantations of Douglas-fir, hemlock, true fir and ponderosa pine affected by Armillaria root disease in central Oregon and Washington: 20 to 30 year results. Gregory M. Filip (presenter), Pacific Northwest Region, Portland, OR; Lisa Ganio, Oregon State University, Corvallis; and Stephen Fitzgerald, Oregon State University, Redmond.

Shore pine dwarf mistletoe: Should it be classified as a subspecies or a race of hemlock dwarf mistletoe? Ed F. Wass, Pacific Forestry Centre, Victoria, BC and Robert L. Mathiasen (presenter), Northern Arizona University, Flagstaff, AZ.

Long-term monitoring of tree damage caused by porcupine feeding in the Khutzeymateen Inlet. Stefan Zeglen (presenter) and Alex Woods, BC Ministry of Forests, respectively Nanaimo and Smithers, BC.

Fire and dwarf mistletoe. Robert Tinnin, Portland State University, Portland, OR.

Armillaria root disease in campgrounds of southern Colorado. Jim Worrall (presenter), Kelly Sullivan, Rocky Mountain Region, respectively Gunnison and Lakewood, CO; Tom Harrington and Joe Steimel, Iowa State University, Ames, IA.



The spruce is Brewer spruce, but what is this mistletoe?

The common and endemic spruce of the Siskiyou Mountains is Brewer or weeping spruce (the branches and twigs are droopy). Engelmann spruce is another montane spruce in the general region; its branches are spreading and only somewhat drooping but the twigs are stiff. Sitka spruce is coastal. Although Brewer spruce is not the principle host for any dwarf mistletoe, it is a secondary host for *A. abietinum* f. sp. *concoloris*, an occasional host for *A. monticola*, and a rare host for *A. tsugense* subsp. *mertensianae*. Therefore, in context, the mistletoe is probably the same as found in the associated white fir, white pine, or mountain hemlock. Morphologically, the best way to distinguish among these mistletoes is by shoot color: the fir dwarf mistletoe is the most yellowish, the hemlock dwarf mistletoe is greenish to reddish; the white pine dwarf mistletoe is dark brown.

Early Thinning in Mixed-Species Plantations of Douglas-fir, Hemlock, True Fir and Ponderosa Pine Affected by Armillaria Root Disease in Central Oregon and Washington: 20 to 30 Year Results

Gregory M. Filip, Lisa M. Ganio, and Stephen A. Fitzgerald

Methods

Four 10- to 20-year-old plantations were treated to determine the effects of precommercial thinning on tree growth and mortality caused by *Armillaria ostoyae* in the Cascade Range of western Oregon and Washington. One plantation was of Douglas-fir and noble fir, one of Douglas-fir and western hemlock, one of Douglas-fir alone, and one of Shasta red fir and mountain hemlock. Four paired 0.25-acre square plots were established in each plantation. Paired plots were initially located so that approximately equal amounts of root disease (tree mortality) were present in each plot pair. Quadratic mean diameter and basal area/acre were calculated for trees in one 0.05-acre circular plot established in the center of each square plot. Mortality was determined every other year and diameter every decade.

Results

After 20 years, differences in crop tree mortality between thinned and unthinned plots were not significant (P=0.9768). Quadratic mean diameter growth of crop trees, however, was significantly (P=0.0053) greater in thinned than in unthinned plots (figure 1). Crop tree basal area/acre growth was significantly (P=0.0008) greater in thinned plots (figure 2). There were no significant (P=0.6647) differences in basal area/acre growth of all trees between thinned and unthinned plots.

In a 30-year-old stand of ponderosa pine affected by *Armillaria ostoyae*, four 0.25-acre square plots were established in 1966, treated, and data collected as above. Crop tree mortality was significantly (P=0.02) less in thinned plots than in unthinned plots after 30 years (figure 3). Tree diameter growth was not significantly (P=0.17) increased by thinning. Crop tree basal area/acre growth was significantly (P=0.03) greater in thinned plots. Apparently, from a root-disease perspective, precommercial thinning does not affect incidence of crop-tree mortality after 20 to 30 years, but per acre tree growth increases significantly.

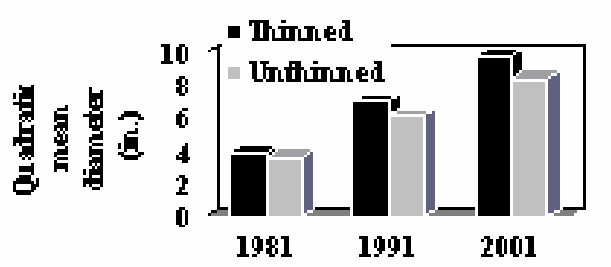


Figure 1—Quadratic mean diameter growth, all fir/hemlock sites.

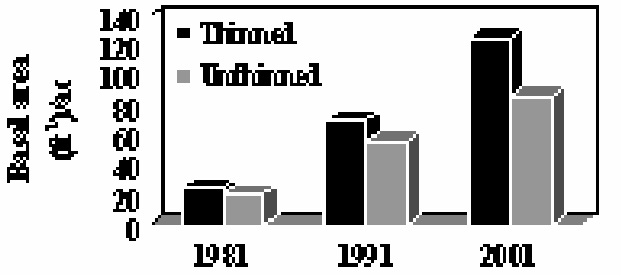


Figure 2—Basal area per acre growth, all fir/hemlock sites.

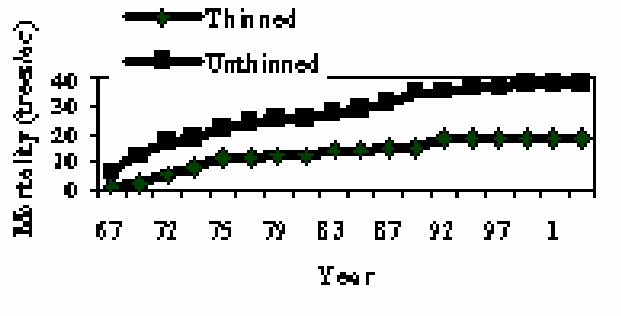


Figure 3—Cumulative crop-tree mortality on ponderosa site.

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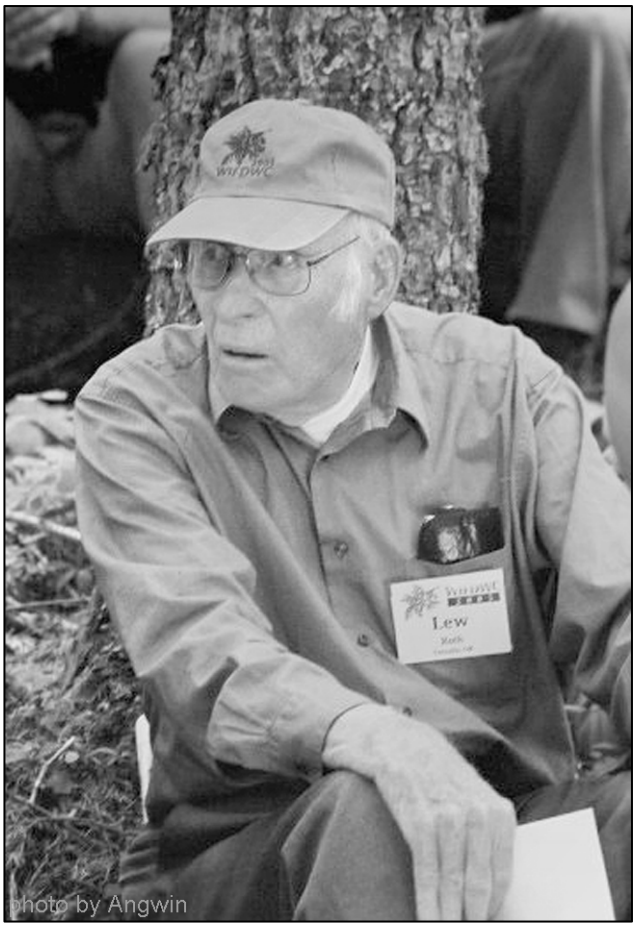
Filip, Gregory M.; Goheen, Donald J. 1995. Precommercial thinning in *Pseudostuga*, *Tsuga*, and *Abies* stands affected by armillaria root disease: 10-year results. Canadian Journal of Forest Research. 25(5):817–823.

Filip, Gregory M.; Goheen, Donald J.; Johnson, David W.; Thompson, John H. 1989. Precommercial thinning in a ponderosa pine stand affected by armillaria root disease: 20 years of growth and mortality in central Oregon. Western Journal of Applied Forestry. 4(2):58–59.

Filip, Gregory M.; Fitzgerald, Stephen A.; Ganio, Lisa M. 1999. Precommercial thinning in a ponderosa pine stand affected by armillaria root disease in central Oregon: 30 years of growth and mortality. Western Journal of Applied Forestry. 14(3):144–148.

Johnson, David W.; Thompson, John H. 1975. Effect of precommercial thinning on ponderosa pine, *Pinus ponderosa*, infected with *Armillaria mellea*. Plant Disease Reporter. 59:308–309.



The fir dwarf mistletoes appear to be identical, but they are faithful to their host.

The two taxa of fir dwarf mistletoes, *A. abietinum*, can not be distinguished by morphology or phenology but the mistletoe produced on white fir does not cross-infect red fir and the mistletoe produced on red fir does not cross-infect white fir. These are designated as formae speciales—*concoloris* (white fir) and *magnificae* (red fir). White fir has a wide, western distribution; it is distinguished from the red fir by resinous buds and two-ranked leaves. Red fir is found in the Sierra region; in Jefferson, the Shasta red fir has exserted, reflexed bracts over the cone scales and is intermediate between typical red fir and Noble fir. (see treatment of *Abies* by Rich Hunt in Flora of North America, Volume 2).

Shore Pine Dwarf Mistletoe: Should It Be Classified as a Subspecies or a Race of Hemlock Dwarf Mistletoe?

Ed F. Wass and Robert L. Mathiasen

Abstract—The dwarf mistletoe parasitizing shore pine in British Columbia, Canada and Washington, U.S.A. is described as a subspecies of hemlock dwarf mistletoe, *Arceuthobium tsugense*, based on morphology and differences in host susceptibility of western hemlock and shore pine to the shore pine dwarf mistletoe, *Arceuthobium tsugense* subspecies *contortae*.

Introduction

The taxonomic classification of the dwarf mistletoe parasitizing shore pine (*Pinus contorta* Douglas ex Louden var. *contorta*) in British Columbia and Washington has long been discussed (Gill 1935; Hunt and Smith 1978; Hawksworth 1987; Hawksworth and Wiens 1972, 1996; Nickrent and Stell 1990; Hawksworth and others 1992; Nickrent and others 1994). In their monograph of *Arceuthobium*, Hawksworth and Wiens (1972) classified the dwarf mistletoe parasitizing shore pine as hemlock dwarf mistletoe (*Arceuthobium tsugense* (Rosendahl) G.N. Jones), whose principal host is western hemlock (*Tsuga heterophylla* (Rafael) Sargent). Hawksworth (1987) summarized the taxonomy of hemlock dwarf mistletoe and separated this dwarf mistletoe into three different races—a western hemlock race, a shore pine race, and a mountain hemlock race. Hawksworth and others (1992) presented yet another interpretation for the classification of hemlock dwarf mistletoe. They described the mountain hemlock race proposed by Hawksworth (1987) as a subspecies of hemlock dwarf mistletoe (mountain hemlock dwarf mistletoe as *A. tsugense* (Rosendahl) G. N. Jones subsp. *mertensianae* Hawksworth & Nickrent), but maintained the dwarf mistletoe parasitizing shore pine as a race of western hemlock dwarf mistletoe. In a revision of their monograph on *Arceuthobium*, Hawksworth and Wiens (1996) maintained the race designation for the dwarf mistletoe on shore pine.

In 1997, we began intensive taxonomic studies of hemlock dwarf mistletoe throughout its geographic range. Below, we discuss our work on the shore pine dwarf mistletoe and compare our findings with those of previous workers. We have used the designations western hemlock dwarf mistletoe (WHDM), shore pine dwarf mistletoe (SPDM), and mountain hemlock dwarf mistletoe (MHDM) below, instead of referring to these mistletoes as subspecies or races of hemlock dwarf mistletoe.

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Methods

Morphology

Eleven SPDM populations scattered throughout its principal range in British Columbia and on Orcas Island, WA were sampled (figure 1). Six WHDM populations from within the geographic range of SPDM and 14 other WHDM populations were also sampled so comparisons of morphological characters could be made between these mistletoes (figures 1 and 2). From each population, 20 infections (ten male and ten female) were collected and the dominant shoot from each infection was used for the following morphological measurements (Hawksworth and Wiens 1996)—1) height, basal diameter, third internode length and width, and color of the tallest male and female shoot from each infection collected; 2) mature fruit length, width, and color; 3) seed length, width and color; 4) staminate flower diameter; 5) number, length and width of staminate perianth lobes; 6) anther distance from the perianth lobe tip; 7) anther diameter; and 8) pre-flowering lateral staminate spike length. Analysis of variance (ANOVA), using a general linear model procedure for unbalanced designs, was used to determine significant differences between SPDM and WHDM morphological measurements ($F < 0.05$).

Host Susceptibility

Hawksworth and Wiens (1972, 1996) established five natural susceptibility classes based on the percentage of trees infected within 6 m of severely infected hosts of dwarf mistletoes—1) principal (90 percent to 100 percent infection), 2) secondary (50 percent to 90 percent infection), 3) occasional (5 percent to 50 percent infection), 4) rare (trace to 5 percent infection), and 5) immune (no infection). In order to determine the susceptibility of shore pine and western hemlock to SPDM and WHDM, temporary plots 6 m in radius (0.04 ha) were established around large, severely infected residual trees (western hemlock or shore pine) in or near areas where dwarf mistletoe samples were collected for morphological measurements (figure 1). Plots were completed in areas with mistletoe and tree

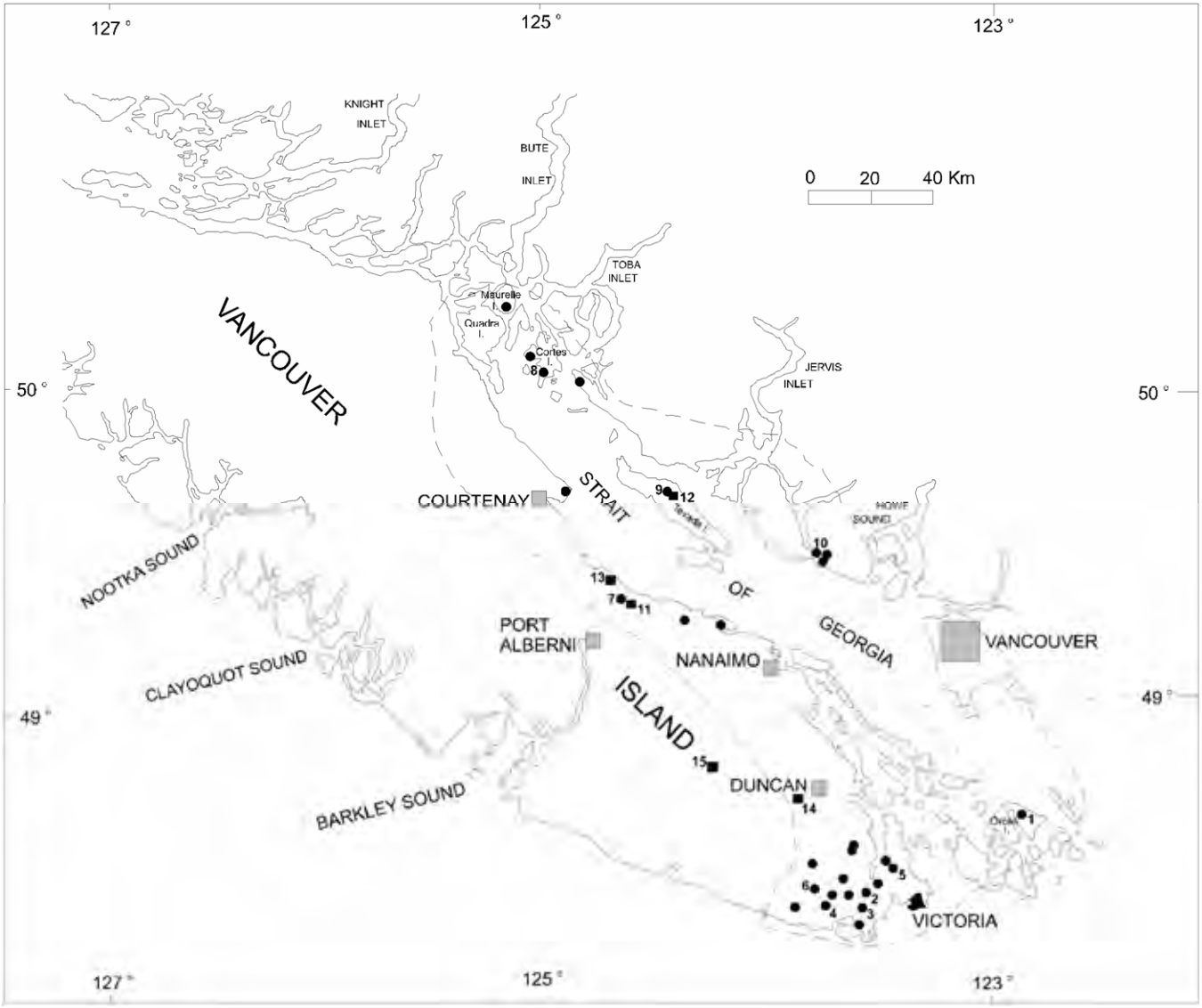

Figure 1—Dashed line encloses the distribution of *Arceuthobium tsugense* subsp. *contortae*. Black circles indicate known populations and numbered circles indicate populations sampled for morphological measurements and host susceptibility, Washington—1) Orcas Island; British Columbia—2) Mount Wells, 3) Mount Helmcken, 4) Bluff Mountain, 5) Mount Work, 6) Trap Mountain, 7) Spider Lake, 8) Cortes Island, 9) Texada Island, 10) Sechelt. Black squares indicate populations of *Arceuthobium tsugense* subsp. *tsugense* sampled for morphological measurements and host susceptibility, British Columbia—11) Spider Lake, 12) Texada Island, 13) Bowser, 14) Holt Creek, 15) Caycuse Summit.

compositions that fell into four categories—1) SPDM in pure shore pine forests; 2) SPDM in shore pine forests mixed with some western hemlock; 3) WHDM in pure western hemlock forests, and 4) WHDM in western hemlock forests mixed with some shore pine.

In each plot, trees greater than 1.37 m in height were sampled and the following data recorded for each tree: species, diameter at breast height (d.b.h., 1.37 m above the ground), condition (living or dead), and dwarf mistletoe rating (DMR) (Hawksworth 1977). Dwarf mistletoe ratings were only assigned to dead trees when an accurate rating was possible.

Distribution

To confirm the distributions of these dwarf mistletoes, we visited reported locations or checked disease reports from the Canadian Forest Service, Pacific Forest Research Centre of shore pine dwarf mistletoe from northern British Columbia (Queen Charlotte Islands, Malcolm Island, and near Terrace) and examined reported populations of western California recorded in Hawksworth and Wiens (1996).

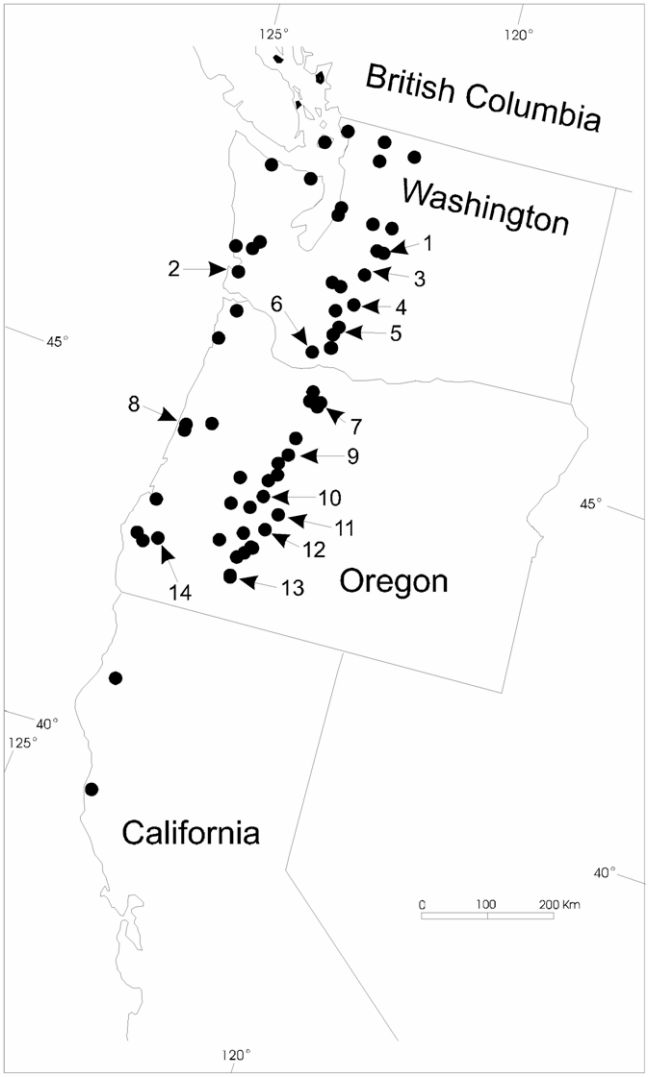

Figure 2—Distribution of *Arceuthobium tsugense* subsp. *tsugense* in Washington, Oregon and California (Hawksworth and Wiens, 1996). Black circles indicate known populations and numbered circles indicate populations where morphological data were collected. Washington—1) Snoqualmie Pass, 2) Westport, 3) Huckleberry Creek, 4) Cortright Creek, 5) Clearwater Creek, 6) Wind River Experimental Forest; Oregon—7) Wapinitia Pass, 8) Desolation Saddle; 9) Huckleberry Creek, 10) Indigo Spring; 11) Wall Creek, 12) Calapooya Ridge, 13) Union Creek, 14) Iron Mountain.

Phenology

Observations of flowering and seed dispersal were made during the summer and fall of 1972, 1973, 1997, and 1998 for both SPDM and WHDM near Horne Lake on Vancouver Island (locations 7 and 8 on figure 1). The sites for both dwarf mistletoes were similar in aspect, climate, physical geography and elevation. In 1973, observations were also taken for SPDM on Hill 22 (location 16 on figure 1) and for WHDM on the southwest side of Sooke Lake (location 17 on Figure 1) near Victoria, British Columbia. The ecological characteristics of these sites have been previously described by Wass (1976). At each site, individual plants were tagged and male flowering and seed dispersal were observed from early July through late November. Flowering and seed dispersal were classed into five categories—1) not yet started; 2) started but not at peak; 3) at peak (most flowers open or most mature fruit dispersing seed); 4) past peak but not yet completed (almost all flowers open or seed dispersed); and 5) completed.

Results

Morphology

Male plants of SPDM were significantly shorter (mean 5.6 cm) than those of WHDM (mean 7.8 cm). Although the range in male plant height of the two mistletoes overlaps slightly, the tallest plants of SPDM do not reach the maximum heights of WHDM (table 1). Shoot third internode length was significantly shorter and third internode width was significantly wider for SPDM than for WHDM (table 1). The flowers of SPDM are larger than those of WHDM; staminate flower diameter, perianth length and perianth width was significantly larger for SPDM than for WHDM. Another difference between SPDM and WHDM is plant color; male plants of SPDM are predominately green-brown whereas male plants of WHDM are predominantly green-yellow. For both mistletoes, the number of perianth lobes on male flowers is three or four, but the predominance of 3-merous or 4-merous flowers varies between populations.

Female plants of SPDM were significantly shorter (mean 6.6 cm) than those of WHDM (mean 8.0 cm) (table 2). Again, the range in heights of the dominant female plants overlaps for these mistletoes, but the height of SPDM female plants does not reach the size of WHDM (table 2). Shoot third internode length was significantly shorter for SPDM than for WHDM. The fruit width and seed width of SPDM was significantly wider than for WHDM. The color of female plants of SPDM is predominantly green-brown, while the female plants of WHDM are green-brown, green-yellow, or purplish. Fruit and seed color is similar for both mistletoes, as is seed length (table 2).

Host Susceptibility

A total of 173 plots were completed at ten locations (figure 1). Ninety-nine percent of the shore pine sampled (765 trees) in the plots located in shore pine forests was infected by SPDM (table 3). This high level of infection demonstrates the high level of susceptibility of shore pine to this mistletoe. The mean DMR of shore pine in these forests was 5.0 indicating that the trees in the plots we sampled were severely infected.

Table 1—Morphological characteristics of male plants for shore pine and western hemlock dwarf mistletoes for collections from British Columbia and the United States.

Male Plant Characters*	Shore Pine Dwarf Mistletoe			Western Hemlock Dwarf Mistletoe			Probability
	Mean	Range	N/n**	Mean	Range	N/n**	> F
Tallest shoot length	5.6	3.2–10.8	11/110	7.8	3.4–16.1	20/270	0.0001
Shoot basal diameter	2.8	1.7–4.7	"	2.6	1.3–5.0	20/269	0.2104
Shoot third internode length	9.2	5.8–15.7	"	11.8	4.5–23.0	20/270	0.0001
Shoot third internode width	1.8	1.1–2.5	"	1.6	0.8–3.0	"	0.0111
Third internode length/width ratio	5.3	2.9–8.8	"	7.5	2.7–15.0	"	0.0001
Staminate spike length	12.6	5.0–22.0	"	10.8	5.0–27.4	16/260	0.1590
Staminate flower diameter	4.3	2.8–5.9	"	3.6	2.2–6.4	"	0.0056
Anther diameter	0.7	0.4–1.2	"	0.7	0.3–1.3	"	0.7650
Staminate perianth length	1.8	1.2–2.8	"	1.5	1.0–2.2	"	0.0015
Staminate perianth width	1.4	1.0–2.3	"	1.2	0.8–2.2	"	0.0248
Anther distance from perianth tip	0.9	0.4–1.5	"	0.5	0.2–1.2	"	0.0002

*Shoot length—measured in cm other characters measured in mm.
**N/n—Number of populations sampled over number of individual measurements.

Table 2—Morphological characteristics of female plants for shore pine and western hemlock dwarf mistletoes for collections from British Columbia and the United States.

Female Plant Characters*	Shore Pine Dwarf Mistletoe			Western Hemlock Dwarf Mistletoe			Probability
	Mean	Range	N/n**	Mean	Range	N/n**	> F
Tallest shoot length	6.6	4.0–9.5	11/110	8.0	3.8--13.7	20/265	0.0031
Shoot basal diameter	3.3	1.8–5.0	"	2.7	1.3--5.5	"	0.0214
Shoot third internode length	10.7	5.3–16.4	"	12.3	6.0--22.0	"	0.0050
Shoot third internode width	1.7	1.3–2.5	"	1.6	1.0--3.1	"	0.2584
Third internode length/width ratio	6.2	3.6–9.4	"	7.8	3.3--16.0	"	0.0007
Fruit length	4.6	3.3–5.7	"	4.4	3.3--5.5	18/210	0.2058
Fruit width	3.1	2.1–4.2	"	2.9	2.2--3.5	"	0.0363
Seed length	2.5	1.8–3.0	10/100	2.6	1.8--3.5	17/200	0.1193
Seed width	1.4	1.0–1.7	"	1.1	0.8--1.4	"	0.0001

* Shoot length—measured in cm other characters measured in mm.
**N/n—Number of populations sampled over number of individual measurements.

In forests composed of mixtures of shore pine and western hemlock infested with SPDM, 95 percent of the shore pine was infected with a mean DMR of 3.7. Out of the 802 western hemlock sampled in these forests, only 21 percent were infected by SPDM (table 3). This places western hemlock in the occasional host susceptibility class of Hawksworth and Wiens (5 to 50 percent infection). The lower susceptibility of western hemlock to SPDM is also demonstrated by the extremely low mean DMR of 0.3 for western hemlock in these plots.

In forests of mixed shore pine and western hemlock infested with WHDM, 96 percent of the 138 western hemlock sampled were infected and the mean DMR was 4.3 (table 3). In contrast, WHDM infection of the shore pine sampled (77 trees) was 1 percent and mean DMR for these trees 0.01. These infection incidences demonstrate the high susceptibility of western hemlock to WHDM (a principal host) and very low susceptibility of shore pine (a rare host).

Distribution

Shore pine dwarf mistletoe occurs on Vancouver Island and other islands of British Columbia as far north as Maurelle Island and on the mainland coast of British Columbia from south of Sechelt to Powell River (figure 1). In Washington, it only occurs on Orcas Island (figure 1). The known elevation range for SPDM is from sea level to 800 m.

There are also reports of SPDM near Port Clements on the Queen Charlotte Islands, on Malcolm Island, and near Terrace, British Columbia (Smith and Wass 1976; Wass 1976; Hawksworth, 1987). These represent possible populations of SPDM that are approximately 400 to 500 km north of the northern most populations on Vancouver Island or the mainland coast of British Columbia. These reports are based on tree disease collections made by personnel from the Canadian Forest Service, Pacific Forest Research Centre. Examination of the dwarf mistletoe collection records for the Port Clements and Malcolm Island sites and a visit to the Terrace site indicated that these sites are actually rare crossovers of WHDM onto shore pine. Therefore, the northern range limit of SPDM is still Maurelle Island (figure 1).

Table 3—Infection of shore pine and western hemlock by shore pine dwarf mistletoe and western hemlock dwarf mistletoe.

Dwarf Mistletoe	Forest Type	Shore Pine			Western Hemlock		
		Trees Sampled	Percent Infected	Mean DMR	Trees Sampled	Percent Infected	Mean DMR
Shore Pine	Pure Shore Pine	765	99.2	5.0			
	Shore Pine and Western Hemlock mix	811	95.1	3.7	802	20.7	0.3
Western Hemlock	Western Hemlock and Shore Pine mix	77	1.3	0.1	138	96.4	4.3

We did not locate WHDM populations that extended the geographic range of this mistletoe. However, we did locate several populations that extend the upper elevation limit of WHDM to well over 1500 m in Oregon. Thus far, WHDM ranges in elevation from near sea level at several locations in British Columbia, Washington, Oregon, California, and southeast Alaska to as high as 1735 m in the Calapooya Mountains north of Diamond Lake in south central Oregon. In the Calapooya Mountains, WHDM is sympatric with MHDM in several locations.

Phenology

Flowering periods of SPDM and WHDM varied a great deal during the four observation years (figure 3). Flowering typically began from mid July to the first week in August for both mistletoes. Flowering started at approximately the same time for both mistletoes in two of the observation years (1972 and 1997). However, flowering for SPDM was completed earlier than WHDM in three of the four observation years (figure 3). Peak flowering periods were similar for both mistletoes in 1972 and 1997. In 1973, the peak of flowering was one week later for SPDM; while in 1998, it was one week earlier than WHDM (figure 3).

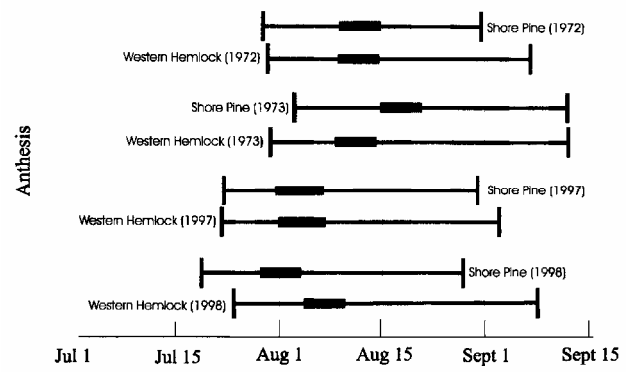

Figure 3—Flowering periods for shore pine dwarf mistletoe and western hemlock dwarf mistletoe. Peak periods are shown by solid bars.

Seed dispersal began earlier for SPDM in two (1997 and 1998) of the four observation years (figure 4). The sequence of initial seed dispersal and completion varied a great deal for these dwarf mistletoes. However, in 1972, 1973, and 1997, SPDM seed dispersal peaked one week ahead of WHDM (figure 4). In 1998, SPDM started seed dispersal nearly two weeks before WHDM and ended a week earlier. However, peak seed dispersal was during the second week of October for both mistletoes that year.

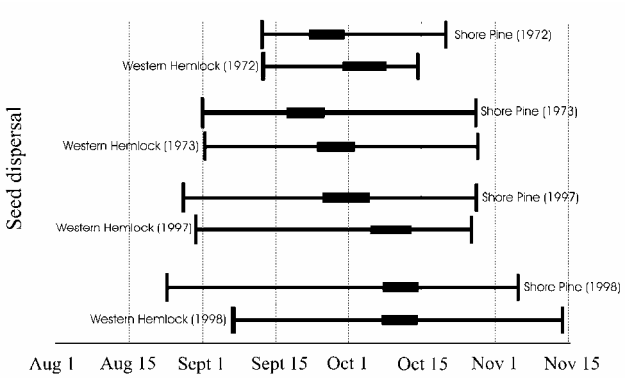

Figure 4—Seed dispersal periods for shore pine dwarf mistletoe and western hemlock mistletoe. Peak periods are shown by solid bars.

Discussion

Based on our comparisons of the morphology, phenology, and host range of SPDM, we recommend that it be classified as a subspecies of hemlock dwarf mistletoe (*Arceuthobium tsugense* subsp. *contortae*), instead of treated as a race of WHDM. Table 4 summarizes the principal differences between SPDM and WHDM.

Plants of SPDM and WHDM are morphologically similar, but they also have several consistent morphological differences. Male and female plants of SPDM are consistently smaller than WHDM. In addition, the color of male SPDM plants is frequently green-brown and occasionally yellow-green; while the color of WHDM male plants is consistently yellow-green. Staminate flowers of SPDM are consistently larger than the flowers of WHDM. Because we sampled several populations of both dwarf mistletoes and completed at least 100 measurements of the morphological characters selected for analysis, we feel the morphological differences we found between SPDM and WHDM are consistent and are taxonomically important discontinuities.

The flowering and seed dispersal periods for these mistletoes are similar also, but distinct differences occur. Flowering occurs from mid-July to mid-September for both mistletoes. Peak flowering periods fluctuate from year to

Table 4—Principal morphological and physiological differences between shore pine dwarf mistletoe (SPDM) and western hemlock dwarf mistletoe (WHDM).

Character	Shore Pine Dwarf Mistletoe	Western Hemlock Dwarf Mistletoe
Mean Plant Height, ♂	5.6 cm	7.8 cm
Mean Plant Height, ♀	6.6 cm	8.0 cm
Mean Length 3rd Internode, ♂	9.2 mm	11.8 mm
Mean Length 3rd Internode, ♀	10.7 mm	12.3 mm
Mean Flower Diameter	4.3 mm	3.6 mm
Plant Color, ♂	Greenish brown	Yellowish green
Plant Color, ♀	Greenish brown	Yellowish green/purple
Susceptibility, Shore Pine	Principal Host	Occasional Host
Susceptibility, Western Hemlock	Rare Host	Principal Host
Peak Seed Dispersal	One week earlier than WHDM	One week later than SPDM

year, but peaks usually occur for approximately one week in early to mid August. In the same forests, flowering started earlier for SPDM than for WHDM in two of the four years we observed flowering; but WHDM started flowering before SPDM the other two years. The start and finish of seed dispersal were similar for both mistletoes as well, except in 1998 when SPDM started and ended earlier than WHDM. However, peak seed dispersal usually occurs a week earlier for SPDM, even when these mistletoes occur in the same area. Therefore, there are slight differences in the phenology of these two dwarf mistletoes when they occur in the same area and at the same elevation.

The principal host of SPDM is definitely shore pine, and not western hemlock, which we classify as an occasional host of SPDM using the host susceptibility system of Hawksworth and Wiens (1972, 1996). The much lower susceptibility of western hemlock to SPDM is also demonstrated by low mean DMR ratings for western hemlock in stands infested with only SPDM. The SPDM infections produce fusiform swellings on shore pine with the absence of typical broom formation, which is characteristic of WHDM on western hemlock. In addition, SPDM infections on western hemlock have unusually poor shoot development and large swellings, indicative of host parasite incompatibility (Smith and Wass 1979; Hawksworth and Wiens 1972, 1996). When exposed to WHDM in mixed western hemlock/shore pine forests, shore pine is a rare host of this mistletoe. These large differences in susceptibility between these hosts represent distinct and consistent physiological discontinuities between these mistletoes and represent taxonomically important characters separating them.

Hawksworth and Wiens (1972, 1996) defined subspecies in *Arceuthobium* as "geographically restricted populations delimited by a few relatively small but consistent variations." Although the distributions of the SPDM and WHDM overlap, they have consistent morphological differences between them, clearly demonstrate different levels of parasitism on shore pine and western hemlock, and flower and disperse seed at slightly different times when they occur in the same area. Because of these morphological and physiological differences, the SPDM should be recognized as a subspecies of hemlock dwarf mistletoe based on Hawksworth and Wiens' concept of subspecies in *Arceuthobium*.

Furthermore, giving SPDM taxonomic status as a subspecies is more consistent with the Hawksworth and others (1992) classification of MHDM as a subspecies of hemlock dwarf mistletoe. Our field studies in the Calapooya Mountains of south central Oregon indicate that MHDM is not geographically restricted from populations of WHDM. In addition, only a few morphological and physiological differences distinguish MHDM from WHDM (Hawksworth and others 1992, Hawksworth and Wiens 1996). Differences in plant size, phenology, and host range are the principal characters used by Hawksworth and others (1992) to separate MHDM from WHDM. These are the same characters that distinguish SPDM from WHDM. Therefore, the classification of SPDM as a subspecies of hemlock dwarf mistletoe (*Arceuthobium tsugense* subsp. *contortae*), instead of a race, is more consistent with the taxonomic treatment of hemlock dwarf mistletoe proposed by Hawksworth and others (1992) as well as the taxonomic framework for *Arceuthobium* established by Hawksworth and Wiens (1972, 1996).

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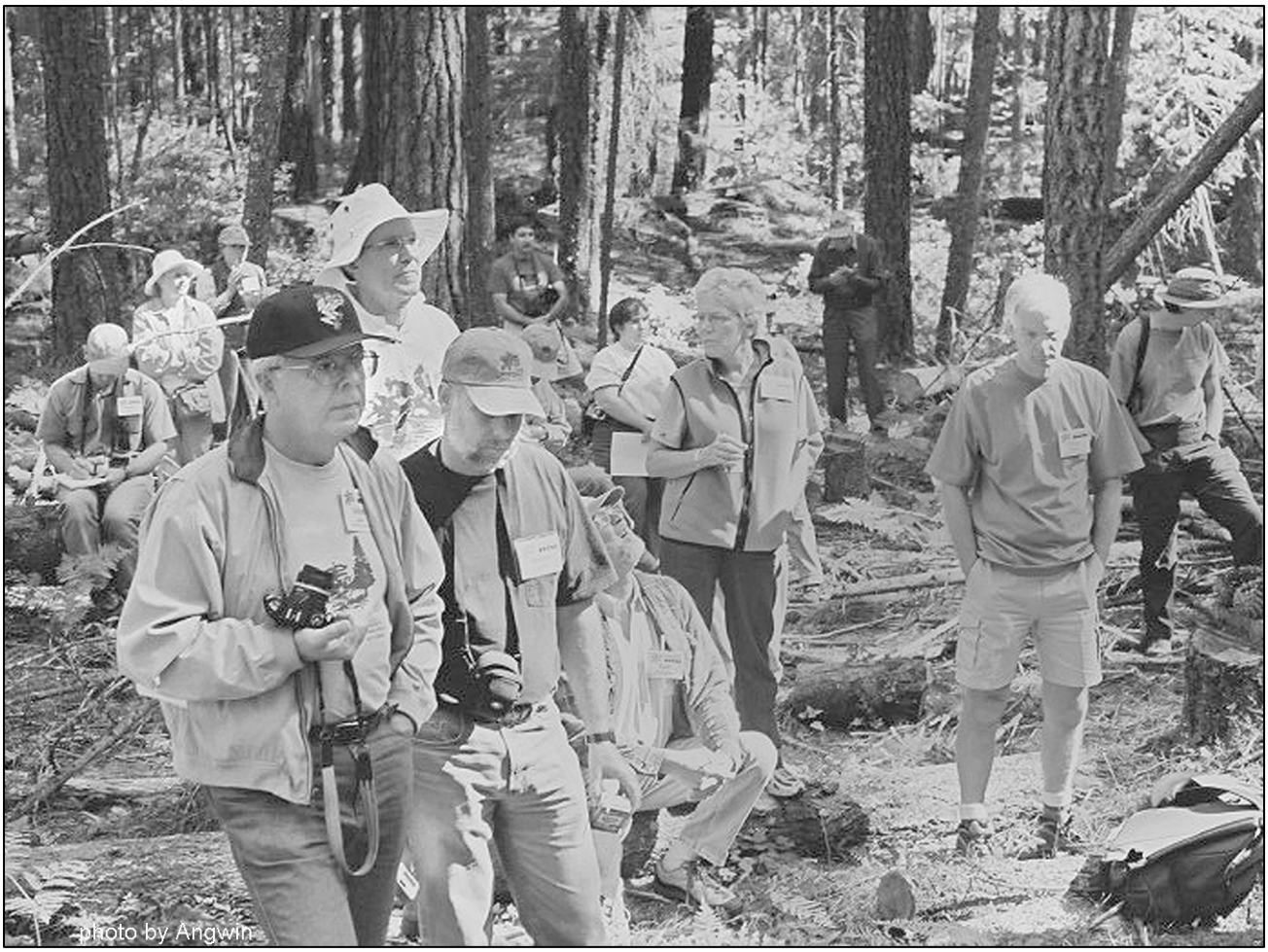
But there are other mistletoes on fir as well.

Another fir of the Jefferson region is Pacific silver fir, *Abies amabilis*. This coastal species resembles the montane white fir with resinous buds and two-ranked leaves, but without rows of stomata on the adaxial leaf surface (looks green not glaucous). The dwarf mistletoes on Pacific silver fir are *A. tsugense* (commonly), *A. douglasii* (occasionally), and *A. abietinum* f. sp. *concoloris* (rarely). The presence of a heavily infected principle host is the best clue for identification, but there are morphological differences. *Arceuthobium douglasii* has the smallest shoots (2 cm); *A. abietinum* is yellowish; *A. tsugense* is greenish or reddish.



So you know the hemlocks, how about their mistletoes?

Both western hemlock with leaves appearing two-ranked and mountain hemlock with leaves spread around the twig occur in the region and serve as hosts for the hemlock dwarf mistletoe, *Arceuthobium tsugense*. Differences in host preference, phenology, and morphology are sufficient that Hawksworth and Nickrent recognize a subspecies primarily associated with mountain hemlock. Besides primarily infecting mountain hemlock and rarely infecting western hemlock, *A. tsugense* subsp. *mertensianae* is on average shorter—5 cm (range 3 to 9 cm) than *A. tsugense* subsp. *tsugense* which primarily infects western hemlock and only rarely mountain hemlock with shoots on average 7 cm tall (range 3 to 13 cm). *Arceuthobium tsugense* is the only mistletoe on western hemlock, but mountain hemlock is also infected by *A. laricis* (principal host) and *A. cyanocarpum* (secondary host). Although the larch dwarf mistletoe (*A. laricis*) does not occur in the Jefferson region, the limber pine dwarf mistletoe (*A. cyanocarpum*) is common on the north slope of Mt. Shasta and occurs at few other locations in the region.

Long-term Monitoring of Tree Damage Caused by Porcupine Feeding in the Khutzeymateen Inlet

Stefan Zeglen and Alex J. Woods

Abstract—To determine the effects of porcupine (*Erithizon dorsatum*) feeding on four conifer species, a series of 69 permanent sample plots was monitored over 15 years. Annual visits revealed that western hemlock (*Tsuga heterophylla*) was the species most preferred by porcupine based on the number of trees attacked and the repeated nature of the feeding. Eighty-six of the 100 sample trees that died during the monitoring period were dominant or co-dominant western hemlock. Most tree damage occurred in the middle third of the stem, with trees between 12.5 and 32.4 cm in diameter preferred over smaller or larger diameter trees. While stand composition remained unchanged over 15 years, western hemlock trailed other conifers in diameter and height growth and gross volume increment. In addition, a major portion of the mean stand volume of western hemlock is composed of trees that have been damaged by porcupine feeding, the wounds of which may be prone to colonization by decay fungi. Comparison with current inventory estimates suggest that these observed losses due to porcupine feeding are accounted for in the timber supply analysis.

Introduction

A number of mammals feed on various parts of coniferous and deciduous trees. In North America, the most damaging of these mammals is the American porcupine (*Erithizon dorsatum*). The porcupine is primarily a herbivore whose winter diet consists almost exclusively of the cambium, phloem, and foliage of woody shrubs and trees (Dodge 1982). The severity of feeding damage to trees ranges from small (<50 cm^2) patches or "tasting wounds" (Eglitis and Hennon 1997) to complete bark stripping or girdling of pole-size trees. Such damage leads to reduced tree growth, structural defects that can result in stem breakage, lowered wood quality, and infection courts for decay fungi. Reduced growth and yield from damaged trees is also of significant concern for timber supply and forest management.

In the early 1980s, extensive mortality in second growth western hemlock (*Tsuga heterophylla*)–Sitka spruce (*Picea sitchensis*) forests was discovered in the North Coast and Kalum Forest Districts of northwestern British Columbia (BC). Mortality was scattered throughout susceptible stand types, usually centered on a denning site. Tree wounds corresponded to those typical of porcupine feeding with broad horizontal and diagonal incisor marks prominent in the exposed sapwood (Lawrence and others 1961). This damage caused great concern as these second growth stands, many logged during the previous 40 years, provided a potentially vast food source with few natural predators.

In order to quantify the extent of this problem, Ministry staff conducted a damage survey in the Khutzeymateen Inlet in the spring of 1985. The results of this survey were published in Sullivan and others (1986). In order to quantify the continuing effects of porcupine feeding on potential crop trees, the following year the sample plots were permanently established and revisited annually. The results of 15 years of observations form the basis of this presentation.

Objectives

There were two main objectives to this study:

- Report the annual rate and cumulative amount of feeding damage that occurred over a 15-year period; and
- Illustrate how this damage is affecting stand structure, species composition and timber volume in hemlock-dominated second growth stands of north-coastal BC.

Methods

The study area is located in the North Coast Forest District along the Khutzeymateen Inlet, about 45 km northwest of Prince Rupert, BC. The area sits within the coastal western hemlock very wet maritime (CWHvm) biogeoclimatic zone and is typical of low elevation, second growth forests within the region. Stands are dominated by western hemlock with lesser amounts of Sitka spruce, amabilis fir (*Abies amabilis*), western redcedar (*Thuja plicata*) and red alder (*Alnus rubra*). Much of the study area was logged between 1947 and 1954.

In 1986, 69 variable radius plots were installed along both sides of the inlet capturing all trees >7.5 cm diameter at

In: Geils, B. W. comp. 2004. Proceedings of the 51st Western International Forest Disease Work Conference; 2003 August 18–22; Grants Pass, OR. Flagstaff, AZ: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station.

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breast height (dbh). All sample trees were tagged and dbh and height measured. Crown class and form defects (forks, crooks, dead or broken tops) were also noted along with all current and previously inflicted damage caused by porcupine feeding.

Every spring from 1986 to 1995, the plots were visited to record new winter feeding damage. The number of new wounds and their relative position on the stem (bottom, middle or top third) were recorded. Any tree mortality or defect was also noted. In-growth was ignored. From 1996 to 1999, no visits were made. In 2000, a final visit was made to record tree damage as well as take height and dbh measurements.

Analysis

Due to the consistency of forest types within the study area, the 69 plots were pooled to facilitate data analysis and improve statistical testing. Chi-square tests were used to detect significant differences in porcupine feeding preference by tree species, diameter class, stem location and tree mortality. One-way ANOVA were used to test for differences in trees/ha and volume/ha of both attacked and non-attacked trees by species. In all cases, the level of significance was $p \leq 0.05$.

Gross tree volumes were determined by using the BC Ministry of Forests whole stem volume equations. Comparisons were made between these calculations and those from the 1998 provincial re-inventory for the species in question. These comparisons were net of decay, waste and breakage estimates.

Results

A total of 589 conifers formed the sample for this study—398 western hemlock, 90 Sitka spruce, 81 amabilis fir, and 20 western redcedar.

Incidence of feeding

By far, western hemlock was the most attractive feeding target for porcupine followed by Sitka spruce and amabilis fir. No western redcedar was attacked during this study. Three-quarters of tree feeding damage occurred prior to plot establishment. From 1986 to 2000, 74 previously un-damaged trees were attacked, bringing the total to 50.4 percent of all sample trees. Annual attack rates varied from 2.8 to 8.8 percent for western hemlock with the "recruitment" rate of previously undamaged trees being about 1 percent per year.

Intensity of feeding

If one considers the number of individual wounds on a tree to be an indication of feeding intensity, then western hemlock suffered the greatest intensity of damage. The 260 damaged hemlock accumulated 1697 feeding wounds, for an average of 7.6 wounds per tree. Over 60 percent of these wounds occurred prior to 1986. Comparable numbers for spruce and fir were 1.0 and 0.3 wounds per tree, respectively, with most damage occurring after 1986.

Porcupines also appeared to prefer feeding on certain parts of a tree. Dividing the stem into thirds revealed that, for western hemlock, the majority of feeding wounds (50 percent) occurred in the middle third, with the bottom (36 percent) following, and the top (14 percent) the least popular location. The pattern is similar for spruce and fir. Examining the distribution of wounds across the diameter range of hemlock shows that over 86 percent of feeding wounds happened to trees between 12.5 and 32.4 cm dbh (figure 1).

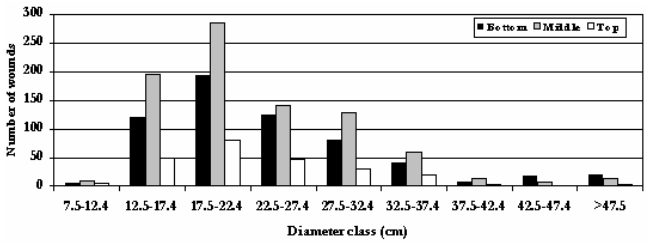

Figure 1—Distribution of porcupine feeding wounds across all diameter classes of western hemlock sample trees.

Mortality

Over the 15-year study period, 100 of the sample conifers died; 86 of these were western hemlocks. Twice as many porcupine-attacked hemlocks died as compared to non-attacked hemlocks. Of note is that of the dead hemlock that had been wounded by porcupine, over 82 percent were dominant or co-dominant trees. Of the dead hemlock not attacked by porcupine, 93 percent were smaller diameter intermediate or suppressed trees.

Impact

This loss of trees did not appreciably change the species composition of the sample over 15 years, as losses were roughly proportionate with the other conifer species present. However, appreciable effects on the diameter and height growth of western hemlock were noted, when compared to the other conifers present (table 1). Over 15 years, hemlock grew less rapidly in both height (39 percent) and diameter (25 percent) than all other conifer species. Sitka spruce dominated in both categories.

The impact of reduced growth becomes apparent when gross tree volumes are calculated for each species on a per hectare basis (figure 2). Western hemlock volume

increased 54 percent compared to 211 percent, 169 percent and 100 percent for spruce, fir and redcedar, respectively.

Table 1—Increase in diameter and height growth of sample conifers during the 15-year study period.

Species	Increased dbh, percent	Increased height, percent
Western hemlock	25	39
Sitka spruce	53	63
Amabilis fir	43	44
Western redcedar	30	61

The impact on mean volume (subtracting for losses from decay, waste and breakage due to harvesting), and factoring in the potential for further volume loss due to increased activity by decay fungi in damaged trees, is even more dramatic (figure 3).

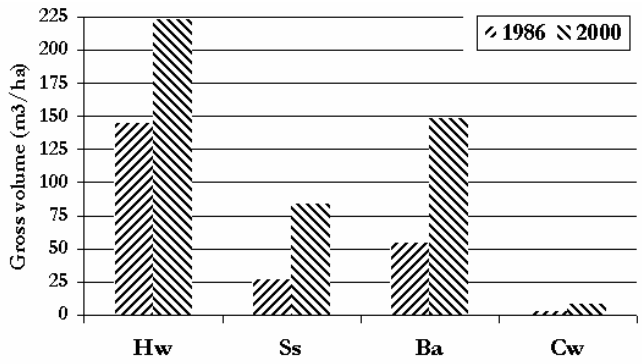

Figure 2—Increase in gross volume per hectare for all species over 15-year study period for western hemlock (Hw), Sitka spruce (Ss), amabilis fir (Ba), and western redcedar (Cw).

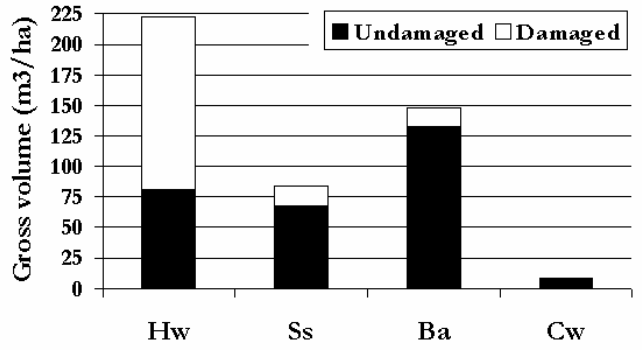

Figure 3—Adjusted mean volume after accounting for harvesting netdowns and potential loss of trees damaged by porcupine feeding on western hemlock (Hw), Sitka spruce (Ss), amabilis fir (Ba), and western redcedar (Cw).

Discussion

It is clear that porcupines prefer western hemlock as opposed to the other tree species in this study. This is supported by a recent study on porcupine feeding (Zimmerling and Croft 2001) conducted in the adjacent Kalum Forest District and by observations of feeding behaviour in other forest types (Roze 1984, Harder 1980). Porcupine tend to feed on any tree provided it is the most numerous in a stand; and will rarely vary to more than two species, even when more are available.

This study of porcupine feeding is unique in its having followed the actual incidence of feeding for a long period. This extended observation has allowed us to avoid the pitfalls inherent in short-term studies namely, as Curtis (1941) warned, over-estimating the severity of the problem. The annual rate of "recruitment" by porcupine of undamaged trees was 1 percent in our study compared to the 5.1 percent rate originally forecast by Sullivan and others (1986) from one year of data. This higher rate led to the prediction that porcupine feeding could render the western hemlock portion of the timber supply un-merchantable due to increased morbidity from decay, a prediction that now seems overly dire.

There are several explanations for this rapid decline in the rate of porcupine feeding damage. These include greater difficulty for porcupine in scaling larger trees, thicker bark making it more work to access the cambium, and less light on the forest floor reducing the herbaceous vegetation that porcupine feed on in the summer. Another factor is that, like most forest epidemics, most damage was done prior to being noticed by humans—the so-called "upslope" of the damage curve. By the time Sullivan and others (1986) and this study were initiated, the stand character had already altered to be less suitable for porcupine and the attack rate was on the "downslope". This is supported by our findings of reduced attack on larger trees and a reduction in feeding on hemlock after 1986, as compared to pre-1986, combined with an increase in feeding on the less numerous spruce and fir after 1986.

This study is also unique in that we have addressed porcupine impact in terms of volume/ha. Our results indicate that porcupine damage has reduced the current stand volume/ha by about 37 percent if all damaged trees are assumed to not be commercially viable at rotation. This will depend greatly on the amount of decay that occurs as a result of the wounds created by porcupine. Previous studies (Hennon and DeMars 1997, Wright and Isaac 1956, Woods, unpublished) have shown that hemlock and spruce are both prone to decay in these types of coastal stands. A future study intends to document the development of decay in wounded trees.

In terms of timber volume, it appears that provincial inventory projections are currently accounting for losses to the standing gross volume/ha. Our calculation of mean volume of undamaged trees of about 287 m^3/ha is very

close to the projection of 291 m^3/ha made for the last timber supply review.

The situation that this study examined was driven by the creation of large areas of even-aged, second growth stands in the North Coast area that led to a sudden increase in both the winter and summer forage available to the porcupine. Responding to this, porcupine numbers grew leading to localized feeding that created enough mortality to become readily noticeable. This natural reaction to a sudden increase in food supply has resulted in a significant, but not unaccounted for, loss in terms of standing tree volume and a greater potential loss via the action of tree decays through wounds created on a substantial number of remaining trees. It is likely that these losses will return to the more typical, less detectable, level in second growth stands if the pattern of harvesting that encouraged them is not repeated.

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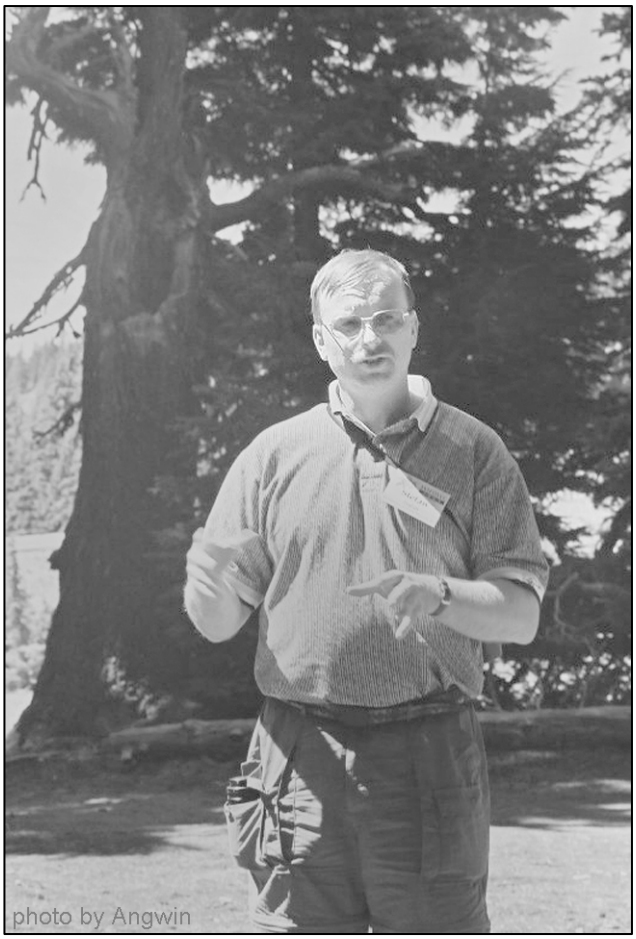

A little more about the blue-fruited dwarf mistletoe.

Arceuthobium cyanocarpum is a recognizable mistletoe on high-elevation white pines (whitebark and western white), foxtail pine (the only mistletoe on this species), and even mountain hemlock. Shoots are short (3 cm), yellow-green, and densely clustered around the infected stem. Branch flagging and small, compact brooms are common; and host mortality is often very high.

Fire and Dwarf Mistletoe

Robert Tinnin

Abstract–Fire and dwarf mistletoes (*Arceuthobium* spp.) have long been viewed as interactive. Dwarf mistletoes can influence fuel loads on the ground and in tree crowns, and they shape crown architecture in ways that can increase the intensity and probability of crown fires. However, the extent and importance of these effects on fire occurrence and performance remain to be clarified.

Introduction

Fire has been an important functional element of forests in the western United States for thousands of years (Agee 1994). Both natural and human caused ignition has occurred; but natural causes seem to have been more important, generally (Agee 1991). Until recently, fires, once started, burned in response to naturally occurring barriers and stimuli with little or no human intervention. Natural patterns of fire in the West are complex with return intervals ranging from decades to several centuries (Agee 1991). Whatever the pattern, it was commonly fire, in concert with other ecological processes, that forged the forest structure found by Europeans during the settlement period. Superimposed over this dynamic forest system was a patchy mosaic of dwarf mistletoe (*Arceuthobium* spp.). Many species of this common parasite ranged throughout pre-settlement forests, and fire is thought to have been the primary factor that shaped the mosaic (Alexander and Hawksworth 1975).

Plant communities that endure chronic disturbance of low intensity (of which fire can be an example) tend to become tolerant of that disturbance (Sousa 1984). The resulting structure can be maintained only by continued disturbance of approximately the same intensity and frequency with some species depending on the disturbance. Some of these species may produce conditions that encourage the disturbance, thereby helping ensure their continued existence (Mutch 1970). Dwarf mistletoes are reported to influence fire in several ways (Alexander and Hawksworth 1975) and, given a seral host, possibly even affect their own continued existence within a community by encouraging fires that would tend to perpetuate their host (Tinnin and others 1982, Hawksworth and Johnson 1989).

When Europeans established themselves in the West, fire frequencies changed (Everett and others 2000). As suppression techniques improved throughout the twentieth century, the frequency of fires was greatly reduced. Fire control is one of several factors that have contributed to an increased abundance of dwarf mistletoe. Now, with decades of freedom from fire, the accumulated fuels in many areas and warming climates are fostering conditions ideal for massive stand-replacing burns (Gehrke 2003). In this context and given the potential connection between fire and dwarf mistletoe, do dwarf mistletoes increase the risk, intensity and consequences of fire in today's forests?

What Is Seen

Prior to the arrival of Europeans, many western forests routinely experienced surface fires that removed accumulated fuels while killing many seedlings and small saplings of tree species and less resistant mature trees (Agee 1994). Given enough intensity, even more resistant species were killed. Tree death as a direct result of fire can result from cambial destruction, severe scorching of the crown or ignition of the crown. Dwarf mistletoe may increase the probability of any or all of these destructive conditions under certain conditions. Most commonly, dwarf mistletoes modify host crown structure and locally increase ground based fuels.

Crown structure changes in response to witches' broom formation and as a result of increased longevity of infected limbs. Witches' brooms take many forms, but some achieve great size and mass. Much of the broom volume is composed of fine fuels as a consequence of prolific twig production. The most prominent example of massive broom formation in the West is seen in Douglas-fir (*Pseudotsuga menziesii*). In addition to biomass, some types of brooms produce long twigs that can extend well below the limb that supports them which effectively lowers the crown base. However, the more common cause of lower crown base is increased longevity of infected branches. The longer branches remain on a tree near the ground, the longer the window of time that surface fires have to readily access the tree crown. Given the relationship between fire and crown height, it is remarkable that the effect of infection on crown height has not been more thoroughly studied. Recently, Godfree and others (2002a) report that infection in the lower crown of lodgepole pine (*Pinus contorta*) significantly lowers the crown base, but dwarf mistletoe functions as only one of several independent variables. The most important variable observed was bole diameter, which is itself affected by dwarf mistletoe. This multi-dimensional effect of dwarf mistletoe on crown height serves to illustrate the complexity of the relationship.

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Local increases of fuel on the ground occur as witches' brooms break out of host trees. Breakage is common in both Douglas-fir and western larch (*Larix occidentalis*), less so in other host species. In some cases, increased populations of needles and twigs on infected trees may also contribute to increased fuels, although Godfree (2000) found no relationship between dwarf mistletoe and needle cover on the forest floor when studying lodgepole pine. However, there was an increase in large woody debris (Godfree and others 2002b), presumably as a function of dwarf mistletoe increasing tree mortality. Given more woody debris, fire duration is increased which generally increases damage to and often death of standing trees. Again, curiously, few data are available to clarify the relationship between dwarf mistletoe and fuel accumulation. For example, Conklin and Armstrong (2001) found that ponderosa pine (*Pinus ponderosa*) generally survives crown scorching quite well but mortality increases dramatically in heavily scorched trees. However, they also observed higher than normal mortality in heavily infected trees, an outcome for which there is no clear explanation.

If a crown should ignite, dwarf mistletoe may influence an increase in the intensity of the fire. The increased number of twigs in some brooms has already been mentioned. However, even when there is not a great increase in twig or needle number, brooms can form a platform that intercepts and holds dead needles and twigs that would otherwise fall to the ground (Alexander and Hawksworth 1975). There may also be an increased abundance of resins in the branch wood of brooms or a difference in wood properties that could affect fire intensity. Taking these factors together, one might anticipate that brooms would burn hotter and longer than healthy branches. I know of no data to support these ideas but testing will begin soon.

What It May Mean

Given that conditions are ripe for extensive and intensive fires in western forests, can one say that dwarf mistletoe contributes to the risk, intensity and impact of a fire? The answer seems to be a qualified yes. Extant studies show that all else being equal, dwarf mistletoes increase fuel loads. But all is not equal and many other factors seem to be more important in determining the fuel loads that actually develop. Crown architecture affects fire performance and without a doubt dwarf mistletoe affects crown architecture. However, do the induced changes significantly increase the effects of fire? In some cases, yes, but quite possibly not in all cases. These questions remain to be studied more thoroughly. Wherever there are stand replacing fires, dwarf mistletoes will be largely eliminated from the landscape. Even so, patches of residuals will remain and these will provide the inocula for future infections. Dwarf mistletoes have and will continue to play a role in shaping forest structure and function, and in limited ways contribute to the consequence of fire. There is no doubt that dwarf mistletoes are but one factor in the complex equation that defines fire potential; the true importance of their influence awaits elucidation.

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Armillaria Root Disease in Campgrounds of Southern Colorado

Jim Worrall, Kelly Sullivan, Tom Harrington, and Joe Steimel

Summary of Results

In hazard tree inspections of many campgrounds in southwestern Colorado, Armillaria root disease was very common. The disease was often associated with failure of living trees. Property damage and near misses prompted increased focus on the problem. In six campgrounds that we studied, we found 10.5 percent infection of live trees at or near the root collar; adjacent forest trees had 12.7 percent infection. Host species varied in infection from 7.0 percent (subalpine fir, significantly lower than other species) to 15.7 percent (Douglas-fir). Infected trees were slightly but significantly greater in d.b.h. than uninfected trees, and dominant trees had the highest incidence of infection. All trees (infected and uninfected) were more clustered than were infected trees; the pathogen was relatively randomly distributed. In the spruce-fir campground, all tested isolates belonged to a single genet; in the two mixed-conifer campgrounds there were several genets (for example, figure 1), but they were largely contiguous. The pathogen was identified as *Armillaria ostoyae*.

Management Implications

Although there is no effective and practical measure to reduce future infections in this setting, our data facilitate and support two management approaches—disease avoidance and reducing current incidence. Based on the size of the genets, they are at least several hundred years old and the stands were infested long before campground establishment. Campground establishment has apparently not exacerbated the disease, because incidence is generally the same or lower than in adjacent forest. Site selection is therefore a critical factor in determining disease incidence. The data do not support targeting trees for removal based on proximity to the pathogen. Instead, careful attention to symptoms can focus belowground inspection for mycelium on trees most likely to be infected, so that most infected trees can be detected efficiently and removed.

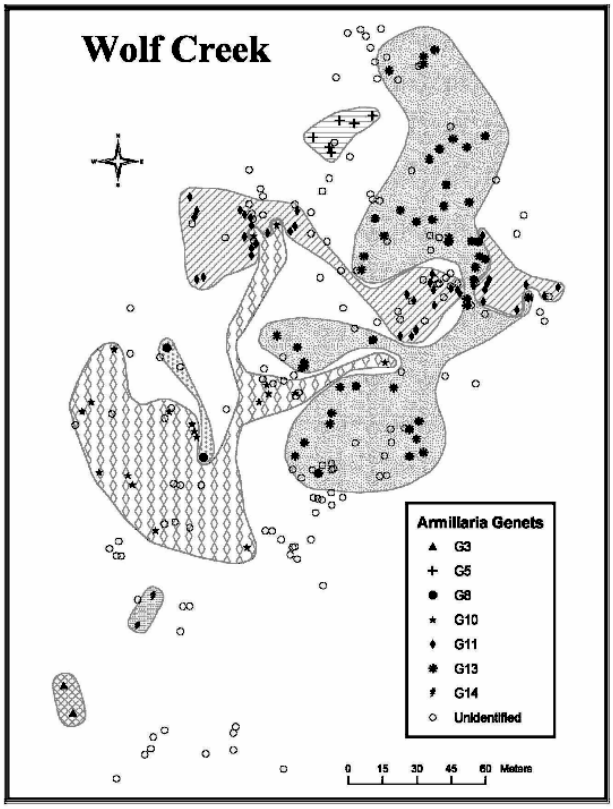

Figure 1—Genets of *Armillaria ostoyae* identified at Wolf Creek Campground, San Juan National Forest, in southwest Colorado. Additional occurrences of the pathogen that were not identified to genet are also mapped.

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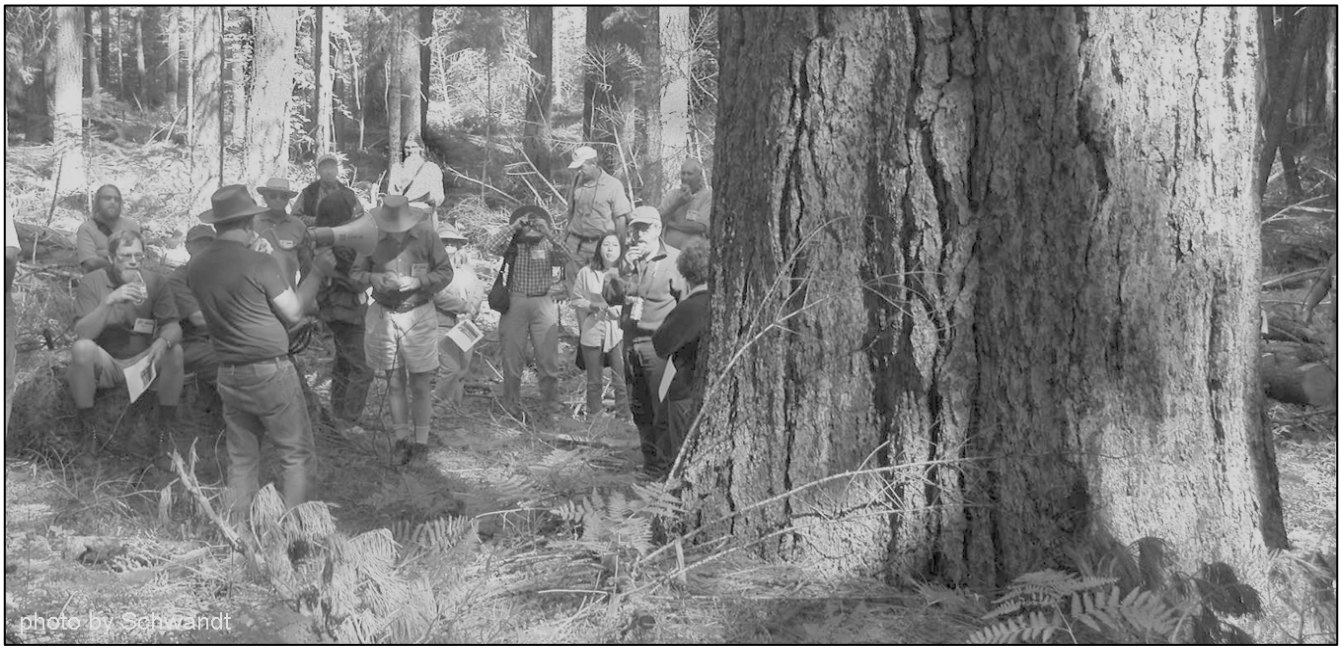
Western white pine dwarf mistletoe, a local endemic.

Arceuthobium monticola is restricted to the Klamath and Siskiyou Mountains of extreme southwestern Oregon and northwestern California. This mistletoe was previously submerged under the similar, more widely distributed, but not sympatric mistletoe, *A. californicum*. *Arceuthobium monticola* differs from *A. californicum* in its darker shoot color, later seed dispersal (October–November versus September–October), and host preference for western white pine over sugar pine. *Arceuthobium monticola* differs from *A. tsugense* subsp. *mertensiana* (which occasionally infects western white pine when in association with mountain hemlock) by its dark brown rather than greenish and reddish coloration and later seed dispersal (August–September for mountain hemlock dwarf mistletoe).



Sugar pine dwarf mistletoe, big brooms for big trees.

The only principle host for *Arceuthobium californicum* is sugar pine but western white pine is a secondary host at some locations where these two pine occur together. The mistletoe shoots are greenish to bright yellow but becoming brown at the base of older shoots; brooms are dense and attain large size; infection can be common and damaging. This is a California endemic found from Mount Shasta south. Sugar pines are infected by the white pine dwarf mistletoe, and rarely by the white fir dwarf mistletoe and the western dwarf mistletoe.

Poster Session

Kelly Sullivan, Moderator

Program

***Phytophthora ramorum* wound inoculations of conifer saplings important to British forestry.** Sandra Denman (presenter), Alice Holt Lodge, Surrey. UK; Susan A. Kirk, Joan Rose, Joan F. Webber and Clive M. Brasier.

Systematic study of *Nattrassia mangiferae*, the cause of madrone canker. Marianne Elliott (presenter) and Robert L. Edmonds, University of Washington, Seattle, WA.

Could white pine blister rust spread by atmospheric transport from California to New Mexico? Katrina L. Frank, Laurence S. Kalkstein, University of Delaware, Newark, DE; Brian W. Geils (presenter), Rocky Mountain Research Station, Flagstaff, AZ; Harold Thistle, Forest Health Protection, Morgantown, WV; and Eugene P. Van Arsdel, Rocky Mountain Research Station, volunteer.

Genetic variation of *Armillaria ostoyae* within the western United States. J.W. Hanna (presenter), N.B. Klopfenstein, M.-S. Kim, G.I. McDonald, Rocky Mountain Research Station, Moscow ID; and J.A. Moore, University of Idaho, Moscow, ID.

First report of A1 mating type of *Phytophthora ramorum* in North America. Everett M. Hanson, Paul W. Reeser, Wendy Sutton (presenter), Loretta M. Winton, Oregon State University, Corvallis, OR; and Nancy K. Osterbauer, Oregon Department of Agriculture.

Is the alternate host for white pine blister rust present in Colorado? Holly S.J. Kearns, (presenter), William R. Jacobi, Colorado State University, Fort Collins, CO; Kelly Sullivan, Rocky Mountain Region, Lakewood, CO; and Brian W. Geils, Rocky Mountain Research Station, Flagstaff, CO.

Influence of inoculum source and density on white pine blister rust infection of whitebark pine: early results. A. Kegley (presenter), R.A. Sniezko, R. Danchok, J. Danielson, and S. Long, Dorena Genetic Resource Center, Cottage Grove, OR.

Characterization of North American *Armillaria* species: phylogenetic relationships from ribosomal DNA sequences. M.-S. Kim (presenter), J.W. Hanna, N.B. Klopfenstein, P.J. Zambino, and G.I. McDonald, Rocky Mountain Research Station, Moscow, ID.

Tree pathogen survival in wood chip mulch. Ronda D. Koski and William R. Jacobi (presenters), Colorado State University, Fort Collins, CO.

Wood-Decomposing fungi on fertilized sites in the northwestern U.S.A. Raini C. Rippy (presenter) , Ned B. Klopfenstein, Mee-Sook Kim, Paul J. Zambino, Deborah S. Page-Dumroese, James A. Moore, and Paul A. McDaniel, Rocky Mountain Research Station, Moscow, ID.

Simply inherited resistance to *Phytophthora lateralis* in Port-Orford-Cedar: Greenhouse testing. R.A. Sniezko (presenter), Dorena Genetic Resource Center, Cottage Grove OR; E.M. Hansen, Oregon State University; and S.E. Kolpak (presenter), Dorena Genetic Resource Center, Cottage Grove OR.

Exotic pathogens, resistant seed, and restoration of forest tree species in western North America. Richard A. Sniezko, Dorena Genetic Resource Center, Cottage Grove OR; Diana F. Tomback, University of Colorado, Denver, CO; Regina M. Rochefort, North Cascasde National Park, Sedro-Wooley, WA; Ellen Goheen, SW OR Forest Insect and Disease Service Center, Central Point OR; Rich Hunt, Pacific Forestry Centre, Victoria, BC; Jerry S. Beatty, Forest Health Protection, Arlington VA; Michael Murray, Crater Lake National Park, OR; and Frank Betlejewski, SW OR Forest Insect and Disease Service Center, Central Point OR.

Western dwarf mistletoe, a serious pathogen of ponderosa and Jeffrey pines.

From western Idaho to Washington and south to Baja, the most common and damaging mistletoe of the yellow pines, ponderosa and Jeffrey, is *Arceuthobium campylopodum*. This stout, olive-green to yellow mistletoe is a member of a close complex of taxa including *A. occidentale* and *A. siskiyouense* which also occur in the Jefferson region. These mistletoes and the lodgepole pine dwarf mistletoe, *A. americanum*, may infect ponderosa pine or Jeffery pine.

Knobcone pine dwarf mistletoe, another local endemic.

Arceuthobium siskiyouense is restricted to the Klamath and Siskiyou Mountains and principally occurs on knobcone pine and rarely on ponderosa, Jeffrey, and lodgepole pine when these are in association with infected knobcone pine. Compared to western dwarf mistletoe, the knobcone pine dwarf mistletoe is shorter (6 to 10 cm versus 10–14 cm), finer (shoot diameter 2–2.5 mm versus 3–6 mm), and most distinctively—does not form brooms (common and well developed in hosts infected by western dwarf mistletoe). The seed cones of knobcone pine are serotinous, persistent, asymmetrical, and in whorls.

Phytophthora ramorum Wound Inoculations of Conifer Saplings Important to British Forestry

Sandra Denman, Susan A. Kirk, Joan Rose, Joan F. Webber and Clive M. Brasier

Abstract—Heavy mortality of oak trees in California (Sudden Oak Death) due to *Phytophthora ramorum* together with its presence in Europe has led to major concern about the damage this pathogen could cause to British forests and vegetation. As part of a pest risk analysis, stems of ten conifer species important to British forestry were wound inoculated. Eight of the ten conifers were susceptible to *P. ramorum* isolates from both Europe and the US. Saplings of *Picea sitchensis* (Sitka spruce) and *Tsuga heterophylla* (western hemlock) died. Large lesions occurred on the stems of *Abies procera* (noble fir) and *Pseudotsuga menziesii* (Douglas-fir). Necrosis developed more slowly in *Picea abies* (Norway spruce) and to a lesser extent in *Chamaecyparis lawsoniana* (Port-Orford-cedar), *Pinus contorta* (lodgepole pine) and *Taxus baccata* (yew). *Pinus nigra* (Corsican pine) and *P. sylvestris* (Scots pine) were unaffected. Implications for UK forests are discussed.

Introduction

Phytophthora ramorum sp. nov. (Werres and others 2001) is responsible for the current heavy mortality of *Quercus* and *Lithocarpus* species in coastal forests of California and southern Oregon, known as "sudden oak death" (Rizzo and others 2002) and for shoot die-back, cankers and foliar lesions on various understorey shrubs and saplings in the same areas. Simultaneously, *P. ramorum* is also spreading in the ornamental nursery trade in western Europe and the UK. In Europe, it causes mostly leaf necrosis and shoot blight, mainly on Rhododendron but also on Camellia, Kalmia, Leucothoe, and Pieris; plus basal stem cankers and wilting of Viburnums (Lane and others 2003, Orlikowski and Szkuta 2002). The pathogen is largely confined to nurseries and has not yet been found on tree hosts in Europe except in one case.

P. ramorum appears to be a recent introduction on both continents. Cultural studies indicate that European populations of *P. ramorum* are predominantly A1-mating type, and the US isolates are predominantly A2. The European population is also adaptively different from the US population (Brasier 2003). Since it appears that *P. ramorum* could have a wide potential host range intensive screening is underway to assess the risk the pathogen may pose to UK and European forests and vegetation. Tests are being conducted on a wide range of broadleaf, conifer and shrub hosts. These include—1) tests on foliage both for its susceptibility and as an inoculum source (Lane and others 2003); 2) tests on excised logs of mature trees to assess stem and bark susceptibility (Brasier and others 2002); and 3) tests on potted saplings of trees and shrubs. We describe here the first results of wound inoculation of saplings of ten conifer species important to British forestry (table 1), many of them native to the USA and Canada.

Table 1—Number of dead and live branches per four inoculated branches

Host	Dead	Live
Abies procera, noble fir	4	0
Chamaecyparis lawsoniana, Lawson cypress	0	4
Picea abies, Norway spruce	3	1
Picea sitchensis, Sitka spruce	4	0
Pinus contorta, lodgepole pine	1	3
Pinus nigra var. *maritma*, Corsican pine	0	4
Pinus sylvestris, Scots pine	0	4
Pseudotsuga menziesii, Douglas-fir	4	0
Rhododendron	2	2
Taxus baccata, yew tree	0	4
Tsuga heterophylla, western hemlock	4	0

Materials and Methods

An A1-mating type of *P. ramorum* from the UK, representing the European population, and an A2 isolate from California, representing the US population, was used. Isolates were inoculated separately into shallow flap incisions (5 mm long) in the bark of 2- to 3-year-old saplings about 10 cm above soil level and in two side branches. A drop of sterile water was added. The wound was then wrapped with wet filter paper and secured with parafilm and shrink wrap. Therefore, for each host there were two plants and six inoculation points. A single control plant of each host species was inoculated with agar plugs only. Plants were incubated in a quarantine chamber at 20° C with 12 hours light per day, and watered as necessary. At four weeks, resulting necrotic bark lesions were measured; and the area of lesion calculated. The number of wilting or dead side branches was also recorded. Isolations were made onto selective medium from bark tissue at 1.5 cm and 3 cm above and below the visible lesion and from the centre and margin of the necrotic area.

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Results

Stem lesion area

The lesion area of the inner bark of inoculated plants was significantly different from the control plants ($P < 0.001$). No statistically significant difference was found between the European and the US *P. ramorum* isolate with respect to lesion area.

There was a significant host by treatment interaction ($P = 0.016$). In general, the lesion areas of inoculated plants were much larger than those of the controls; but in Corsican pine (*Pinus nigra*), control lesions were larger than those of the inoculated saplings (figure 1).

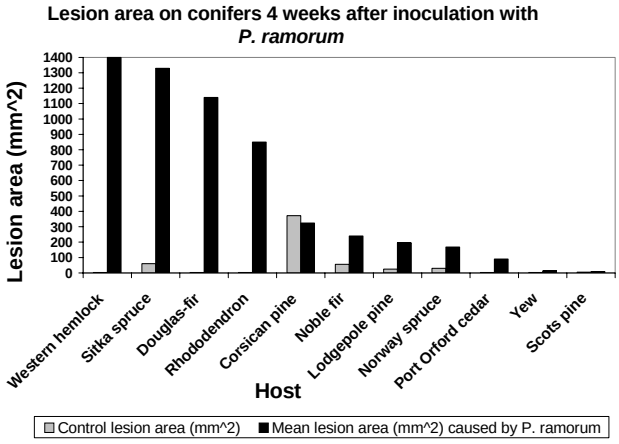

Figure 1—Lesion area on conifers 4 weeks after inoculation with *P. ramorum*

At 4 weeks both inoculated western hemlock (*Tsuga heterophylla*) trees were dead. One of the two inoculated Sitka spruce (*Picea sitchensis*) trees was wilted (figure 2) with a girdled stem. The other Sitka spruce also had a large lesion.

Large near girdling lesions also developed on both Douglas-fir (*Pseudotsuga menziesii*) and on rhododendron which represented the positive control (figure 1).

Smaller lesions developed on the Norway spruce (*Picea abies*) and noble fir (*Abies procera*) stems, but severe wilt, leaf desiccation and death of side branches were evident.

Only small lesions developed in the inoculated yew trees (*Taxus baccata*) (figure 1), but wilting was evident in the branches.

Moderate lesions developed both in the control and inoculated plants of Corsican pine. That is, *P. ramorum* was probably non-pathogenic on this species, but lesions developed in response to wounding.

Figure 2— Sitka spruce (*Picea sitchensis*) 4 weeks after inoculation with *P. ramorum*

Inoculated branch vitality

All four inoculated branches were dead in Douglas-fir, noble fir, Sitka spruce and western hemlock saplings (table 1). Branches of Norway spruce and rhododendron were also severely affected. None of the Corsican and Scots pine (*Pinus sylvestris*), Port-Orford-cedar (*Chamaecyparis lawsoniana*), yew tree branches died after inoculation, although significant wilting occurred with the yew trees.

Back isolation

P. ramorum was successfully re-isolated from the wound inoculation points on the *P. ramorum*-inoculated material. It was recovered from the lesion centres of all host species and from the margin between healthy and necrotic tissue in all hosts except the Corsican and Scots pines. It was also isolated at 1.5 to 3 cm above and below the visible lesion edge in both rhododendron and Douglas-fir. None of the control inoculations were positive.

Discussion

On wound inoculation, saplings of eight of the ten conifers tested were susceptible to *P. ramorum* isolates from both Europe and the USA. Douglas-fir, Sitka spruce and western hemlock were the most susceptible. This is in agreement with results of previous tests on the susceptibility of these species using excised log tests (Brasier and others 2002). In the UK, about 80 percent of timber utilised is softwood (that is, of conifer origin); and conifers represent 63 percent of the total woodland area. Sitka spruce accounts for 30 percent of that area and Douglas-fir for 2 percent. Considerable economic, social and environmental consequences might be felt if *Phytophthora ramorum* were to seriously affect these hosts in the field in the UK.

To further assess the level of the risk to UK conifers, information is required on whether the pathogen is able to enter unwounded host material; on which plant parts (stems, shoots, leaves) are susceptible to P. ramorum; and on whether inoculum propagules such as sporangia can be produced on these hosts or on other susceptible hosts in the same environment. Climatic influences and inoculum thresholds required for disease development also need to be established. These studies are currently in progress at several institutes in the UK.

In North America, young shoots of Douglas-fir are known to be susceptible to *P. ramorum* in the field (Davidson and others 2002). The present results, together with those from the excised log tests, indicate that if conditions required for infection and spread of *P. ramorum* were satisfied, stems of not only Douglas-fir but also Sitka spruce and western hemlock might be at risk in the USA and Canada.

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Gray pine dwarf mistletoe, another California native.

Arceuthobium occidentale occurs commonly on gray pine throughout the foothills and low mountains surrounding the Central Valley, including the southern portions of Jefferson. Gray pine (formerly digger pine) has heavy, symmetrical, ovoid cones that open soon after maturing. Compared to western dwarf mistletoe, the gray pine dwarf mistletoe is light green to yellow (verse olive-green), finer (shoot diameter 1.5–4 cm versus 3–6 mm), shoots form more open clusters, and seldom induce production of brooms. The only mistletoes on gray pine are *A. occidentale* (for which it is a principal host) and *A. campylopodum* (for which it is an occasional host at certain localities outside our region).

Systematic Study of *Nattrassia mangiferae*, the Cause of Madrone Canker

Marianne Elliott and Robert L. Edmonds

Abstract—The fungus identified as *Nattrassia mangiferae*, which causes a canker disease of Pacific madrone, was studied using morphological and molecular methods. Only asexual spores were observed, but sequencing of the ITS region of the ribosomal rDNA places the sexual state in the genus *Botryosphaeria*. The fungus resembles *Fusicoccum anamorphs* of closely related *Botryosphaeria* species and has a similar pathology.

Introduction

The coelomycete fungus *Nattrassia mangiferae* has been implicated as the cause of a canker disease in Pacific madrone (*Arbutus menziesii* Pursh) (figures 1–2).

Figure 1—Declining madrone trees in Seattle, WA. Notice the extensive dieback in the crown and the bumpy, irregular appearance of the stems due to cankering.

Some anamorphs of *Botryosphaeria* are similar to *Nattrassia*, both in their behavior on the host plant and morphologically. They attack stressed, especially drought stressed, trees, and are primarily wound-invading. They cause canker and branch dieback, and are often endophytic, triggered to cause symptoms when the host is under drought, shade, or defoliation stress. These fungi primarily infect angiosperms but are found on some gymnosperms (*B. dothidea* on Sequoia and *Nattrassia* on pine).

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Figure 2—*Nattrassia* cankers on the bole of a young Pacific madrone. Older cankers are sunken. Staining under the bark may be necrosis from toxins produced by the fungus.

Nattrassia is a polymorphic fungus that has two spore stages, the pycnidial and the arthroconidial (*Scytalidium* state). Sutton and Dyko (1989) revised the genus *Hendersonula* and created the new monotypic genus *Nattrassia*, whose type species is *N. mangiferae*. Earlier names for this fungus have been *Dothiorella mangiferae*, *Exosporina fawcettii*, *Fusicoccum eucalypti*, *Hendersonula cypria*, *H. agathidis*, and *H. toruloidea*. The arthric syanamorph is known by the name *Scytalidium dimidiatum*, also *Torula dimidiata*, and *S. lignicola*. *Exosporina fawcettii* was originally described as a parasite of fruit trees in California and is similar to *Nattrassia* isolates from madrone. A sexual stage for *Nattrassia* has not been described.

Objective

The objective of this research was to determine the proper classification of *N. mangiferae* isolates from Pacific madrone. This was accomplished using cultural and molecular methods.

Methods

Morphology

Fifteen isolates of *Nattrassia* were grown on potato-dextrose agar (PDA) plates in the dark at 25° C for one week, then moved to a temperature-controlled room at 10° C with a 10 hour light and 8 hour dark cycle for six weeks. Macroconidia and arthrospores were measured in a compound microscope with an ocular micrometer. In addition, the shape, color, and septation was noted. Presence or absence of microconidia was recorded.

Molecular

Frozen mycelium was extracted and diluted to a concentration of 200:1 with sterile deionized water. PCR of the ITS region was performed in a 25 µl volume using 5 µl DNA and the primers ITS-1F and ITS-4. PCR products were purified and sequenced in the University of Washington Biochemistry DNA Sequencing Facility on the ABI 3700 high throughput capillary DNA analyzer.

ITS sequences from closely related species (determined from a BLAST search) were downloaded from GenBank and included in the analysis. The ingroup contained 23 isolates with aligned length 538 bp. *Sphaeropsis sapinea* (AY160200) was used as the outgroup. *Nattrassia* sequences which were identical to each other were removed from the analysis.

Data analysis

Conidial length, width, and length to width ratios, and arthrospore area (calculated using the formula for an ellipse) were compared for each isolate using 1-way ANOVA with SPSS version 10.0. Homogeneous subsets were determined using Tukey's HSD.

DNA sequences were aligned and edited using CLUSTAL X v. 1.8 (Thompson and others 1997). Phylogenetic relationships were determined using PHYLIP v. 3.573 (Felsenstein 1985) programs for parsimony, maximum-likelihood, and distance methods. To determine the support for each clade, 1000 bootstrap replicates were used (100 in the maximum likelihood analysis). Phylogenetic trees were drawn using the TREEVIEW program (Page 1996).

Results and Discussion

Morphology

Madrone isolates of *Nattrassia mangiferae* have hyaline, aseptate, guttulate conidia becoming 1- to 3-septate, verisicolored or brown (figure 3). Guttules were not visible in mature conidia. Conidia were thin-walled, and mostly fusoid in shape but occasionally oblong or clavate with truncate bases. The isolates differed in conidial length, width, and L:W, as well as in arthrospore size. Although there were statistically significant differences, there does not appear to be a geographical pattern in spore size or shape.

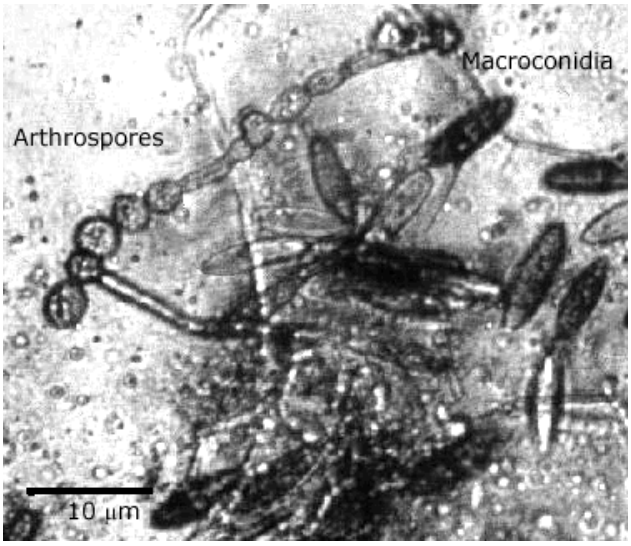

Figure 3—Arthrospores and macroconidia of *N. mangiferae* taken from a madrone canker.

Fusicoccum anamorphs of *Botryosphaeria* are described as having aseptate, hyaline conidia which may turn dark and septate with age. They are thin-walled and fusoid with truncate bases. A summary of conidium morphology and size is presented in table 1. *Nattrassia* is the only species in this group possessing arthrospores. *B. ribis*, its closest relative, has been reported to produce "chains of chlamydospore-like cells" (Rayachhetry and others 1996) or "swollen hyphal elements" (Morgan-Jones and White 1987) resembling arthrospores.

Phylogeny

ITS sequence analysis using all three methods placed the madrone isolates of *N. mangiferae* in the genus *Botryosphaeria* with *B. ribis* as its closest relative (figure 4). This dataset was not sufficient to resolve *B. dothidea* and *B. ribis* into strongly supported clades when *B. eucalyptorum* and *B. protearum* were added. However, other authors have

Table 1—Morphological characteristics of *Nattrassia mangiferae* and related species. ITS groups refer to Figure 4. Ranges of spore sizes are given in µm.

Species	Ascospores			Conidia		
	Length	Width	Description	Length	Width	Description
Nattrassia mangiferae (Sutton and Dyko 1989)	unknown	unknown	unknown	10–16 Arthrospores: 4–16.8	3.5–6.5 Arthrospores: 8.5	Hyaline, aseptate, fusoid, becoming 2–3 septate with central cell dark brown; L/W ratio= 0.6
Exosporina fawcettii (Wilson 1949)	unknown	unknown	unknown	11.9–16 Arthrospores: 4.6–7.3	3.2–6.5	Hyaline, aseptate, fusoid, becoming 2–3 septate with central cell dark brown;L/W ratio=2.7
Nattrassia mangiferae, Madrone isolates (ITS Group 2)	unknown	unknown	unknown	Macro: 15–25 Micro: 3-4 Arthrospores: 5-12.5	Macro: 5–7.5 Micro: 1 Arthrospores: 2.5-10	Macro: hyaline, aseptate, fusoid, becoming 2–3 septate with central cell dark brown; L/W ratio= 3.2; Micro: hyaline, oblong
Botryosphaeria ribis (Morgan-Jones and White, 1987, Rayachhetry and others, 1996) (ITS Group 1)	17–23	7–12	Hyaline, aseptate, ovoid	Macro: 17–25 Micro: 2–3 Chlamydo-spores: 18.6 (maximum)	Macro: 5.7 Micro: 1 Chlamydo-spores: 8.6 (maximum)	Macro: hyaline, fusoid, becoming 1-3 septate L/W ratio = 3.1;Micro: hyaline, allantoid
Botryosphaeria parva (Pennycook and Samuels 1985) (ITS Group 1)	18–27	8–11	Hyaline, aseptate becoming light brown and 1- or 2-septate.	15–20	4.5–6	Hyaline, aseptate, thin walled, rarely becoming darker and 1-septate, L/W ratio= 3.2
Botryosphaeria eucalyptorum (Smith and others, 2001) (ITS Group 3)	20–28	7–11	Hyaline, aseptate, granular, becoming light brown with age, fusoid	18–25	7–12	Hyaline, granular, ovoid to clavate, subtruncate base, aseptate; L/:W ratio= 2.2
Botryosphaeria dothidea (Crous and Palm 1999) (ITS Group 4)	18–25.5	7.5–12	Hyaline, aseptate, widest in the middle to upper third	21–28.5	4–4.5	Hyaline, aseptate, thin walled, rarely becoming darker and 1-septate; L/W ratio= 5.3
Botryosphaeria protearum (Denman and others 2003) (ITS Group 5)	25–37	9–13	Hyaline, aseptate, granular, becoming light brown with age, fusiform, inequilateral	Macro: 20–40 Micro: 3–6	Macro: 7–10 Micro: 1–1.5	Macro: hyaline, ovoid to clavate, granular, becoming irregularly fusiform, base bluntly rounded; L/W ratio= 3.5; Micro: smooth, aseptate, rod-shaped with rounded ends
Botryosphaeria luteum (Pennycook and Samuels 1985) (ITS Group 6)	18–22.5	7.5–12	Hyaline, aseptate	18–22.5	4.5–6	Hyaline, aseptate, thin walled; L/W ratio= 3.6

done this in datasets without these two closely related species (Jacobs and Rehner 1998, Smith and Stanosz 2001). There is some controversy over whether *B. ribis* and *B. dothidea* are different species or are both part of a single species complex. Isolates of *N. mangiferae* from Pacific madrone clearly belong in this group and are more closely related to *B. ribis* than to *B. dothidea*.

There were two smaller clades within *Nattrassia* due to a single bp difference in the 5.8s region. This region is identical within *Botryosphaeria* (Zhou and Stanosz 2000).

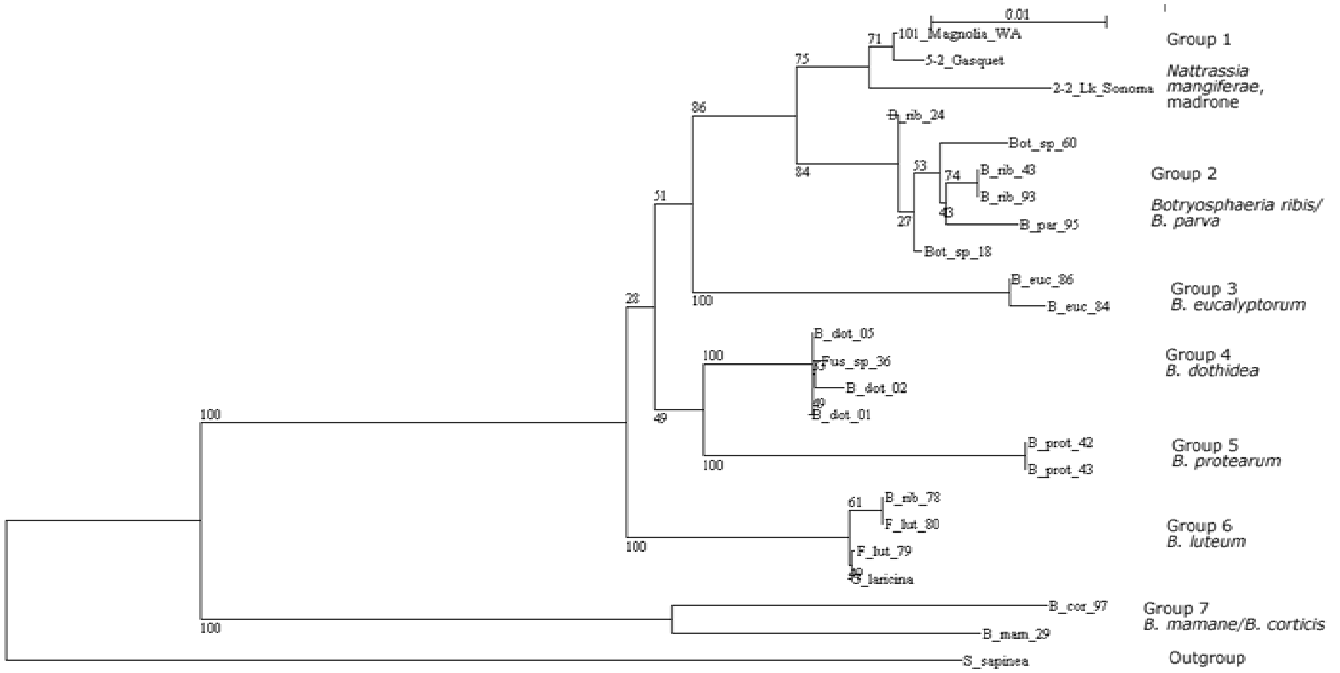

Figure 4—Phylogenetic relations among *Nattrassia mangiferae* isolates from Pacific madrone and other *Botryosphaeria* and *Fusicoccum* species using the NJ (Neighbor Joining) method of Saitou and Nei (Thompson and others. 1997). The distance matrix was calculated using Kimura's 2-parameter model (Felsenstein 1989). Statistical support for branches was based on 1000 bootstrap replicates.

Conclusions

Nattrassia mangiferae isolated from Pacific madrone belongs in the teleomorph genus *Botryosphaeria*. Disease cause by this fungus can be managed using methods developed for other *Botryosphaeria* pathogens, such as *B. dothidea*.

Acknowledgements

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Could White Pine Blister Rust Spread by Atmospheric Transport from California to New Mexico?

Katrina L. Frank, Laurence S. Kalkstein, Brian W. Geils, Harold Thistle, Eugene P. Van Arsdel

Abstract—White pine blister rust (WPBR) was introduced into western North America near Vancouver, British Columbia in 1910. The rust spread thereafter in a series of jumps during favorable years. A description of the spatial and temporal patterns of long-distance WPBR spread is useful for assessing the risk of further spread in the Great Basin, Southern Rockies, and into Mexico and investigating possible gene flow among western populations. We group patterns of large-scale, upper-level atmospheric conditions and rank these groups for transport capacity with an Upper Level Synoptic Index (ULSI). We then select and rank periods when surface conditions are considered favorable for aeciospore deposition, germination, and infection. Circumstantial evidence supports the hypothesis that WPBR in the Sacramento Mountains is the result of a single introduction by long-distant atmospheric transport from the Sierra Nevada and that spread in this situation is restricted not by transport opportunities but infection requirements.

Introduction

White pine blister rust (WPBR) was introduced into western North America near Vancouver, British Columbia in 1910 (Mielke 1943). The rust spread thereafter in a series of jumps during favorable years. By 1942, rust distribution included much of the range of white pines in British Columbia, Washington, Oregon, northern Idaho, and northern California (Mielke 1943). Within a few more decades, the rust spread throughout the Sierra Nevada, the Canadian–Northern Rockies, and into the Central Rockies (see Smith and Hoffman 2000). Hawksworth (1990) first reported WPBR in the Sacramento Mountains of New Mexico, over a thousand kilometers from the nearest known infestations in California, Idaho, or Wyoming. Van Arsdel and others (1998) suggested the New Mexico infestation may have resulted from long-distance, atmospheric transport rather than human introduction. Hamelin and others (2000) provided genetic evidence the New Mexico rust may have originated from California or other western locations.

A description of the spatial and temporal patterns of long-distance WPBR spread is useful for assessing the risk of further spread in the Great Basin, Southern Rockies, and into Mexico and investigating possible gene flow among western populations. In this study, we apply our understandings of rust epidemiology and meteorology to describe when and how often atmospheric conditions might be suitable for spread of WPBR from the Sierra Nevada and establishment in the Sacramento Mountains. We group patterns of large-scale, upper-level atmospheric conditions and rank these groups for transport capacity with an Upper Level Synoptic Index (ULSI). We then select and rank periods when surface conditions are considered favorable for aeciospore deposition, germination, and infection. Finally, we combine the upper level–transport index and the surface–infection index to determine the relative likelihood of successful rust spread from 1965 to 1974.

Methods

The Upper Level Synoptic Index provides a ranking to identify periods when atmospheric conditions over western North America are suitable for long-distance transport of rust spores. Atmospheric data are based on historic weather records (UCAR 2003) processed by the NCEP/NCAR Reanalysis Project (NOAA-CIRES 2002). The processed data matrix consists of four meteorological variables, projected on a 6-hour frequency (observations) over a 2.5˚ latitude by 2.5˚ longitude grid. The variables are height of the 500 mb pressure surface (m), specific humidity (kg/kg), u-wind component (m/s) and v-wind component. Our study area extends from 20ºN to 60ºN and 60ºW to 140ºW; our study tracks the period 1965 to 1974.

The data were systematically sampled and subjected to principal components analysis. Retained components were entered into a clustering algorithm and 16 clusters identified. Clusters were characterized by the season of their most common appearance and their structure. Isopleths of pressure surface height were mapped for each cluster using a median observation time-point (date and hour). The mean 500 mb flow pattern for each cluster was

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examined and rated for the degree to which the flow passes over the Sierra Nevada and Sacramento Mountains. The Summer-trough pattern (figure 1) was rated very high; the Winter-trough-ridge and Summer-trough-ridge patterns were rated as high; the Winter-zonal-barotropic was rated moderate; and all other clusters were rated as low likelihood of transport. Persistence of favorable flow for transport was determined with a complex algorithm using an 18-hour moving average of transport ranks (1 to 4) with a penalty to reduce scores of nonconsecutive events. With this procedure, we determined relative likelihood of upper level flow events from 1965 to 1974 for proper orientation and duration to transport rust spores from the Sierra Nevada to the Sacramento Mountains.

The surface condition analysis ranked periods for concordance of atmospheric conditions over the Sacramento Mountains with epidemiological requirements for rust spores establish infection. For this analysis, we assumed establishment could occur only during April, May, June, or July. Other assumptions were that germination and infection requires a period of at least 6 hours of saturated air at the leaf surface and a temperature above 13º C within 3 weeks of spore deposition. Our estimates of surface conditions were from the NCAR reanalysis data set and used the grid point nearest to the Sacramento Mountains. We assumed that saturated air at the leaf surface would correspond in this regional data set as a relative humidity greater than 85 percent. We used a double weighting algorithm to score periods of suitable humidity and temperature for duration and proximity to the transport event and rank periods from their position in the distribution of scores. We, thereby, determined relative likelihood of surface condition periods throughout 1965–1974 when season, humidity, and temperature in the Sacramento Mountains would allow for rust infection.

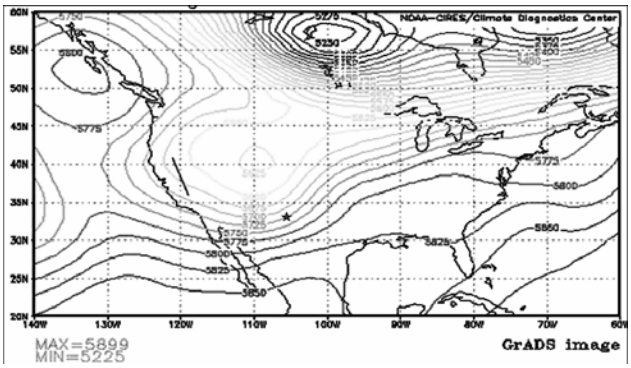

Figure 1—An upper level synoptic pattern for a Summer-trough over North America; also shown are the general location from which rust spores may originate(straight line) and the Sacramento Mountains, New Mexico (star). Air-flow and therefore spore transports tends to follow along pressure isobars (curved lines).

An infestation could only establish when atmospheric conditions permit both transport and infection. We summed the rank scores of transport and infection for each 6-hour observation and determined classification thresholds from a sensitivity analysis. With the combined score, we identified for each observation the relative likelihood of rust spread—transport and infection—as very high, high, moderate, or low.

Results and Discussion

Atmospheric conditions have developed and persisted numerous times from 1965 to 1974 that satisfy expected requirements for the transport of WPBR aeciospores from the Sierra Nevada to Sacramento Mountains and their subsequent infection.

A not-unlikely potential for atmospheric transport from the Sierra Nevada to Sacramento Mountains occurs when any one of four synoptic patterns develop. Upper level air flow from west to east, from California to New Mexico is a common situation. Flow from the north-northwest or north that could transport rust spores from Idaho or Wyoming to New Mexico, however, is very uncommon, occurring in only seven percent of observations.

A synthesis of meteorology and epidemiology allows us describe in detail when and how often atmospheric conditions are suitable over sufficient duration for transport followed by infection to occur. Although our rankings for transport, for infection, and their linkage are relative, they serve as a useful filter to sort among the thousands of observations and identify the most relevant periods when rust spread could occur.

For the study period seasons, May through July from 1965 to 1974, we identified 33 observations (0.68 percent of all observations) with a very high score for rust spread. These 33 observations were nested into five periods of various durations in four of the ten years examined. In 1972, there was a single observation; in 1971, there were two non-consecutive observations; in 1968, there were seven observations during the first week of July; and in 1969, there were 23 observations during the first two weeks of June. The single, extended period in early June, 1969 included three continuous periods of 24 to 42 hours when conditions for spread remained very high (figure 2). A successful establishment of WPBR in 1969 would be consistent with an estimate by Van Arsdel and others (1998) that the oldest cankers discovered in the Sacramento Mountains originated from infections occurring about 1970.

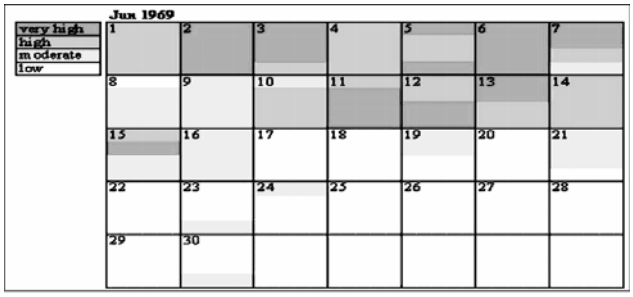

Figure 2—Calendar for June 1969, with periods indicated when conditions were very high, high, moderate, or low for long-distance spread and infection.

Conclusions and Implications

Direct observations of WPBR cankers date the rust as present in numerous locations of the Sierra Nevada after 1960 and in several locations of the Sacramento Mountains only ten years later. We have not yet found WPBR in Arizona or western New Mexico along the likely transport pathways. Hamelin and others (2000) found no genetic diversity within the Sacramento rust population. In this study, we found that upper level atmospheric conditions capable of transporting spores from the Sierra Nevada to the Sacramento Mountains are common (frequent each year), but only occasionally (at least once each decade) followed by surface conditions of proximity and duration suitable for infection.

Circumstantial evidence supports the hypothesis that WPBR in the Sacramento Mountains is the result of a single introduction by long-distant atmospheric transport from the Sierra Nevada and that spread in this situation is restricted not by transport opportunities but infection requirements. Although this is a single-case study, the synoptic methodology is readily applicable to other sites with and without current WPBR infestations. With further development, a relative hazard map of the southwestern US and northern Mexico could be produced.

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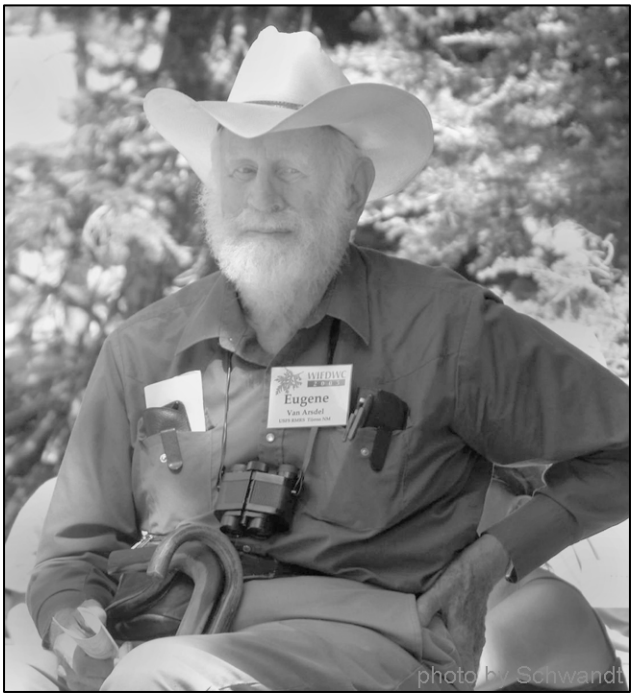

Lodgepole pine dwarf mistletoe, a little different.

Arceuthobium americanum is a common, widespread, and damaging mistletoe principally on lodgepole pine (*Pinus contorta*) but occasionally or rarely on several conifer species that occur within the Jefferson region—whitebark, Jeffery, and ponderosa pines and Douglas-fir. The shoots are a typical mistletoe height of 5 to 9 cm, but a little finer at 1–3 mm than many other mistletoe species. The most distinctive character (for mistletoes of Canada and the United States) is the verticillate branching pattern.

Mistletoe identification

The reduction and simplification of mistletoe morphology due to its parasitic habit and similarity due to its close phylogeny do make species identification difficult. Taxonomy and systematics are also controversial and therefore subject to different interpretation. Except in challenges such as a quiz to “Name that mistletoe”, we are interested in populations of plants in natural communities with known associations and locations. Host preferences, phenology, and disease reactions (brooming) are as informative as shoot and flower morphology (color, size, proportions, and branching pattern). Keys, text descriptions, and even pictures have some but limited value. Experience with the variety of populations and comparison to reference collections are proper. Generally, if you know the host, where you are, and what are the likely mistletoes, identification is possible.

A picture guide

Although we provided keys and descriptions in the Mistletoes of North American Conifers, cost and availability prohibited including a complete galley of detailed, color images of each mistletoe. Color photographs of each dwarf mistletoe were presented in the Hawksworth and Wiens monograph (Agriculture Handbook 709); these published and online images, however, were scanned from 35 mm slides of various quality.

A galley of high-quality, color portraits of each mistletoes species would provide a useful supplement to the Mistletoes of North American Conifers. I (Brian Geils) am now compiling and archiving such a gallery. I will accept and credit digital images in JPEG-format (with date, location, host, and identification data) from contributors. Each species would be represented by close portraits for staminate flowering, pistillate flowering and fruiting plants. Those judged best for illustrating the species taxonomically and aesthetically would be included in an online and printed publication.

Genetic Variation of *Armillaria ostoyae* within the Western United States.

J.W. Hanna, N.B. Klopfenstein, M.-S. Kim, G.I. McDonald, and J.A. Moore

Abstract—Intraspecific and intragenomic variation of *Armillaria ostoyae* were observed through sequencing of ribosomal DNA (rDNA) including nuclear large ribosomal subunit (nLSU), internal transcribed spacer (ITS), 5.8S rDNA, and intergenic spacer (IGS-1). Many of the *A. ostoyae* genets contained heterogeneous sequences, an indication of intragenomic variation/intraspecific hybridization. Intragenomic variation was verified by visual analysis of sequence chromatograms and PCR with specific internal primers. Intraspecific and intragenomic variation was found to exist in all rDNA regions analyzed, with the exception of the 5.8S rDNA. Variation will be further analyzed using Parsimony and Neighbor-Joining methods for phylogenetic analysis. Genetic diversity within *A. ostoyae* can be examined for relationships to ecological function (for example, pathogenicity, host specificity, and habitat type), geographic origin, forest management practices (for example, fertilization), and interactions among *Armillaria* genotypes.

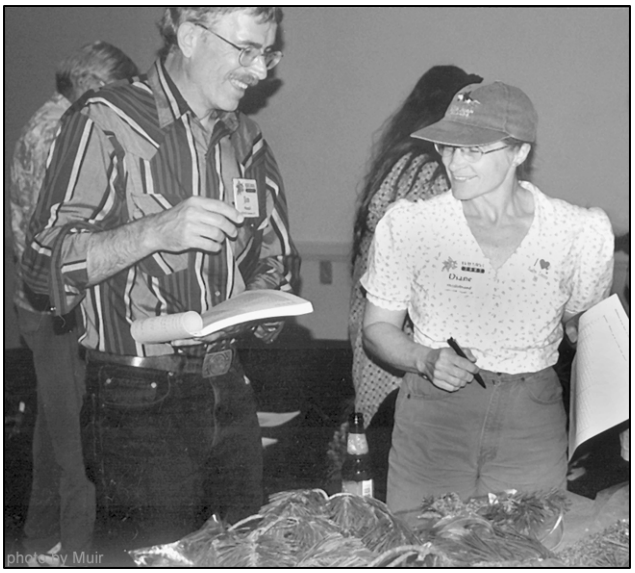

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photo by Angwin


photo by Pronos

First Report of A1 Mating Type of *Phytophthora ramorum* in North America

Everett M. Hansen, Paul W. Reeser, Wendy Sutton, Loretta M. Winton, Nancy K. Osterbauer

Introduction

Phytophthora ramorum is known from Europe and the West Coast of the United States (Davidson 2003). In Europe, it is found in nurseries and landscape plantings. In the United States, it has been confined to coastal forests and, in California, a few horticultural nurseries. All European isolates tested have been A1 mating type, while all Oregon and California isolates were A2 mating type (Werres 2001). AFLP markers also indicated that the populations on the two continents are genetically distinct and that nearly all North American isolates are from a single clone (Ivors 2002). In June 2003, *P. ramorum* was isolated from diseased *Viburnum* (figure 1), *Camellia*, and *Pieris* cultivars from two Oregon horticultural nurseries, one in Clackamas County and another in Jackson County (figure 2). As part of the effort to determine the origin of these infestations, we tested the nursery isolates for mating type and compared their genotypes with those of known European and Oregon forest isolates using DNA microsatellite markers.

Figure 1—Infected *Viburnum* in nursery production block.

Mating Types

Mating type was determined by pairing seven Oregon nursery isolates, three Oregon forest isolates (representative of the predominant North American clone), and two European nursery isolates. Agar plugs from 3-day old colonies were placed in close proximity on carrot agar plates. Plates were examined for oogonia (figure 3) after 3 days and 10 days (Brasier unpublished). Genotype was determined using four polymorphic microsatellite loci (figure 4) that distinguish *P. ramorum* isolates from Europe and North America.

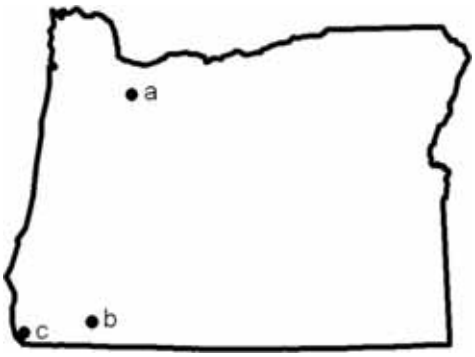

Figure 2—Locations in Oregon where *Phytophthora ramorum* has been isolated. a—nursery in Clackamas Co., b—nursery in Jackson Co., c—forest near Brookings.

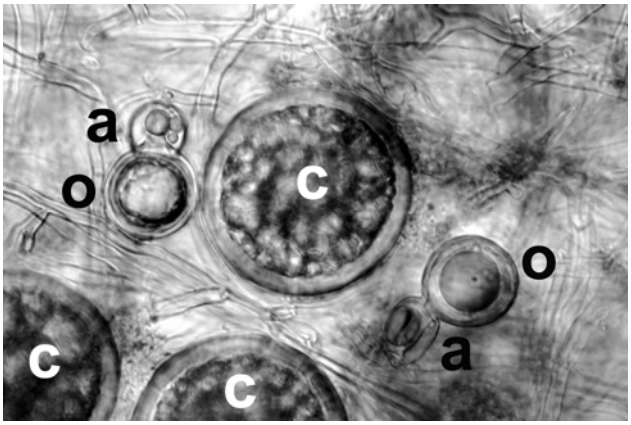

Figure 3—Oospores (o) with amphigynous antheridia (a) generated by paired A1 and A2 isolates of *P. ramorum*. Large spherical structures are chlamydospores (c).

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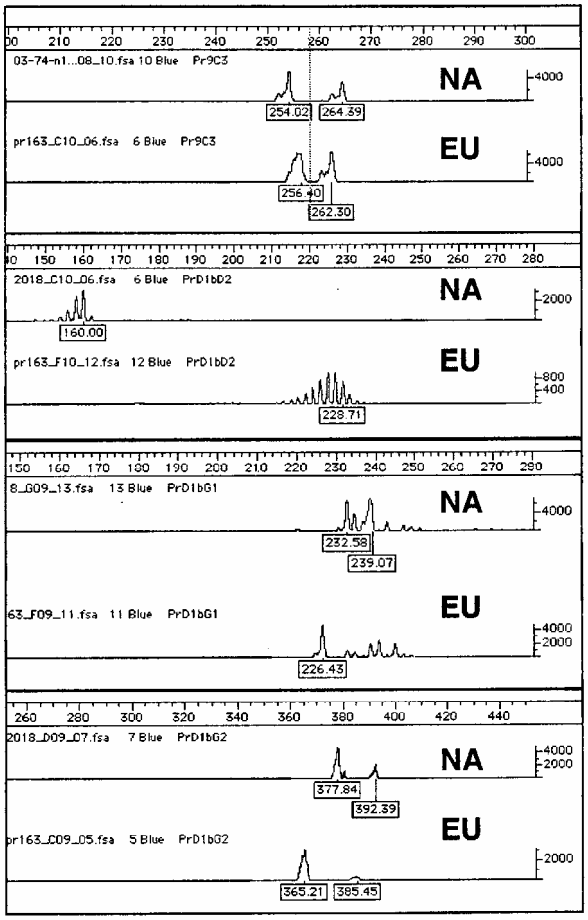

Figure 4—Allele sizes of North American (NA) and European (EU) *P. ramorum* isolates at four polymorphic microsatellite loci.

Oogonia and antheridia typical of *P. ramorum* formed when isolates from Clackamas County were paired with the Oregon forest isolates and when Jackson County isolates were paired with the European isolates. Sexual structures also formed in pairings between Oregon forest isolates and European isolates, but not in any other combinations. Microsatellite marker patterns of Clackamas County isolates were identical to the European isolates. Marker patterns of the Jackson County isolates were identical to the Oregon forest isolates (table 1).

Table 1—Mating type and microsatellite results.

Isolate	Micro-satellite	Mating Type	Host	Isolate Source
03-74-1	EU	A1	*Pieris* sp.	Clackamas
03-74-2	EU	A1	*Viburnum* sp.	Clackamas
03-116-1	EU	A1	*Viburnum* sp.	Clackamas
03-147-9	EU	A1	*Pieris* sp.	Clackamas
DO3-29B-1	EU	A1	*Viburnum* sp.	Clackamas
DO3-29B-2	EU	A1	*Viburnum* sp.	Clackamas
03-156-10B	NA	A2	*Camellia* sp.	Jackson
03-156-10A	NA	A2	*Camellia* sp.	Jackson
03-156-6	NA	A2	*Camellia* sp.	Jackson

These results indicate that the recent Oregon nursery infestations are of different origin. The Clackamas County isolates are of A1 mating type and European genotype. According to shipping records, the nursery has received no host nursery stock directly from Europe. However, host nursery stock has been received from a nursery in British Columbia, suggesting this may be the source of this infestation. The Jackson County isolates are of A2 mating type with a microsatellite pattern similar to the Oregon forest isolates. The latter result is consistent with the reported origin of these infested plants from a California nursery (CDFA, personal communication). The Oregon nursery infestations highlight the dangers of unregulated or under-regulated transport of host nursery stock from infested areas to non-infested areas. All host plants from infested nursery blocks at the affected Oregon nurseries have been destroyed by incineration (figure 5) and a regular monitoring program implemented. Other host nursery stock on site have been taken off-sale pending verification of free-from disease status per USDA, Animal and Plant Health Inspection Service requirements.

Figure 5—Incineration of all plants in an infested nursery block

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Is the Alternate Host for White Pine Blister Rust Present in Colorado?

Holly S. J. Kearns, William R. Jacobi, Kelly Sullivan, and Brian W. Geils

Introduction

White pine blister rust (*Cronartium ribicola*) is a disease of five needle pines. The disease recently spread into northern Colorado and threatens limber and Rocky Mountain bristlecone pine stands in the rest of the state. To determine if the alternate host (species of *Ribes*) is present near white pines in Colorado a survey was conducted in the summer of 2003.

Methods

A survey for the occurrence of *Ribes* species was conducted on four National Forests in Colorado (Pike, Rio Grande, Roosevelt, and San Isabel). This survey consisted of randomly located linear plots (20 ft by 200 ft) over a range of elevations, aspects, and habitat types on which the number of *Ribes* bushes, linear length of stems, and number of stems per bush by species were collected. On the four forests, 244 plots were established.

Results and Discussion

Five species of *Ribes* susceptible to white pine blister rust were found in the four National Forests surveyed within elevation ranges that correspond to the distribution of both limber and Rocky Mountain bristlecone pines (figures 1 to 5). Information was obtained on *Ribes* density, constancy, and elevation range (table 1 to 5). From these data it does not appear that the distribution of white pine blister rust within the sampled Colorado National Forests will be limited by the distribution of the alternate host. Future work will relate these data to developing a hazard model for white pine blister rust in the Central Rocky Mountains.

Figure 1—*Ribes cereum*. This currant is unarmed, has pink flowers, and red fruit.

Table 1—Distribution of *Ribes cereum* in four Colorado National Forests.

Forest	Density[a]	Constancy[b]	Elevation[c]
Pike	784 (2570)	0.18 (13/72)	9390–10600
Rio Grande	1241 (4184)	0.17 (11/62)	8790–11200
Roosevelt	1591 (3616)	0.49 (29/59)	7770–9005
San Isabel	2632 (9075)	0.21 (11/51)	8400–10065

[a]Mean ft of stem per acre (standard deviation).
[b]Fraction of plots occupied (plots occupied/examined).
[c]Elevation range, ft as lowest–highest observed.

Figure 2—*Ribes inerme*. This gooseberry usually has nodal spines and develops smooth red berry. The spots on this specimen are infections of *Cronartium ribicola*.

Table 2—Distribution of *Ribes inerme* in four Colorado National Forests.

Forest	Density[a]	Constancy[b]	Elevation[c]
Pike	3498 (10283)	0.34 (25/72)	9370–11800
Rio Grande	2967 (11512)	0.27 (17/62)	8670–11200
Roosevelt	1583 (2573)	0.61 (36/59)	7400–9005
San Isabel	2632 (3650)	0.43 (22/51)	8400–11501

[a]Mean ft of stem per acre (standard deviation).
[b]Fraction of plots occupied (plots occupied/examined).
[c]Elevation range, ft as lowest–highest observed.

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Figure 3—*Ribes laxiflorum*. This currant is distinguished by a creeping habit.

Table 3—Distribution of *Ribes laxiflorum* in four Colorado National Forests.

Forest	Density[a]	Constancy[b]	Elevation[c]
Pike	17 (135)	0.02 (2/72)	10700–10925
Rio Grande	109 (778)	0.03 (2/62)	9730–9970
Roosevelt	0	– (0/59)	–
San Isabel	0	– (0/51)	–

[a]Mean ft of stem per acre (standard deviation).
[b]Fraction of plots occupied (plots occupied/examined).
[c]Elevation range, ft as lowest–highest observed.

Figure 4—*Ribes lacustre*. This spiny currant has flowers similar to those of *R. montigenum*, but the glandular fruit develops a deep blue color and leaves are not hairy. (photo by M. Newcomb)

Table 4—Distribution of *Ribes lacustre* in four Colorado National Forests.

Forest	Density[a]	Constancy[b]	Elevation[c]
Pike	0	– (0/72)	–
Rio Grande	0	– (0/62)	–
Roosevelt	285 (1247)	0.10 (6/59)	8540–9390
San Isabel	0	– (0/51)	–

[a]Mean ft of stem per acre (standard deviation).
[b]Fraction of plots occupied (plots occupied/examined).
[c]Elevation range, ft as lowest–highest observed.

Figure 5—*Ribes montigenum*. This other spiny currant develops bright red, glandular fruits and usually has hairy leaves.

Table 5—Distribution of *Ribes montigenum* in four Colorado National Forests.

Forest	Density[a]	Constancy[b]	Elevation[c]
Pike	3183 (9784)	0.37 (27/72)	10000–12000
Rio Grande	6703 (14926)	0.48 (30/62)	9730–11840
Roosevelt	0	– (0/59)	–
San Isabel	1187 (3255)	0.17 (9/51)	10500–11800

[a]Mean ft of stem per acre (standard deviation).
[b]Fraction of plots occupied (plots occupied/examined).
[c]Elevation range, ft as lowest–highest observed.

Influence of Inoculum Source and Density on White Pine Blister Rust Infection of Whitebark Pine: Early Results

A. Kegley, R.A. Sniezko, R. Danchok, J. Danielson, and S. Long

Abstract —Performance of a bulked seedlot of whitebark pine exposed to a factorial series of white pine blister rust treatments (two inoculum sources x three inoculum densities) was examined. More than 95 percent of the seedlings developed needle lesions, and the percentage seedlings exhibiting stem symptoms ranged from 69 to 100 percent. While there were significant differences between the two inoculum sources in number of needle lesions and stem symptoms as well as percentage of seedlings with stem symptoms at the two early assessments (9 and 18 months after inoculation), these differences were not significant at the third assessment (22 months after inoculation). For inoculum density, however, there were significant differences in number of needle lesions, number of stem infections, and percentage seedlings with stem symptoms at all three assessments. Survival through 22 months after artificial inoculation was still high, ranging from 76.3 to 93.8 percent among the treatments. Thus far, there is no difference in survival based on inoculum source or density.

Introduction

All nine North American five-needle pine species (*Pinus* subsection *Strobus*) are susceptible to white pine blister rust, caused by the non-native invasive pathogen *Cronartium ribicola* J. C. Fisch. in Raben., with heavy levels of mortality occurring in natural stands as well as plantings of western white pine (*Pinus monticola*), sugar pine (*P. lambertiana*), and whitebark pine (*P. albicaulis*). Although whitebark pine is among the most highly susceptible species to the pathogen, most of the previous emphasis in developing blister rust resistant pine in western North America has focused on the more commercially valuable sugar pine and western white pine. However, whitebark pine is a keystone species in subalpine communities. It provides watershed protection, food for wildlife species, and is an important component of the aesthetics of high-elevation sites (Morgan and Murray 2001). Development of resistant populations of whitebark pine will be a key to maintaining this species.

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There is little published information about artificial screening of whitebark pine seedlings for blister rust resistance. Based on protocols developed for inoculating western white pine and sugar pine, seedlings of whitebark pine were subjected to a prototype inoculation trial in September 2001 at Dorena Genetic Resource Center (DGRC). Treatments consisted of a factorial of two inoculum sources and three inoculum densities. The objectives of this study were to help refine an inoculation and assessment protocol for operational screening of whitebark pine seedlings at DGRC and to examine gross differences in disease expression among seedlings exposed to two geographic sources of the pathogen. This paper presents preliminary analyses of results from the first two years following inoculation.

Materials and Methods

Plant Material

The whitebark pine seedlings were from a bulked seed collection (of >50 parent trees; CDA # 7425) from the Shoshone National Forest, southwest of Dubois, Wyoming (~43º30'N ~109º50'W, elevation ~2987 m) (E. Jungck, personal communication). There were two age classes of material: an April 1999 sowing (Ray Leach SC-10-Super Cells) and an April 2000 sowing (Beaver Plastic Styroblock 6S). Both sets of material were sown at the Forest Service Coeur d'Alene (CDA) nursery. The material from the 1999 sowing was transported to Dorena Genetic Resource Center (DGRC) in July 2001, and the 2000 sowing was delivered to DGRC in September 2001.

Inoculation

Inoculation treatments consisted of a factorial of two geographic sources of inoculum (*Ribes* sp. leaves infected with *C. ribicola* at the telial stage) and three targeted inoculum densities representing a low, medium, and high level (basidiospores/cm^2) (table 1). For the main study, seedlings from the 1999 CDA sowing were divided into six groups with approximately 48 seedlings per group. Each group of seedlings was allocated to one of the six treatment combinations (table 1). The inoculation treatments were randomly assigned in the chamber, and there was no

replication of the treatments until after inoculation. In addition to the main study, approximately 90 seedlings from the 2000 CDA sowing received the medium density Silver Lake treatment (table 1).

Table 1—Treatments of whitebark pine inoculated in September 2001.

Year Sown	*Ribes* Source[a]	Inoculum Density (spores/cm^2)[b]		Spore Germ[c]	Inoc. Time[d]
		Target	Actual (se)		
1999	MA	1000	825 (165.2)	95	15.5
1999	MA	2500	2500 (248.3)	87	26.5
1999	MA	5000	5150 (585.2)	99	40.5
1999	SL	1000	1000 (91.3)	99	11.0
1999	SL	2500	2625 (342.5)	100	47.0
1999	SL	5000	5400 (393.7)	87	37.3
2000	SL	2500	2625 (342.5)	100	47.0

[a] *Ribes* leaves collected from Silver Lake, Oregon (SL) and Mt. Adams, Washington (MA).
[b] Where 1000 spores/cm^2 represents a low density treatment, 2500 spores/cm^2 represents a medium density treatment, and 5000 spores/cm^2 represents a high density treatment.
[c] Basidiospore germination, percent.
[d] Time, hours, to reach target inoculum density.

Inoculation followed standard DGRC procedure. Temperature within the inoculation chamber was maintained at around 16.7° C (62° F) and relative humidity at 100 percent. Seedlings were moved into the inoculation chamber on September 4, 2001. Since the seedlings were in containers, standard DGRC boxes (0.9 m wide x 1.2 m long x 0.3 m high) were topped with plywood and wetted down. Racks or styroblocks of seedlings were placed atop the boxes.

Ribes sp. leaves infected with *C. ribicola* were collected from two sites located more than 150 miles apart: Silver Lake, central Oregon (SL) and Mount Adams, southwest Washington (MA). The leaves were placed, telial side down, on wire racks above the seedlings, and the structures were then covered with plastic tents (on September 5, 2001). Both *Ribes* sources were in the inoculation chamber at the same time, with the plastic tents in place to minimize potential contamination between inoculum sources. Spore fall was monitored until the desired inoculum density was reached for each treatment (table 1); the *Ribes* leaves were then removed. After the target inoculum density was reached for the last group, the seedlings were left in the inoculation chamber for approximately 48 hours at 20° C (68° F) to provide optimum conditions for basidiospore germination (table 1).

The seedlings were transplanted into three standard DGRC boxes on September 27, 2001. Each box contained 12 treatment row plots, with each plot having nine or ten trees. After artificial inoculation, each treatment was randomly assigned to five rows in a completely randomized designed with the exception of the outer two rows of each box. Those rows were planted with the smaller CDA 2000 seedlings inoculated with the SL source at 2500 spores/cm^2. During the transplanting, inoculum source information was lost for 29 seedlings (three-row plots) that had a target inoculum density of 5000 spores/cm^2.

Assessments

Seedlings were assessed in June 2002 (~9 months after inoculation) for height (cm) and number of needle lesions. Number of needle lesions, number of stem infections, and survival were assessed in March 2003 (~18 months after inoculation) and July 2003 (~22 months after inoculation).

Analyses of variance were performed using SAS Proc GLM (SAS 1999). Plot means by assessment date for seedlings in the six treatments for number of needle lesions, number of stem symptoms, percentage seedlings with needle lesions, percentage seedlings with stem symptoms, and percentage seedlings surviving at the third assessment (~22 months after inoculation) were used in the analyses. The model included the main effects of inoculum source and inoculum density as well as the interaction between inoculum source and density. Both inoculum source and inoculum level were considered fixed effects in these analyses. Seedlings from the CDA 2000 sowing and the seedlings with unknown inoculum source were excluded from the analyses.

Results

Inoculation Success

Inoculation was very effective. More than 95 percent of the seedlings in the six treatments showed needle lesions during at least one of the three inspections (table 2). Sixty-nine to 100 percent of the seedlings in the six treatments developed stem symptoms (table 2). Basidiospore germination was high for all treatments (87 to 100 percent, table 1).

Needle Lesions

Number of needle lesions ('spots') differed significantly among the two inoculum sources at the first assessment (9 months after inoculation (figure 1, table 3). Seedlings in the Silver Lake (SL) treatments had more needle lesions than those in the Mt. Adams (MA) treatments; treatment means ranged from 7.7 to 29.7 needle lesions per tree for SL compared to 4.8 to 12.1 for the MA treatments (table 2, figure 1). The main effect of inoculum source was not significant at either of the subsequent assessments (table 3, figure 1).

Table 2—Treatment means by assessment date (months after inoculation)

Year Sown	*Ribes* Source[a]	Inoc. density[b]	n[c]	Ht (cm)	Months after Inoculation										
					# needle lesions			percent with needle lesions			# stem symptoms[d]		percent stem symptoms		percent survival
					9	18	22	9	18	22	18	22	18	22	22
1999	MA	1000	46	10.6	6.8	19.5	17.7	95.8	100	100	1.4	2.5	56.3	69.2	93.8
1999	MA	2500	49	10.1	4.8	16.8	15.3	92.0	100	95.8	2.2	3.9	56.7	81.6	90.0
1999	MA	5000	28	10.4	12.1	20.3	27.0	100	100	95.8	4.8	7.2	85.2	100	76.3
1999	SL	1000	47	10.6	8.0	12.0	14.7	97.8	100	97.5	2.4	3.9	76.4	91.6	84.9
1999	SL	2500	49	11.0	7.7	15.3	19.4	93.8	97.8	95.8	3.0	3.8	69.6	81.8	82.0
1999	SL	5000	37	10.4	29.7	34.2	37.2	100	100	100	6.6	7.3	88.8	95.0	83.5
2000	SL	2500	60	5.9	4.7	5.5	11.7	95.0	93.3	85.9	1.9	3.0	81.7	88.3	76.7

[a] Geographic source of *Ribes*, the alternate host. MA=Mt. Adams, WA, SL=Silver Lake, OR.
[b] Targeted inoculum density (basidiospores/cm2), where 1000 represented a low density treatment, 2500 represented a medium density treatment, and 5000 represented a high density treatment. See Table 1 for actual inoculum densities.
[c] Total number of seedlings inoculated.
[d] Mean number of stem symptoms per infected tree.

In contrast, inoculum density significantly affected number of needle lesions at all assessments (table 3). Seedlings in the high density treatments had more needle lesions at all inspections than the lower density treatments (table 2, figure 1). There was evidence of a linear trend for number of needle lesions per seedling at all assessment dates (unpublished data). Within an inoculum source, the numbers of needle lesions in the low and medium density treatments were generally similar (figure 1, table 2). Also, the number of needle lesions per seedling increased in all treatments from the first to the second assessment. Four of the six treatments also showed a small increase in number of needle lesions from second to third assessment (table 2).

For number of needle lesions, the inoculum source x density interaction was significant for the first two assessments (~9 and 18 months after inoculation) but not for the third assessment (table 3). The interaction from the 9 month assessment is probably driven by the high number of needle lesions in the SL high density treatment (figure 1). At 18 months after inoculation (second assessment), the seedlings that received the MA treatments had similar numbers of spots at all inoculum densities (16.8 to 20.3) while seedlings treated with the SL source had a fairly linear increase in number of needle lesions as inoculum density increased (figure 1). Also in contrast to the 9 month results, the low density MA treatment had more needle lesions than the corresponding low density SL treatment at 18 months (figure 1).

Table 3—Analyses of variance F- and p-values for Whitebark Pine 2001 inoculation trial

Trait	F (p-value)		
	Source[a]	Density[b]	Source x Density
Number of Needle Lesions			
Assessment 1[c]	26.45 (<0.0001)	39.52 (<0.0001)	12.24 (<0.0001)
Assessment 2[d]	0.47 (0.4931)	8.35 (0.0003)	6.01 (0.0028)
Assessment 3[e]	1.70 (0.1940)	10.69 (<0.0001)	1.63 (0.1976)
Number of Stem Symptoms			
Assessment 2	7.43 (0.0069)	25.95 (<0.0001)	0.47 (0.6249)
Assessment 3	0.74 (0.3895)	19.01 (<0.0001)	0.83 (0.4385)
Percent with Needle Lesions			
Assessment 1	0.17 (0.6806)	1.79 (0.1915)	0.04 (0.9618)
Assessment 2	0.76 (0.3935)	0.82 (0.4524)	0.82 (0.4524)
Assessment 3	0.08 (0.7739)	0.98 (0.3927)	0.95 (0.4010)
Percent with Stem Symptoms			
Assessment 2	4.68 (0.0422)	6.22 (0.0076)	0.68 (0.5185)
Assessment 3	1.45 (0.2423)	4.53 (0.0232)	3.08 (0.0670)
Percent Survival			
Assessment 3	0.25 (0.6209)	0.68 (0.5180)	0.59 (0.5627)

[a] Inoculum source
[b] Inoculum density
[c] 9 months after inoculation
[d] 18 months after inoculation
[e] 22 months after inoculation

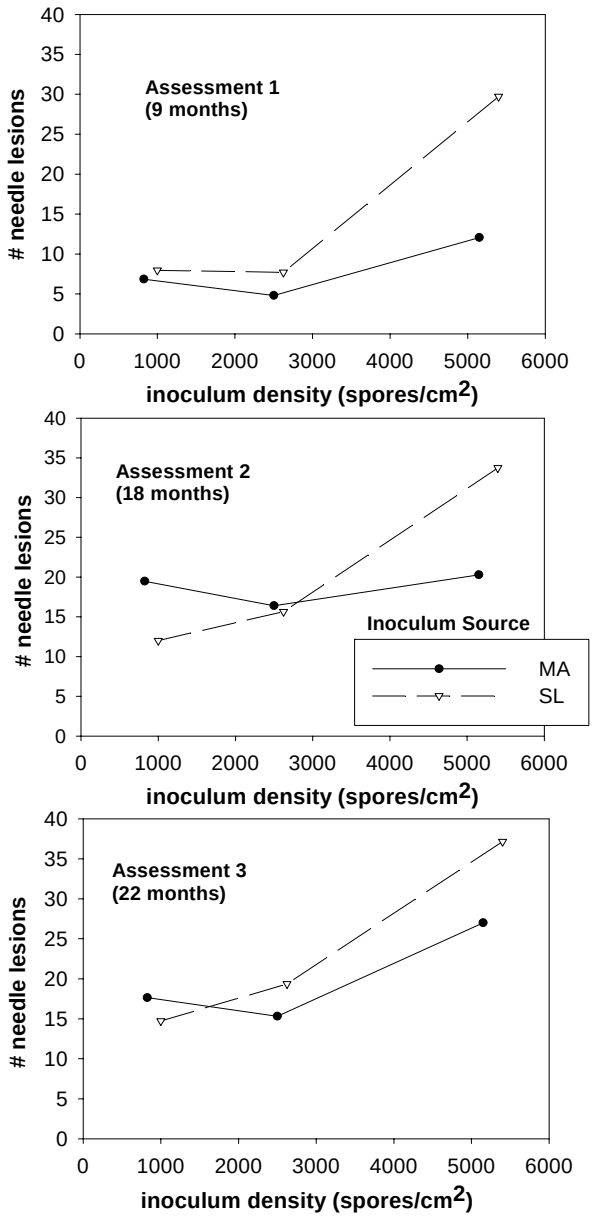

Figure 1—Number of needle lesions by treatment.

There was no difference among inoculum sources or inoculum densities for percentage seedlings with spots (table 3); treatment means varied from 92 to 100 percent at all assessments. It should be noted that the younger material (sown in 2000) tended to have fewer spots at all assessments (table 2).

Stem Symptoms

The main effect of inoculum source was significant at 18 months after inoculation for number of stem symptoms (SS) per infected seedling as well as percentage seedlings with stem symptoms (percent SS) (table 3). Eighteen months after inoculation seedlings that received the SL treatments had more SS per tree as well as a higher percentage trees with SS than seedlings in the MA treatments (figure 2, figure 3, table 2). This effect was not significant nearly 4 months later (22 months after inoculation) for either number of SS per infected tree or percentage seedlings with stem symptoms (table 3).

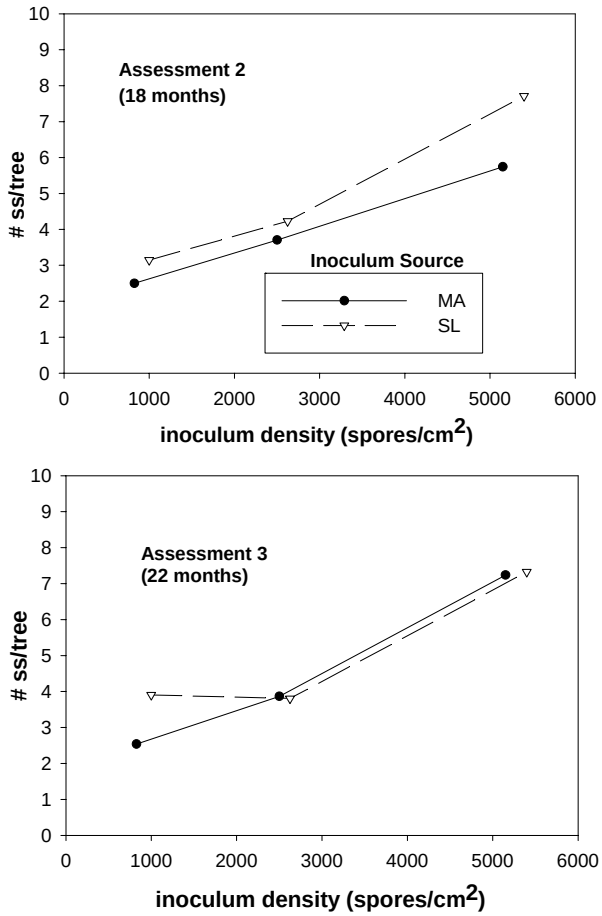

Figure 2—Mean number of stem symptoms (SS) per infected tree.

Number of stem symptoms per infected tree varied significantly by inoculum density at both 18 and 22 months after inoculation (table 3, figure 2). At the second assessment (18 months after inoculation) number of stem symptoms increased as inoculum density increased, regardless of inoculum source (figure 2). Treatment means varied from 1.4 SS/tree (MA 1000 spores/cm^2) to 6.6 SS/tree (SL 5000 spores/cm^2) at 18 months and from 2.5 (MA 1000 spores/cm^2) to 7.3 (SL 5000 spores/cm^2) at 22 months. At the third assessment, seedlings receiving the high density treatments had between three and five more stem infections than the seedlings at the lower densities (figure 2, table 2). All six treatments showed an increase in both SS and percent SS from the second to the third assessment (table 2).

There was a significant inoculum source by density interaction for percent seedlings with SS but not for number

of SS per infected tree 22 months after inoculation (table 3). There was also a change in rank in percent seedlings with SS at the third assessment (22 months after inoculation) (figure 3). The SL low density treatment had more than 20 percent more seedlings with SS (table 2, figure 3) than the corresponding MA treatment. At the medium density, the treatments had approximately the same percent SS (figure 3, table 2); the MA high density treatment reached 100 percent while the SL treatment had 95 percent SS.

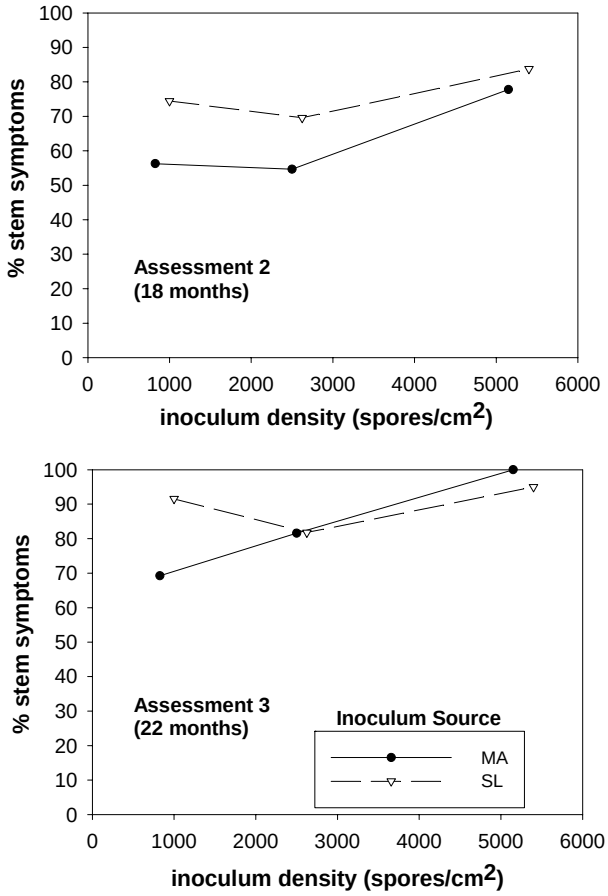

Figure 3—Mean percentage seedlings with stem symptoms.

Survival

In general seedlings receiving the SL treatments had lower survival 22 months after inoculation than seedlings receiving the MA treatments, except at the highest inoculum density (table 2, figure 4). However, these differences were not significant (table 3).

Seedlings inoculated with the SL source had similar levels of survival regardless of inoculum density (82 to 84.9 percent). However, for seedlings inoculated with the MA source, survival was lower for the high density treatment (90 to 93.8 percent versus 76.3 percent). These differences were not significant (table 3).

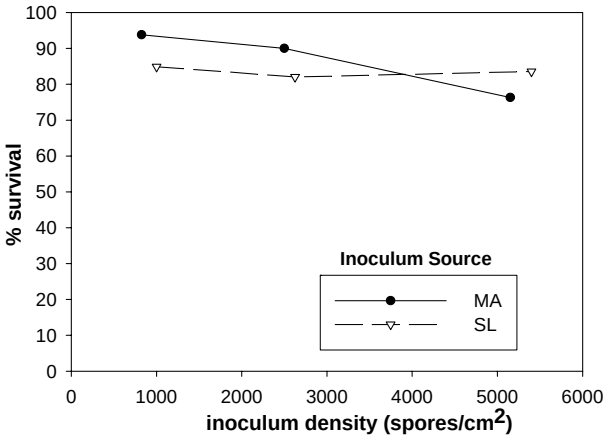

Figure 4—Mean percentage survival approximately 22 months after inoculation.

It is worth noting that the material from the smaller, seedlings from the 2000 sowing, which received the medium density SL treatment, had relatively low survival (76.7 percent).

2000 CDA Sowing

These seedlings received only the Silver Lake (SL) medium density treatment. These trees tended to have fewer spots, fewer stem symptoms, and lower survival relative to the larger 1999 seedlings.

Discussion and Summary

Based on preliminary results from the first two years of this study, the initial effect of inoculum source appears ephemeral. The early needle lesion and stem symptom counts (9 or 18 months or less after inoculation, respectively) tended to show greater differentiation in the development of disease symptoms between inoculum sources (SL or MA) than the later assessments (18 and 22 months after inoculation, respectively). Although the final results may show similar levels of infection, ontogeny of symptoms may vary depending on geographic source of the inoculum. Similarly, Meagher and Hunt (1999) reported that latency (mean years to first signs of stem rust) and infection (needle lesions or stem symptoms present) in western white pine were affected by inoculum source, but survival was similar across inoculum sources.

Results suggest that inoculum density, however, is important for a longer length of time. Both the low and medium density treatments had fewer needle lesions, fewer seedlings with stem symptoms, and fewer stem symptoms per infected tree than the high density treatments. It has been suggested that prevention of needle infection in western white pine is a threshold trait, dependent upon inoculation intensity (Hoff and McDonald 1980). Results from experiments using different inoculum densities of

fusiform rust (*C. quercuum* f. sp. *fusiforme*) on loblolly (*P. taeda*) and slash pine (*P. elliottii*) indicated that intermediate levels of resistance were more distinguishable at lower inoculum densities (Laird and others 1974).

At DGRC, standard inoculation densities for western white pine (WWP) and sugar pine (SP) are ~3000 and 6000 basidiospores/cm^2, respectively. Early reports indicated that whitebark pine is more susceptible to blister rust relative to WWP and SP (Bingham 1972).

For WWP and SP, number of needle lesions typically reaches a maximum approximately nine months after inoculation. At DGRC for WWP and SP, there is often a dramatic decrease in needle lesion number by 15 months after inoculation, the result of needle shed (unpublished data). In this trial the maximum number of visible needle lesions in whitebark pine appears to occur later relative to what is typical for WWP and SP. Additionally, the slight decrease in number of needle lesions or percentage seedlings with needle lesions could be due to loss of needles or merging of lesions.

Mortality of the seedlings has begun. Two years after inoculation, there was little difference in percentage survival among the SL treatments. However, the high density MA treatment had lower survival than the low or medium density treatments. Survival will be monitored for the next two to three years. Final readings will provide guidance on the importance (or not) of inoculum source, and which density is more effective for operational screening of whitebark pine.

Acknowledgments

Melissa Jenkins from the Caribou/Targhee NF provided the whitebark pine seedlings, and Ellen Jungck of the Shoshone NF provided background information on the seed collection. The Coeur d'Alene Nursery grew the seedlings for the first two years for the 1999 sowing and for the first year for the 2000 sowing. Dorena GRC technicians and staff inoculated the seedlings with blister rust, provided nursery culture after inoculation, and assessed the seedlings. The USDA Forest Service Region 6 Genetics and Forest Health programs provided funds to carry out the study.

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Characterization of North American *Armillaria* Species: Phylogenetic Relationships from Ribosomal DNA Sequences.

M.-S. Kim, J.W. Hanna, N.B. Klopfenstein, P.J. Zambino, and G.I. McDonald

Abstract—Phylogenetic relationships among nine North American *Armillaria* spp. were analyzed using ribosomal DNA (rDNA) sequences from intergenic spacer 1 (IGS-1), internal transcribed spacer (including 5.8S rDNA) (ITS+5.8S), and nuclear large subunit rDNA (nLSU) regions. Phylogenetic trees were generated using Neighbor-Joining analysis. The IGS-1, ITS+5.8S, and nLSU sequence data indicate that *A. mellea* is distant from other *Armillaria* spp. *Armillaria ostoyae* and *A. gemina* were well separated from the other *Armillaria* spp. Several *Armillaria* spp. (*A. calvescens*, *A. sinapina*, *A. gallica*, NABS X, and *A. cepistipes*) clustered together, despite their previous separation based on in vitro compatibility and/or morphology. A more detailed phylogenetic analysis and an examination of hybridization among *Armillaria* spp. are underway.

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J.W. Hanna is associated with the U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station at Moscow ID 83843 and with the Department of Forest Resources, University of Idaho, Moscow ID 83844.


photo by Jacobi

Tree Pathogen Survival in Wood Chip Mulch

Ronda. D. Koski and William R. Jacobi

Abstract—Wood chips are commonly used as landscape mulches. Wood chips are derived from trees removed from landscapes and solid wood packing materials. We wanted to know if wood chips could harbor pathogens that cause tree diseases and provide a means for pathogen movement. A study was initiated to determine the longevity of survival of *Thyronectria austroamericana*, the cause of Thyronectria Canker in honeylocust trees, in uncomposted wood chip mulch. Cankered wood pieces produced from inoculated branches of honeylocust trees were placed into mulched areas in an irrigated landscape. Cankered wood pieces were periodically collected and samples placed on agar plates. Recovery of *T. austroamericana* occurred from cankered wood pieces collected after 143 weeks in the mulch. *T. austroamericana* recovered from cankered wood pieces after 98 weeks produced cankers when inoculated into honeylocust branches. Irrigation treatments did not impact recovery of *T. austroamericana*. Since wood chips derived from landscape trees can provide a source of inoculum for pathogenic fungi, additional studies are warranted to investigate the survival of indigenous and/or exotic plant pathogens in chipped solid wood packing materials.

Materials and Methods

Production of Cankered Wood Pieces

An 8 mm diameter cork borer was used to produce wounds at 15 cm intervals along 2 to 4 cm diameter honeylocust branches. A 6 mm plug of one of two isolates of *Thyronectria austroamericana* was placed in each wound, and wrapped with wax film. After fourteen days, branches were cut into 6.5 cm sections so that each piece contained one-half of an inoculation wound (figure 1).

Figure 1—Cankered wood piece on mulch surface.

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Study area

The study area, located near Fort Collins CO, consisted of nine irrigated blocks. A typical turf-type irrigation system with pop-up heads was designed with controls that allowed each of the nine blocks to be controlled independently. Three irrigation treatments, based on levels of measured evapotranspiration (ET) for alfalfa, were replicated three times. The irrigation treatments consisted of 'low' (40 percent of ET), 'medium' (80 percent of ET), and 'high' (160 percent of ET). Only two ‘low’ irrigation blocks and two ‘high’ irrigation blocks were used in this study. Within each block were three-row plots of ‘Skyline’ honeylocust (*Gleditsia tricanthos inermis* 'Skyline') ; and ‘Livingston’ Kentucky bluegrass (*Poa pratensis* 'Livingston') grew throughout the nine-block study area. Four honeylocust trees in the middle row of each of selected block were utilized in this study. Turfgrass was removed from around the base of the trees and a ring of plastic landscape edging material, 0.75 m in diameter, was placed around the base of each tree (figure 2). Within each ring, uncomposted cottonwood wood chips (derived from *Populus deltoides*) were placed to a depth of 10 cm. Cottonwood wood chips were used because species of *Populus* are not hosts of *T. austroamericana*.

Figure 2—Cankered wood pieces in mulch.

Placement of cankered wood pieces in mulch layer

Cankered wood pieces were attached to landscape staples using acrylic yarn. Half of the wood pieces attached to each landscape staple were positioned 10 cm under the mulch (that is, buried); the remaining half were positioned so that they rested on top of the mulch (that is, on surface). Three separate yet similar experiments were conducted between 1998 and 2002.

Analysis of cankered wood pieces

On each collection date, 32 to 64 cankered wood pieces were collected and assessed for percent moisture and fungal viability. A band saw was used to cut a 2.5 cm portion from each cankered wood piece that was weighed and then oven dried and weighed again to calculate the percent moisture. The remaining 4 cm portion of each cankered wood piece was surface disinfected using a 10 percent bleach solution; bark was removed, and eight wood chips were cut from each cankered wood piece (figure 3). These eight wood chips were placed on two petri dishes containing PDA amended with 10 ppm streptomycin. Over a period of 14 days, each plate was examined periodically for the presence of *Gyrostroma austroamericanum* Seeler, the imperfect state of *T. austroamericana*. When grown on PDA, *G. austroamericanum* produced mycelium that appeared wet and pinkish-orange in color (figure 4).

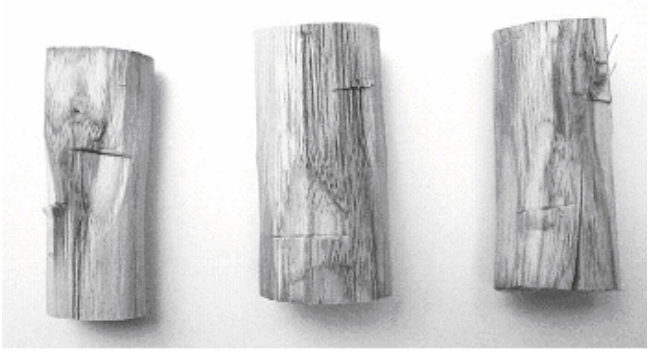


Figure 3—Reddish stain of *T. austroamericana* in wood pieces.

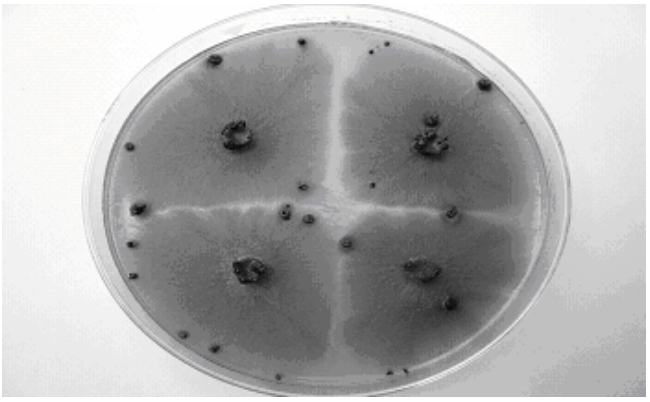


Figure 4—Culture of imperfect state of *T. austroamericana on PDA.*

Pathogenicity of recovered isolates

Thyronectria austroamericana isolates recovered from cankered wood pieces after 98 weeks were used to inoculate branches of honeylocust trees. Six honeylocust trees from two 'low' irrigation blocks; six honeylocust trees from two 'medium' irrigation blocks; and six honeylocust trees from two 'high' irrigation blocks were inoculated with the recovered isolates. On a different branch, inoculations were made using the original two fungal isolates. Each inoculation site was wrapped with wax film and allowed to incubate for two weeks, after which the wax film was removed. Cankers typical of those caused by *T. austroamericana* developed at sites inoculated with the recovered and original isolates (figure 5).

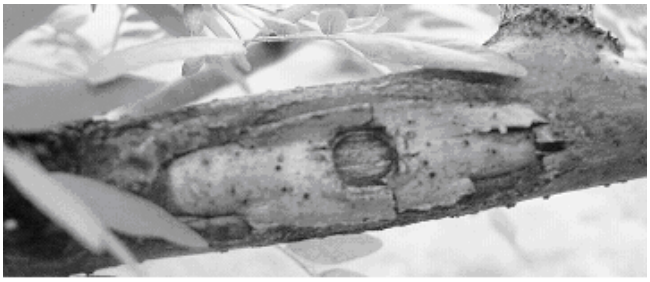


Figure 5—Pathogenicity test with recovered isolate.

Results

Experiments One and Two

T. austroamericana survived in both surface and buried wood pieces for 9 and 10 weeks.

T. austroamericana was viable in 17 percent to 93 percent of the wood pieces after 9 and 10 weeks.

Recovery of *T. austroamericana* was significantly less in surface versus buried pieces.

Percent moisture was significantly less for the surface pieces (11 percent) versus buried pieces (35 percent).

Experiment Three

T. austroamericana survived in both surface and buried wood pieces for over 143 weeks (figure 6).

Percent moisture was significantly less for the surface (10 percent) versus buried pieces (25 percent).

Irrigation treatments had little impact on recovery of *T. austroamericana*.

Conclusions

Recovery of *T. austroamericana* after 143 weeks indicates that the pathogen can survive as an inoculum source.

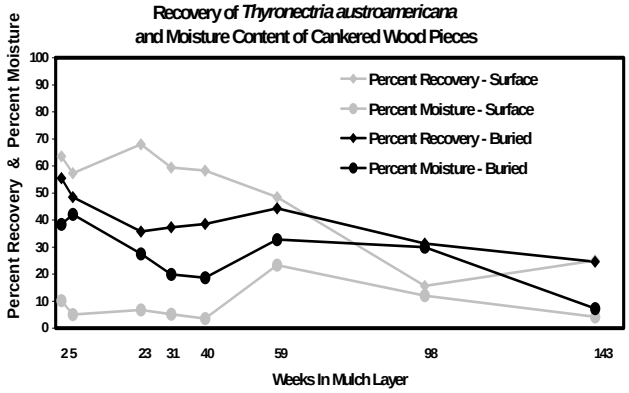


Figure 6—Recovery and moisture content over time.

Recovery of *T. austroamericana* was not affected by the irrigation treatments.

Recovery of *T. austroamericana* decreased over time.

Recovery of *T. austroamericana* during the winter indicates that the pathogen can withstand freezing temperatures.

Even though surface pieces dried out more than buried pieces, recovery of *T. austroamericana* was affected by position in the mulch layer only for a few months.

T. austroamericana isolated from cankered wood pieces that had been on or in mulch layers for 98 weeks produced typical Thyronectria Canker lesions.

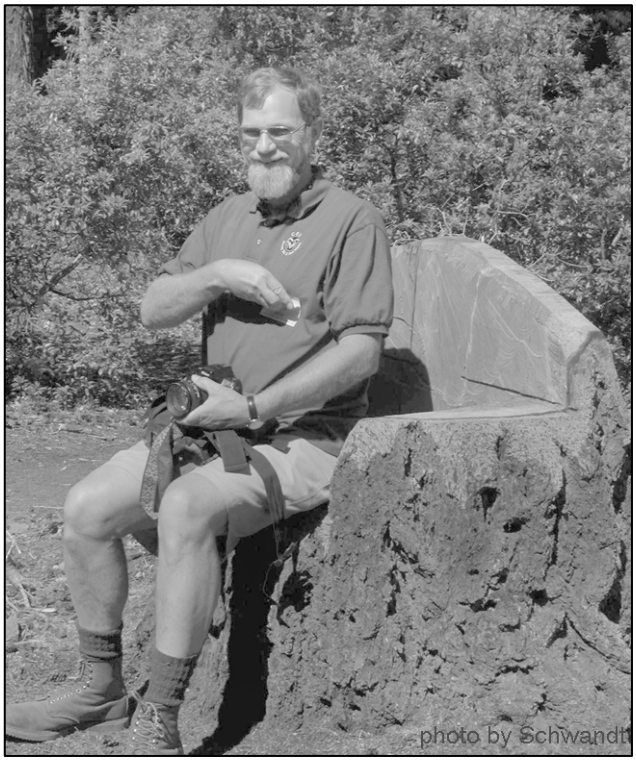
The results of this study suggest that infested wood chip mulch can be a source of inoculum for plant pathogens; infested mulch may allow fungal pathogens to survive for prolonged periods until environmental conditions and horticultural practices favor host infection (figure 7).

Figure 7—Tree debris to be chipped into landscape mulch.

Further research is needed to determine pathogen survival during the process of chipping trees and/or solid wood packing materials, dying of chips, and storage in mulch piles.

The risk of introducing exotic plant pathogens via the importation of wood-based packing materials into the United States is increased by the use of these materials for landscape mulch.

The findings from this research suggest that the risk of introducing exotic plant pathogens and other pests is real, and may justify stricter regulations on wood packing materials.



There once was a student from Korea
Who had a bad case of diarrhea
We gave her some wine
After which she felt fine
But then she was out on her ear a

Composed by: Clive Brasier, Joan Webber, Eun Sung Oh, Kathy Lewis, John Orlowsky, and unknown graduate.

Wood-Decomposing Fungi on Fertilized Sites in the Northwestern U.S.A.

Raini C. Rippy, Ned B. Klopfenstein, Mee-Sook Kim, Paul J. Zambino, Deborah S. Page-Dumroese, James A. Moore, and Paul A. McDaniel

Abstract—Soil organic matter (SOM) is important to the productivity of forest soils because of its roles in water availability, nutrient supply, soil aggregation, and support of soil organisms. This study is aimed toward evaluating the effects of forest management (fertilization) on decomposition rates and wood-decomposing fungi. Decomposition rates of wood stakes from different species (loblolly pine, aspen, and Douglas-fir) are being examined at six fertilized sites in Idaho and Washington to compare fungal diversity across different soil and habitat types. Sapwood stakes were installed in the mineral soil and at the mineral soil/litter layer interface. Fungi isolated from stakes collected at six-month intervals will be identified through molecular techniques (ITS sequencing) and morphological examination.

In: Geils, B. W. comp. 2004. Proceedings of the 51st Western International Forest Disease Work Conference; 2003 August 18–22; Grants Pass, OR. Flagstaff, AZ: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station.

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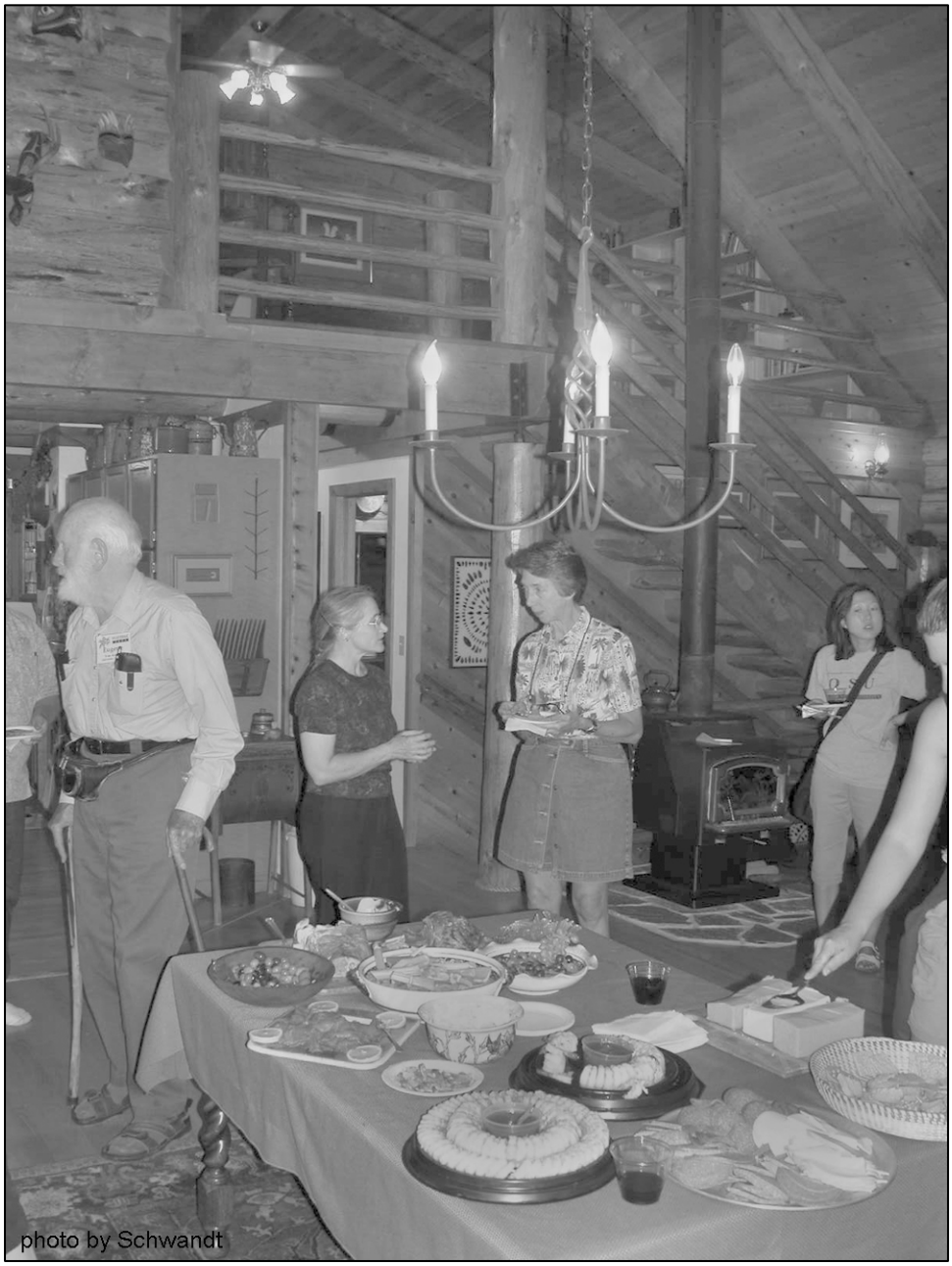
photo by Schwandt

Simply Inherited Resistance to *Phytophthora lateralis* in Port-Orford-Cedar: Greenhouse Testing

R.A. Sniezko, E.M. Hansen, and S.E. Kolpak

Abstract—*Phytophthora lateralis*, a non-native root rot pathogen, is the primary cause of Port-Orford-cedar (*Chamaecyparis lawsoniana*) mortality throughout its native range in southwest Oregon and northwest California. Most trees in field are very susceptible, but greenhouse testing in the late 1980s demonstrated existence of genetic resistance to this pathogen. Greenhouse studies to examine the magnitude and inheritance (whether Mendelian or polygenic) of this resistance have utilized a root dip technique using young seedlings. Susceptible families show 90 to 100 percent mortality over the 10 to 11 month evaluation period, but a few full-sib families show little (0–10 percent) or only moderate (25 to 60 percent) mortality. Results from full-sib families from control crosses using the most resistant parents indicate that a major gene for resistance is present, with survival ratios of 3:1, and 1:1 depending on the parent and cross. There are a few deviations from the survival ratios, including two selfed crosses. A putative homozygous dominant parent, three heterozygous parents, and three homozygous recessive parents were identified from the analysis. The data cannot preclude a mixed mode of inheritance (Mendelian and polygenic) across the entire range of Port-Orford-cedar or multiple resistant mechanisms.

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photo by Schwandt


photo by Angwin

Exotic Pathogens, Resistant Seed, and Restoration of Forest Tree Species in Western North America

Richard A. Sniezko, Diana F. Tomback, Regina M. Rochefort, Ellen Goheen, Rich Hunt, Jerry S. Beatty, Michael Murray, and Frank Betlejewski

Abstract—Non-native invasive pathogens such as white pine blister rust (*Cronartium ribicola*) and Port-Orford-cedar root disease (*Phytophthora lateralis*) are killing trees and disrupting forest ecosystems in western North America. Populations of western white pine (*Pinus monticola*), sugar pine (*P. lambertiana*), whitebark pine (*P. albicaulis*), and limber pine (*P. flexilis*) are declining precipitously from damage by blister rust. Foxtail pine (*P. balfouriana*) and southwestern white pine (*P. strobiformis*) populations are also infected by blister rust in parts of their range. *P. lateralis* continues to spread and kill Port-Orford-cedar (*Chamaecyparis lawsoniana*) in Oregon and California. Because resistant individuals in all these species are rare, genetic variation may be reduced to the point where future populations may not be viable without active management. Seeds from resistant parents are now available for western white pine, sugar pine, and Port-Orford-cedar restoration for some areas. Selection and breeding programs for resistance, coupled with active ecological management, will be needed to create opportunities to restore and retain these species in forest ecosystems on federal or crown lands. Any strategy for restoration must include opportunities for natural regeneration and planting, with the goal of maintaining these species on the landscape until resistance characterizes populations. Intervention to restore more natural conditions in wilderness may be evaluated case-by-case, and weighed against other wilderness values. Organizational and implementation strategies that are developed for managing *C. ribicola* and *P. lateralis* can provide a starting point for work involving other introduced pathogens. See the poster at http://www.fs.fed.us/r6/dorena/posters/index.shtml for more details.

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Michael Murray is with the USDI National Park Service, Crater Lake National Park Service, PO Box 7, Crater Lake, OR 97604.

photo by Angwin

Panel: Quarantines and Problematic Genetic Exchange

Susan Frankel, Moderator

The impetus for this panel comes from two bitter and difficult experiences in California with forest pathogens and quarantines—*Fusarium circinatum* (cause of pitch canker) in the mid-1990s, and more recently, *Phytophthora ramorum* (cause of sudden oak death and other diseases).

Despite lengthy debate, *Fusarium circinatum* was never quarantined. I understand now, after struggling with the *P. ramorum* quarantine, why regulatory agencies refused to quarantine *F. circinatum*. Part of the problem with the debate was, we didn't know what we were asking for. We, forest pathologists in California, did not fully understand what a quarantine is, and how it is enforced.

Program

Our objective in today's panel is to provide a basic understanding of quarantines and how they are developed and enforced in North America. We will also suggest ways the systems used to prevent pathogen establishment may be improved. We have assembled the following speakers:

History of forest disease quarantines in the United States. Borys Tkacz, National Program Manager, Forest Health Monitoring, Arlington, VA.

The regulatory system for forest pathogens in North America, the European Union, and other parts of the world: a pop quiz!. Susan Frankel, Plant Pathologist, Pacific Southwest Region, Forest Health Protection, Vallejo, CA.

***Phytophthora ramorum* quarantine: Challenges of regulating a new organism with a wide host range.** David Rizzo, Associate Professor, UC Davis, CA.

Preventing exotic pathogen threats to forests—a sideways scientific look. Clive Brasier, Pathology Branch, Forest Research, Farnham, Surrey, UK.

Current challenges in forest pathogen protection. Faith T. Campbell, Director, Invasive Species Program, American Lands Alliance, Washington, DC.

Recipe

I will start off with a demonstration of how the quarantine for *Phytophthora ramorum* was formulated.

Recipe for the *Phytophthora ramorum* (Sudden Oak Death) quarantine

☞ Mix in a standard blender:

✓ 1 new red potato (forest pathologists)

✓ 78 peanuts (local citizens)

✓ 1 snack-size box raisons (media and legislators)

✓ 1 box, individual serving-size, Fruit Loops cereal (State regulatory agencies)

✓ 1 cup ice (Federal regulatory agency)

✓ 58 cloves garlic (California County Agricultural Commissioners)

✓ 1 miniature baseball bat (special interest groups)

✓ 1 sugar pine cone (Native American Tribal Members).

⌛ Puree for three years.

👌 Add additional spices, fruits, and nuts upon need or demand. It may be difficult to fit some items in the blender and combine them.

! [Should anyone ask: 1-c, 2-c, 3-c, 4-a, 5-c, 6-d, 7-a,b,c,d]

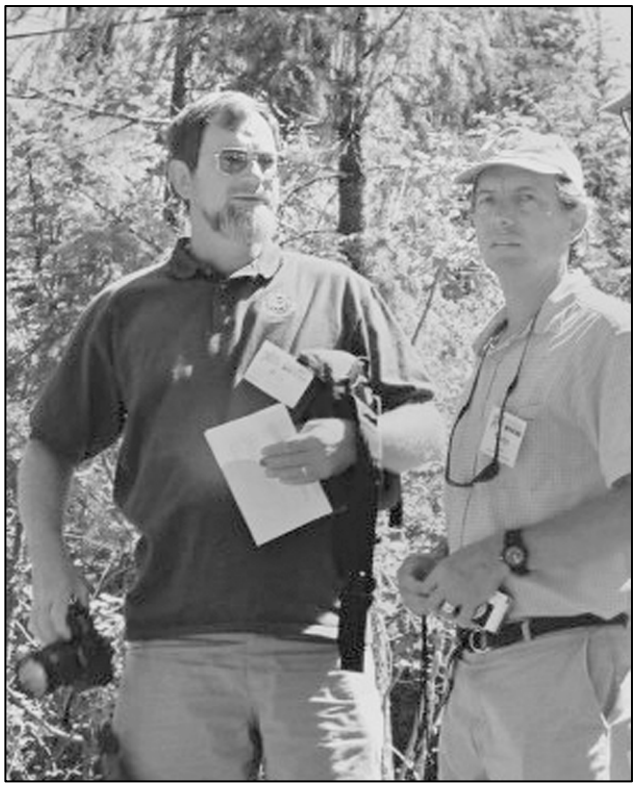


photo by Schwan

History of Forest Disease Quarantines in the United States

Borys M. Tkacz

Abstract —Invasive forest pathogens present significant threats to the health of forests. Previous introductions of invasive forest pathogens into new environments have resulted in severe tree diseases with long-lasting economic and environmental impacts. Quarantine regulations have been promulgated in attempts to limit the spread of established invasive forest pathogens and prevent new introductions. This paper reviews the history of quarantines against invasive forest pathogens in the United States and discusses challenges associated with preventing new introductions.

Introduction

Introductions of non-native forest pathogens into North American forests have resulted in devastating diseases affecting many native and planted forests. Some of the most damaging forest pathogens have appeared since 1900 (Hepting 1964; table 1).

Table 1—Diseases caused by exotic forest pathogens.

Disease	Hosts	Year Discovered
Chestnut blight	*Castanea*	1904
Blister rust	*Pinus* (5-needled)	1906
Beech bark disease	*Fagus*	1920
Larch canker	*Larix*	1927
Dutch elm disease	*Ulmus*	1930
Littleleaf	*Pinus*	1932
Persimmon wilt	*Diospyros*	1933
Mimosa wilt	*Albizia*	1935

The sudden appearance of these and other damaging plant pathogens led to the development of quarantine laws aimed at halting the spread of diseases to new locales. This paper summarizes the history of quarantines against invasive forest pathogens in the United States (US) and discusses the challenges associated with preventing the spread of forest diseases.

Early Quarantine Laws

Discovery of large numbers of brown tail moth and gypsy moth on fruit stock arriving in US in 1909 and 1910 led to introduction of a plant quarantine bill in the House and Senate in early 1909 (Johnson 1964). Although it was initially thwarted by strong nursery interests, the bill finally passed on August 20, 1912 as the **Plant Quarantine Act** (PQA). The Act included the following provisions:

Required permits for imports and inspections of nursery stock;

Notification of Secretary of Agriculture of the arrival of any nursery stock at port of entry and identification of source;

Authority for Sec. of Ag. to promulgate regulations regarding international or interstate quarantine restrictions;

Penalties for violations—misdemeanor—$500 fine and/or one year imprisonment;

Appropriated $25,000 to Sec. of Ag. to carryout provisions of Act.

Quarantine No. 1—The first foreign quarantine promulgated under PQA was to prevent further introduction of the white pine blister rust fungus (*Cronartium ribicola*) by restricting importation of 5-needle pines from "each and every country of Europe and Asia". Subsequent rules targeting forest trees were merely repetitions of the minimum provisions of the bill itself:

Unlimited importation of forest trees was allowed;

Foreign inspection and certification were required;

Additional re-inspection was to be done at destination in US by State inspectors;

Inspections were limited to above-ground portions of plants.

Quarantine No. 26—issued on April 21, 1917—authorized destruction of *Ribes* to control the spread of white pine blister rust. Emphasis was on cultivated European black currant, but all *Ribes* were prohibited within "infection distance" of white pine. There were no provisions for compensation to owners. This provided the basis for a large-scale wild *Ribes* eradication program—"the most extensive forest disease control effort in time, money, men, and materiel in the history of US forestry" (Maloy 1997).

Quarantine No. 37—issued on June 1, 1919—excluded most classes of nursery stock and trees from importation to the US. Admissible plants had to be free of soil and were

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subject to treatment as a condition of entry. This ushered in a "strengthening of our system of plant protection on a valid biological basis" (Johnston 1964). Quarantine 37 was revised on January 1, 1949 and merged a variety of rules, regulations, and appendices into one document:

Specifically listed genera were prohibited entry from certain countries;

Provisions were specified for post-entry quarantine;

Size-age limitations were specified for admissible plants—stage easiest to inspect and least likely to harbor plant pests;

Mandatory treatments were required for some plants;

Forest trees were only admissible as seeds.

Federal Plant Pest Act of 1957—broadened the definition of plant pest to include any known organism that can directly injure a plant. It allowed for restriction of "pest carriers" such as soil and provided authority to take emergency action against pests and issue regulations to prevent spread of plant pests.

More Recent Laws

Cooperative Forest Assistance Act (1978)—authorized Secretary of Agriculture to protect forests and trees from insects and diseases and provided for coordinated action by Federal and non-Federal land managers to prevent and control forest insects and diseases.

Forest & Rangeland Renewable Resources Research Act (1978)—authorized research and dissemination of information on protecting and managing forests.

Agricultural Quarantine Enforcement Act (1989)—prohibited shipment of quarantined materials via mail.

Plant Protection Act of 2000—replaced the Federal Noxious Weed Act and many other Animal and Plant Health Inspection Service (APHIS) plant protection authorities. The law consolidated and modernized all major statutes pertaining to plant protection and quarantine; permitted APHIS to address all types of weed issues; increased the maximum civil penalty for violation; and authorized APHIS to take both emergency and extraordinary emergency actions to address incursions of noxious weeds.

International Plant Protection Agreements

Greatly increased international traffic in agricultural products following World War II led to sudden and severe outbreaks of plant pests and diseases in many countries and aroused interest in international cooperation on plant quarantines. In 1949, the United Nations–Food and Agriculture Organization (FAO) proposed the establishment of a broad international agreement on plant protection (Ling 1964). The **International Plant Protection Convention** (IPPC) was formally approved by the FAO conference in 1951. Contracting governments agreed to:

Set up satisfactory national quarantine services;

Participate in regional plant protection agencies;

Participate in international control campaigns during emergency outbreaks;

Report on occurrence, outbreak or spread of plant pests.

The IPPC specified responsibility for exporting and importing countries for the prevention of international spread of dangerous pests and diseases. It introduced a standard phytosanitary certificate as proof of duly performed export inspection.

The World Trade Organization's **Sanitary and Phytosanitary (SPS)** agreement was ratified in April, 1996. The agreement requires that countries strive towards greater transparency and scientific basis supporting their actions with trade partners. SPS provides procedures to reveal, challenge, and eliminate unjustified phytosanitary trade barriers.

In November, 1997, the IPPC was revised to comply with SPS the agreement. The revised convention states that phytosanitary measures may be no more stringent than measures applied to same pests within importing country. Safeguards may only be applied to regulated pests: "quarantine pests"—those not yet present in importing country and "regulated non-quarantine pests"—those pests regulated within importing country. International standards are developed under the authority of the IPPC Secretariat.

Current Federal Quarantines

Quarantine 37—Current regulations governing imports of living plants into the US are listed in Title 7 of the Code of Federal Regulations (CFR), part 319.37. Specific plants and plant pests are prohibited, although the list of forest trees and associated pathogens is fairly limited (table 2). Permits are required for importation of specifically listed species from specified countries. The regulations allow imports of artificially dwarfed trees or shrubs; specify special foreign inspection and certification requirements for several types of plants; describe required post entry quarantine procedures; and specify special ports of entry. Plants must be imported without any growing media, with certain

exceptions (for example, *Rhododendron* from Europe with phytosanitary certificate).

Regulations Regarding Importation of Unmanufactured Wood—Following concerns raised about the dangers associated with importation of unmanfactured wood and a series of pest risk assessments completed by the USDA Forest Service (Tkacz 2002), APHIS issued general prohibitions and restrictions on importation of unmanufactured wood (Title 7, CFR, part 319.40). These regulations specify:

General permits for Canada and border states of Mexico;

Procedures for permit applications for wood imports;

Importation and entry requirements for wood imports, including—

Universal requirements,

Softwoods from Asia,

Wood from New Zealand and Chile;

Standards for pest risk assessments, treatments and safeguards.

Current federal domestic quarantines restrict the interstate movement of the following forest pests: gypsy moth, European larch canker, pine shoot beetle, Asian long horned beetle, and sudden oak death (Interim).

European Larch Canker Quarantine—(7 CFR 301.91)—quarantines all parts of larch (*Larix* spp.) including logs, pulpwood, branches, twigs, etc., as regulated articles. Also, any other product, article, or means of conveyance whatsoever, is restricted when it has been determined by an inspector that it presents a risk of spread of the disease. The quarantine designates parts of Hancock, Knox, Lincoln, Waldo, and Washington Counties in Maine as the

Table 2—List of prohibited forest trees and pathogens (7CFR319.37-2)

Prohibited trees	Foreign places from which prohibited	Plant pests existing in the places named and capable of being transported with the prohibited article
Abies spp.	All except Canada	50 or more species of rusts including *Chrysomyxa abietis* (Wallr.) Unger; *Phacidiopycnis pseudotsuga* (M. Wilson) G. Hahn (Douglas-fir canker)
Acer spp.	Japan	*Xanthomonas acemea* (Ogawa) Burk.
	Europe, Japan	Maple mosaic or variegaion diseases
Aesculus spp.	Czechoslovakia, Germany, Romania, United Kingdom	Horsechestnut variegation or yellow mosaic disease
Castanea spp.	All	*Cryphonectria parasitica* (Murrill) Barr (chestnut blight); *Dryocosmus kuriphilus* Yasumatsu (gall wasp)
Cedrus spp.	Europe	*Phacidiopycnis pseudotsuga* (M.Wilson) G. Hahn (Douglas-fir canker); *Fusarium fuliginosporum* Sibilia (Seedling disease)
Eucalyptus spp.	Europe, Sri Lanka, and Uruguay	*Pestalotia disseminata* Thuem. (parasitic leaf fungus)
Juniperus spp.	Austria, Finland, and Romania	*Stigmina deflectans* (Karst) Ellis (Needlecast disease)
	Europe	*Phacidiopycnis pseudotsuga* (M. Wilson) G. Hahn (Douglas-fir canker)
Larix spp.	Provinces of New Brunswick and Nova Scotia in Canada, Europe, and Japan	*Lachnellula willkommii* (R. Harteg) Dennis (European larch canker)
Picea spp.	Europe, Japan, and Siberia	*Chrysomyxa ledi* var. *rhododendri* (de Bary) Saville (Rhododendron-spruce needle rust)
	Europe	*Phacidiopycnis pseudotsuga* (M. Wilson) G. Hahn (Douglas-fir canker)
Pinus spp. (2 or 3-leaved)	Europe and Japan	*Cronartium flaccidium* (Albertini and Schwein.) Wint. (Rust causing serious stunting of hard pines)
	Japan	Gall-forming rust
Populus spp.	Europe	*Xanthomonas populi* Ride (canker)
Pseudolarix spp.	Provinces of New Brunswick and Nova Scotia in Canada, Europe, and Japan	*Lachnellula willkommii* (R. Harteg) Dennis (European larch canker)
Pseudotsuga spp.	Europe	*Phacidiopycnis pseudotsuga* (M. Wilson) G. Hahn (Douglas-fir canker)
Quercus spp.	Japan	*Sterium hiugense* Imazeki (White rot); a gall forming rust
Salix spp.	Germany, Great Britain, and The Netherlands	*Erwinia salicis* (Day) Chester (Watermark disease)
Ulmus spp.	Europe	Elm mottle virus

quarantined area from which movement is restricted.

***Phytophthora ramorum* Interim Quarantine—**(7 CFR 301.92)—quarantines 12 counties in California and portions of one county in Oregon and regulates the interstate movement of regulated and restricted articles: nursery stock of listed species, soil, and any other article deemed to present risk of spreading *P. ramorum* including: bark chips, forest stock, or mulch of listed species from the quarantined area. The rule also specifies conditions of interstate movement of regulated and restricted articles from quarantined areas.

Challenges in Excluding Invasive Forest Pathogens

Although quarantine laws have been in effect since 1912, new forest pathogens, such as the recently discovered *Phytophthora ramorum*, continue to appear and spread in the forests of the US. The current situation is best summarized by a quote that is as relevant today as when it was written:

The maintenance of quarantine regulations is beset with difficulties; therefore in the final analysis quarantines must be regarded as measures of delay rather than measures of exclusion. … The present procedure in this country against introduced diseases is unsound. In general nothing is done until the foreign organism has been introduced and become more or less well established; then an eradication or control campaign is begun (John Shaw Boyce 1961)

The following are some of the major challenges we face in excluding invasive forest pathogens from our forests:

Forest pathogens can be carried on a variety of plant products;

Many diseases cannot be easily detected;

It is practically impossible to adequately inspect all commodities and methods of importation; and

It is difficult to predict which exotic pathogens will be damaging to native ecosystems.

As pointed out by Peace (1962a), there is a strong tendency to base quarantines on specific diseases and on our knowledge of their “potential danger and …distribution”. This approach can be “highly dangerous” (Peace 1962b) because of our lack of knowledge of tree pathogens in many parts of the globe and the possibility that “a known pathogen may behave quite differently when moved to a new environment” (Peace 1962a). Our best strategy to stem the tide of new invasions is to continue and expand efforts to assess and mitigate risks associated with the major pathways for movement of invasive forest pathogens into new ecosystems (Tkacz 2002). By focusing on known organisms traveling via these pathways, we can develop effective mitigation strategies that eliminate both known and similar unknown organisms.

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Pop Quiz on Regulating Forest Pathogens
WIFDWC, August 21, 2003
Grants Pass, Oregon!

Multiple choice—circle all the correct answers

1. NAPPO, EPPO and IPPC are

a. part of the USDA Forest Service.
b. characters in a Marx Brothers movie.
c. international organizations that provide guidance for pest regulation.
d. all of the above
e. none of the above

2. NPAG, PERAL, and CPHST

a. are proposed new TV networks.
b. are headquartered in Raleigh, North Carolina.
c. stands for New Pest Advisory Group, Plant Epidemiology and Risk Analysis Laboratory, and Center for Plant Health Science and Technology
d. all of the above
e. none of the above

3. NAPIS stands for the

a. North Alberta Post Secondary Institutions Society.
b. Navy Air Pollution Source Info System.
c. National Agricultural Pest Information System, the database for the Cooperative Agriculture Pest Survey Program (CAPS). It is part of the Center for Environmental and Regulatory Information Systems at Purdue University.
d. all of the above.
e. none of the above.

4. A quarantine pest is

a. a pest of potential economic importance to the area endangered thereby and not yet present there, or present but not widely distributed and being officially controlled.
b. a pest that has significant impacts on the forest ecosystem.
c. often eradicated with help from the US military.
d. all of the above.
e. none of the above.

5. A pest risk assessment is

a. the determination of whether a pest is a quarantine pest.
b. an evaluation of a pest's introduction potential.
c. a document that covers pathway information, likelihood of introduction, consequences of introduction and estimates unmitigated pest risk potential.
d. all of the above.
e. none of the above.

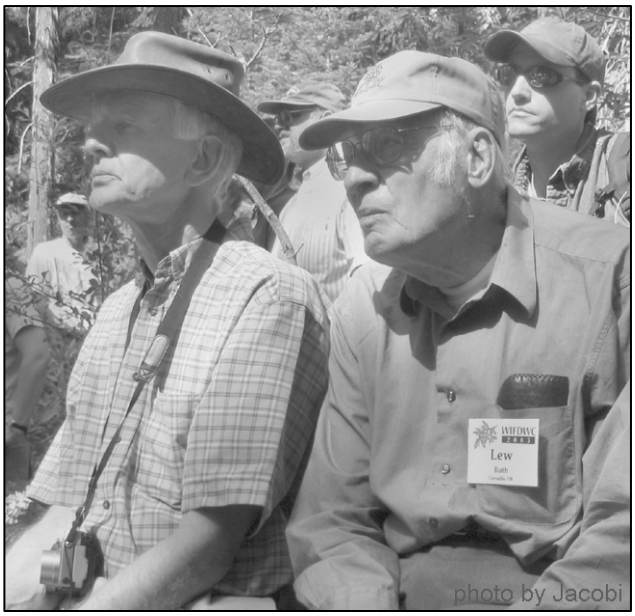
6. The National Plant Board

a. is an organization of the plant pest regulatory agencies of US states.
b. handles agricultural, horticultural and forestry pests.
c. has a Web site at www.aphis.usda.gov/npb/.
d. all of the above.
e. none of the above.

7. Quarantines must be

a. logical.
b. necessary.
c. legally sanctioned.
d. valid.
e. none of the above.

! Answers in footnote for introduction to Panel: Quarantines.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Preventing Invasive Pathogen Threats to Forests—A Sideways Scientific Look

Clive M. Brasier

Abstract—A scientific health check or risk analysis on the current global (WTO/FAO) prevention system is presented. It is concluded that the system, though often well-regulated, is fundamentally flawed, because it is not properly science-based. Possible solutions are presented.

Introduction

The progenitor of this paper was prepared in response to a request from the USDA Forest Service to critique the current global plant health protocols of the World Trade Organization (WTO) and Food and Agriculture Organization (FAO). It was written at a time of increasing international trade in plants and in the context of Sudden Oak Death (SOD) and other invasive pathogens threatening forests and natural ecosystems. The original paper was presented during the Quarantine Panel at the International Symposium on Sudden Oak Death, Monterey, December 2002. An abstract of the original text is on the Symposium website (http://danr.ucop.edu/ihrmp/sodsymp/paper). At Monterey, I pointed out several of my regulatory colleagues from APHIS, UK Plant Health, and Canadian Plant Health were sharing the platform with me and expressed the hope they would still be counted among my friends at the end of my presentation!

The text presented here is a rework of that original paper, and it represents a personal view, based on more than 35 years investigating the behaviour of forest pathogens. It is intended to stimulate and encourage debate in a field where debate is not only seriously lacking but at times is actively suppressed.

The Problem

We have a global prevention system that is

Systematic

International

Well-regulated and well-policed.

Nonetheless, SOD is just another symptom of a historical, and now rapidly growing problem. Although the current system of prevention is often excellently and expertly carried out by regulatory agencies, the system cannot succeed, because it is not yet properly science-based.

Biological Weaknesses

In its current form, the plant health system has a variety of biological weaknesses.

List-dependency—Present protocols are essentially a list-system derived from a 'Noah's Ark' perspective, that is from Linnaean systematics and a morphospecies concept that if a pathogen 'looks like' X, it is X. This perspective is not based upon the species and genotype concepts of modern population biology. Consequently, there are both legal and identification loopholes.

Non-Darwinian—The present protocols have not yet caught up with the science of 1850. Evolutionary theory warns us that greatest threat is from organisms that have evolved in 'other' biogeographical zones, but have not yet 'escaped' and so are still 'waiting' to cause serious damage on new hosts. These threats need to be anticipated. I estimate from recent events that about 80 percent of the pathogens of serious threat to European or U.S. forests are not on the schedules of listed organisms, because they do not cause any notable damage in their natural habitat.

Reactive not proactive—The present schedules tend to cover mainly those pathogens that have already escaped their original evolutionary zone and are causing noticeable damage on new (that is, not co-evolved) hosts. Some examples are Dutch elm disease, North American oak wilt, SOD, chestnut blight, pinewood nematode, and jarrah dieback.

PRA over-dependency—Present protocols, encapsulated by WTA/FAO rules, are based strongly on Pest Risk Analysis (PRA). A PRA is an excellent tool for summarizing current risk information of well-characterized threat organisms that have already escaped. PRAs do not cover the estimated 80 percent of unknown or un-escaped threat organisms.

Structural rigidity—The present system, as an essentially list- not process-driven system, tends to lack the flexibility

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to embrace major new risks related to or even arising directly from the system, including:

Stable-door syndrome. Potential, international, dangerous, and newly identified organisms are often not on plant health schedules nor do they have a PRA. Recognition is too slow. Part of the problem is that the organism may be perceived as benign in the country of first discovery. Another problem is lack of efficient communication between scientists and plant health regulators in country of first discovery and 'would-be preventors' or activists in countries of possible invasion. The new *Phytophthora* of hybrid alder in Europe and the new oak-wilt pathogen in Japan are examples of potentially dangerous organisms that should probably be on many national schedules.

Trojan horse syndrome. Fungistatic compounds are widely used in the international plant and nursery trade. These temporarily mask disease symptoms on exported stock and thereby promote effective spread of exotic pathogens (such as *Phytophthora*).

Typhoid Mary syndrome. Organisms threatening to forest trees are imported on apparently innocuous plant 'carriers'. Consider how SOD may be transported on Rhododendron nursery stock.

Hybridization syndrome. Rapid evolution of new hybrid pathogens is promoted by the present trade practice. For example, at intensive nurseries in Europe, multiple species of exotic pathogens are brought into physical contact and treated with unusual chemicals. These emerging hybrid pathogens are neither detected nor adequately covered by the current system.

In summary—Our present system of prevention tends to respond to the ripples after a splash and not to prevent the splash. The result is that probably only 10 to 20 percent of the exotic pathogens threatening our forests are covered in current quarantine schedules. Basic evolutionary principles are insufficiently encapsulated into the current approach; whereas they should underpin the entire biological rationale. On evolutionary grounds, rapid commercial movement of large numbers of rooted nursery plants and soil between continents and countries probably cannot be carried out with a high degree of long-term safety.

Market Forces and Policy Weaknesses

In addition to the biological weaknesses, there are a number of economic and social weaknesses.

Weakest link—Within multi-state economic political units, such as the European Union (EU), the plant health protection system may tend to operate at the level of the weakest state. This promotes risk and is the very antithesis of how living organisms evolved to restrict the spread of diseases via multiple compartments ('fire walls'). The system itself is non-Darwinian.

Non-Keynesian—The present global system, as operated, is not properly science-based in market economics. It lacks the central, environmental, Keynesian principle that the 'polluter pays'. In this case, neither the trade, the regulator nor the government pay. Therefore, there is no strong feedback loop on the economic system or on the regulatory system to encourage effective bioprotection.

Institutionalisation—A lack of market pressure in favour of progressive and sustainable bioprotection policy can allow governments, trade and regulators to become entrenched and conservative. Therefore, the system itself becomes part of the problem rather than the solution.

Nelsonian approach—Consequences of defensive from regulators over unspoken weaknesses of a system can generate (i) resistance to strategic thinking, (ii) resistance to funding research that expose the weaknesses and (iii) resistance to any policy change. For the regulator, the preferred action is inaction or avoidance because "I see no risks!"

In summary—Market forces don't operate sufficiently and directly on governments, regulators, or commerce to ensure support for effective forest bioprotection measures. The result is that 'nobody' pays for the UK's recent DED disaster or the current alder mortality. In fact, those who ultimately pay are the UK public and its environment.

Diagnosis

A scientific health check indicates the present global plant health system is itself a threat to bioprotection.

Treatment?

Evolutionary biology (science of the 1850s) argues that the present system needs to be better balanced and, in parts, reversed.

Science-process led—The system needs to be based on more environmentally realistic, market-based economics, and sounder bioscience.

Rapid reaction—We need more rapid and comprehensive reporting and assessment of newly identified potential pathogens between the country of discovery and countries at risk.

Proactive approach—A Rio-style agreement among nations should provide for scientific testing of risks posed by a newly identified potential pathogens before they escape. Modern science, technology and infrastructure permit the conduct of these tests either under international co-operation in the geographical centre of origin/discovery or under quarantine conditions in a country at risk.

Fully apply the precautionary principle—The increasing large scale commercial movement of rooted nursery stock between continents and vegetation zones (and subsequent rapid dispersal to other countries) is very high risk. We can significantly reduce the risk from plant imports by:

Regulating plant introductions as we regulate animal introductions.

Importing only meristem cultures or seed, as licensed introductions for propagation (rarely, small quantities of licensed rooted material may be imported for quarantine testing before release).

Encouraging local commercial propagation of forest trees, shrubs and ornamentals.

"Update Noah"—Risk protocols should be extended to fully cover different pathogen genotypes, varieties and unnamed taxa as well as morphospecies.

"Liberté, egalité…"—We should have regular and periodic review of plant health policies and protocols to insure they are processes based on current scientific knowledge, responsive to informed criticism, and open to public debate.

Effective intelligence—Protecting forests and natural ecosystems from invasive pathogens will always be a protracted 'war'. If present invasion trends continue, future losses in terms of species elimination, loss of biodiversity, or ecosystem degradation will be large. Currently, we may be in danger of losing this particular war. Indeed, the increasing damage and threat to forests and natural ecosystems under present international WTO/FAO plant health protocols appears inconsistent with a policy of sustainable forestry and forest protection.

We need to accept that the majority of potential invasive pathogen threats to forests are likely to be currently 'unknown' or so far 'unescaped' organisms. Effective scientific intelligence and scientific insight need to be our first line of defense. We need to keep one at least two steps ahead of the 'enemy'. More globally and locally correct prevention protocols, based on these scientific insights, need to be the second line of defense.

Provision of scientific intelligence in this area is presently very weak. Much of the research currently occurring alongside the formal channels of plant health regulation is too institutionalised and reactive to identify the many risk organisms and processes. Many of these risks are very dangerous but not very obvious. Greater support and encouragement is needed from research funding agencies for intuitive, insightful, and strategic research.


photo by Angwin


photo by Muir

Current Challenges in Forest Pathogen Protection

Faith T. Campbell

Abstract—It is increasingly clear that government programs are failing to curtail introductions of new damaging forest pathogens or to respond effectively to those that were introduced in the past. In some cases, improvements can result from increased funding of exclusion, management, and research programs. In other cases, effective protection depends on amendments to established phytosanitary policies, both national regulations and international standards adopted pursuant to international trade agreements. Forest pathologists and scientific societies to which they belong should exercise their influence to bring about such changes.

Flaws in Phytosanitary Policies

Clive Brasier (these proceedings) has suggested that the international trading system will fail to prevent introductions of additional forest diseases, because it is not based on sound science. Other forest pathologists and mycologists concur. In 1998, the Mycological Society of America (Carroll 1998) said, "We do not believe that pest risk assessments can adequately identify organisms which may cause severe damage in North America." For example, no risk assessment identified the Asian longhorned beetle (*Anoplophora glabripennis* Motschulsky), citrus long-horned beetle (*Anoplophora chinensis* (Forster)), or emerald ash borer (*Agrilus planipennis* Fairmaire) as potentially damaging pests before they were introduced to North America.

Addressing Introductory Pathways for Forest Pathogens

To correct the flaws in the international trading system, scientists should work through their scientific societies to promote conversion to a global phytosanitary system that is pro-active and imposes stringent and effective regulations on the three pathways through which most forest pests enter the North America. Those pathways are imports of wood in the form of logs and lumber and packaging such as crates; imports of woody plants; and incoming ships on which gypsy moths and related species in the Lymantridae have laid eggs.

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Programs Deserving Increased Funds and Staff—I encourage scientific societies to advocate increased funding for such important on-going programs as:

in situ studies of foreign pathogens that might reach North America, especially those that are previously undiscovered or poorly known;

capacity-building to improve trading partners' phytosanitary systems

development and application of improved methods for detecting pathogens and wood-boring insects before they become so widespread that eradication is infeasible;

rapid implementation of eradication programs when a new species of introduced forest insect or pathogen is detected;

development and application of improved control methods; and

development and application of improved methods for measuring ecological impacts of introduced forest insects and pathogens—including ways to describe these impacts in economic terms.

Many of these projects should be undertaken by a revitalized USDA Forest Service research program. However, for the research program to assist in these studies, the severe funding cuts of the past decade and more must be reversed. Funding for forest-protection research fell by 56 percent between 1980 and 2002 (NRC 2002). As a result, the number of entomologists and pathologists on staff declined from about 120 to fewer than 40 (Terry Shaw, USDA Forest Service, personal communication)

The USDA Forest Service germplasm programs also need additional funds to carry out long-term programs to breed resistance into trees that have proved vulnerable to exotic insects and pathogens.

I also suggest that forest scientists press for periodic independent scientific reviews of phytosanitary programs and specific policies.

Reforming National and International Phytosanitary Programs

In addition to educating the Congress and the Parliament about the need to provide sufficient funds to these and similar programs, scientific societies representing forest

pathologists and others should work to reform international and national phytosanitary programs to greatly increase their efficacy in preventing new introductions.

I believe that the best way to prevent introductions is for responsible agencies to adopt phytosanitary regulations that:

aim for the highest level of protection possible;

are effective at achieving that target;

minimize dependence on inspection of incoming commodities at the border; and

are easy to verify.

Curtailing Introductions Hitchhiking on Imported Woody Plants—In working to improve phytosanitary safeguards, the highest priority should be put on imports of living woody plants. These imports pose the greatest risk of introducing new pathogens to North America, as demonstrated by the historic record of probable introductions contained in table 1.

Table 1—Forest pests probably introduced on imported horticultural stock or seeds

Pest	Host(s)
chestnut blight	American chestnut, Allegheny and Ozark chinkapins
Phytophthora cinnamomi	American chestnut, Allegheny and Ozark chinkapins, and a variety of other species in nursery and field situations
white pine blister rust	five-needle (white) pines
Port-Orford-cedar root disease	Port-Orford-cedar
balsam woolly adelgid	balsam and Fraser firs
larch casebearer	eastern and western larches
beech scale	American beech
butternut canker	butternut
dogwood anthracnose	flowering and Pacific dogwoods
sudden oak death	oaks and other hardwood trees and shrubs
citrus longhorned beetle	variety of hardwood species

Furthermore, U.S., Canadian, and North American phytosanitary agencies have all initiated reform of their existing procedures as applied to plant imports, thus presenting timely opportunities for effective scientific input.

The most promising opportunity for achieving more effective phytosanitary measures governing plant imports is a project recently begun by the North American Plant Protection Organization (NAPPO). This effort was initiated by the NAPPO Forestry Panel, including Thomas Hofaker of the USDA Forest Service and Eric Allen of the Canadian Forestry Service. A Propagative Materials Panel, comprised of members and observers from the Forestry, Fruit Tree, and other panels, is now working on a Concept Paper that we hope will lead to drafting of a North American standard.

The premise underlying this project is that current phytosanitary measures applying to the international movement of propagative plant material do not provide adequate protection against the inadvertent introduction of pests of cultivated and wild flora of NAPPO countries. As of August 2003, a draft Concept Paper had been written which reviews the recent history of commerce in propagative plant material, pest introductions associated with these imports, and options for risk mitigation. At present, the majority of group participants favors relying on a clean stock / nursery certification procedure.

The paper will be discussed further at the annual meeting of the NAPPO parties in October 2003. No schedule has yet been put forward for developing an actual standard. Finally, a North American standard adopted by NAPPO would take effect only when implemented by the three member countries (Canada, Mexico, and the United States) through their own rulemakings.

The Canadian Food Inspection Service has already adopted new, more stringent, regulations governing imports of ash, maple and oak. In its current Strategic Plan, the U.S. Department of Agriculture Animal and Plant Health Inspection Service (APHIS) has stated its intention to revise its own regulations governing plant imports (the "Q-37" regulations), but the agency has not moved forward. One reason is that APHIS has not assigned sufficient staff to do the work required.

I concur with Clive Brasier (these proceedings) that the most protective approach is to allow imports only of seed and in vitro cultures. I believe the risk warrants further restrictions, in the form of stringent post-import quarantines to ensure that the imported material is free of pathogens and viruses. Identification, testing, and bulk production of plants to supply the market for horticultural and other purposes would depend on restored domestic capabilities, including USDA plant introduction centers. Neither NAPPO nor national phytosanitary agencies are likely to impose such stringent controls in the absence of strong political pressure supported by scientific data.

Improving Regulations Governing Other Pathways— While forest pathologists and their societies strive to improve phytosanitary safeguards that are applied to imported plants, they should also seek opportunities to tighten controls on other introductory pathways, particularly imported logs, lumber and wood chips (other than trade between Canada and the United States), and imports of wood packaging material.

USDA APHIS adopted regulations governing these imports in 1995, but they have significant weaknesses that make additional introductions via these pathways likely. For example, the 1995 regulations fail to require:

effective mitigation procedures for deep-wood organisms that might reside inside crates, dunnage, and pallets (SWPM) from countries other than China.

phytosanitary treatment of logs and lumber from Mexican states that border the United States.

phytosanitary treatment prior to importation of softwood lumber and railroad ties from most temperate regions long as the importer promises to treat the articles within 30 days.

any phytosanitary treatment other than fumigation and inspection of hardwood logs from tropical and most temperate regions.

effective phytosanitary measures for wood chips (APHIS' own scientists have demonstrated fumigation to be unreliable when the pile of chips is larger than 120 ft^3, that is, about 5 ft by 5 ft by 5 ft (Hobgood and others 1997).

To ensure protection of North American forests, the United States and Canada should require that all logs, lumber and wood chips imported from Mexico or other continents be sterilized by heat treatments (including kiln drying) at sufficient temperatures and duration. This treatment must be conducted in the country of origin rather than in North America.

Correcting Flaws in International Phytosanitary Agreements

One approach to correcting the flaws identified by Brasier (these proceedings) in international phytosanitary procedures might be to seek revisions to the International Plant Protection Convention's (IPPC) International Standard for Phytosanitary Measures (ISPM) #1, Guidelines for Pest Risk Analysis. Pathologists might seek a new definition of "pest", which currently includes 'any species, strain, or biotype'. Or scientists might advocate for a new ISPM dealing specifically with plant pathogens.

Unfortunately, the IPPC and World Trade Organization's Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement) impose severe restraints on a country's ability to prevent re-introductions of organisms that are already established within its border. Preventing continual re-introductions of these established exotic pests and diseases is important to minimize spread of the invader to new areas or infusion of genetic variations that could make the pest or disease more virulent or harder to control.

The SPS Agreement requires any country desiring to prevent further introductions of an exotic pest species already in the country to treat the organisms inside its borders in the same way as it treats their conspecifics at the border. As elaborated by the IPPC, this means that that country may exclude this pest only when the species is not widespread and is the target of an "official control program".

The IPPC's definition of "official control program" requires that the program involve "active enforcement of mandatory phytosanitary regulations...with the objective of eradication or containment…" Under the definition adopted by the IPPC's parent organization, the United Nations Food and Agriculture Organization, an "official" program must be established, authorized, or performed by a nation's phytosanitary agency (for example, APHIS or CFIA). Among forest pathogens that do not qualify as quarantine pests are chestnut blight (*Cryphonectria parasitica* (Murr.) Barr), elm disease (*Ophiostoma ulmi* (Buis.) Narruf. and *O. novo-ulmi* (Brasier)), white pine blister rust (*Cronartium ribicola* J.C. Fisch.), dogwood anthracnose (*Discula destructiva* Redlin), beech bark disease (*Nectria coccinea* var. *faginata* Lohman, A.M. Watson, and Ayers).

The "environment standard" adopted by the IPPC in April 2003 as an amendment to ISPM # 11, Pest Risk Analysis for Quarantine Pests, recognizes that "official control of pests presenting an environmental hazard may involve agencies other than the NPPO [national plant protection organization]." This language does not allow federal agencies other than APHIS to substitute their efforts for those that APHIS might undertake; the NPPO still must supervise and approve such actions for them to qualify as "official control".

Conclusion

Forest pathologists and their scientific societies should redouble efforts to persuade members of the U.S. Congress and Canadian Parliament that introduced tree pathogens pose important threats to the full range of forest values. In consequence, elected legislators should support strengthening of phytosanitary safeguards and provide increased funding, staff, and other resources devoted to minimizing the impacts of those pathogens that have already been introduced.

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Sudden Oak Death—The Year In Review*

Susan Frankel and Pete Angwin

Much has transpired in the world of Sudden Oak Death (SOD) since last September [2001], when WIFDWC met in Monterey. Following is a month-by-month review of major activities and occurrences in California.

A Year of SOD_____

October 2001

The California state legislature passed two SOD bills and provided $3.7 million to the California Department of Forestry and Fire Protection (CDF) for SOD activities.

California coffeeberry, toyon, and California honeysuckle were confirmed as new hosts.

November 2001

University of California researchers received a $1 million grant for SOD research from the Gordon and Betty Moore Foundation (the founder of Intel).

Phytophthora ramorum was confirmed on the campus of the University of California at Berkeley, including in the botanical garden. Plants are shipped worldwide from this location.

December 2001

Senator Barbara Boxer announced $400,000 in agricultural appropriations for SOD research.

January 2002

An arborist from Marin County held a press conference to report that *P. ramorum* was killing redwood trees. In response, University of California researchers revealed that *P. ramorum* had been recovered from redwood sprouts via PCR analysis. The redwood sprouts had been collected on the 2001 WIFDWC field trip!

February 2002

On Valentine's Day, the USDA Animal and Plant Health Inspection Service (APHIS) issued the Federal regulations for *P. ramorum.* The regulations restrict interstate movement of host materials and soil that present a risk for pathogen movement. The Federal quarantine is significantly different from the California state regulation that was implemented in May 2001. Negotiations continued all year to "harmonize" the rules and develop an enforceable quarantine.

Secretary of Agriculture, Ann Veneman approved a $3.5 million SOD proposal from USDA, but the Office of Management and Budget turned down the request.

The USDA Forest Service and CDF teamed up to assemble a $1 million monitoring program for *P. ramorum*. Approximately 14,000 miles of aerial survey were flown, covering 20 million acres. Accelerated measurement of Forest Inventory and Analysis (FIA) plots was done to get a statistically valid assessment of SOD incidence and distribution. A risk-based early detection ground survey was also run in the Sierra Nevada.

The first peer-reviewed scientific paper on *P. ramorum* and extensive oak mortality was published in Plant Disease by Rizzo, Garbelotto, Davidson, Slaughter, and Koike.

March 2002

A Freedom Of Information Act request from the Contra Costa Times to the California Department of Food and Agriculture (CDFA) was the basis for a story that *P. ramorum* was in the Sierra Nevada foothills near Auburn (on the Tahoe National Forest). Maple leaves tested PCR positive. The leaves were collected by Sandy Pursell, a University of California at Berkeley professor working on Pierce's disease on grapes. He was returning from a fishing trip and collected big leaf maple leaves to check for Pierce's disease and gave them to Rizzo to check for *P. ramorum* as well. To date, this report remains unconfirmed, since the pathogen has not been successfully isolated from the area via cultural methods. Symptoms reappeared in the summer of 2002, but numerous isolation attempts from maple and other species have failed.

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May 2002

P. ramorum was confirmed on *Viburnum* at a nursery in England. Since then, over 50 nurseries in the UK have tested positive for the pathogen. We also learned that *P. ramorum* had been isolated from rhododendron in Poland. The discovery was actually made in 2000. The United Kingdom imposed a quarantine, banning imports of plants and wood from parts of United States to prevent spread of SOD.

July 2002

Phytophthora ramorum was confirmed in Humboldt and Contra Costa counties. The Humboldt infestation is in the town of Redway, near the southern edge of the county. The initial report was made by a ranger from Marin county who has a summer home in Redway. It is in the middle of an old-growth redwood forest that is populated with homes. Confirmation was from a symptomatic California bay laurel. SOD was confirmed on the Los Padres National Forest near Big Sur. This was the first confirmed occurrence of SOD on National Forest land. The observed mortality is part of a larger infestation that extends 20 miles from US Highway 1 along the coast up to 3,000 ft in elevation. The area is behind locked gates, but is used to access private properties. Adjacent to the Ventana Wilderness Area, there are no harvests in the area.

August 2002

Steve Tjosvold (University of California Cooperative Extension, Santa Cruz) announced scientific data showing that *P. ramorum* can survive in soil and be transported on tourists' shoes. Tjosvold also showed that *P. ramorum* from the infested soil on tourists' shoes can cause infection in the leaves of California bay laurel seedlings. This work was presented by Jenny Davidson at the American Pathological Society (APS) meetings in Milwaukee.

It was also announced at APS that a second, undescribed *Phytophthora* species (close to *Phytophthora ilicis*) had been occasionally isolated from lethal cankers on tanoak and coast live oak, and from leaves of California bay laurel and tanoak (Hansen, Davidson, Garbelotto, Resser and Rizzo).

The first report of *Phytophthora ramorum* from two nursery shipments of rhododendron in Spain was published in Plant Disease.

September 2002

Dave Rizzo and Matteo Garbelotto confirmed that *Phytophthora ramorum* had been successfully cultured from coast redwood needles, branches and sprouts. They also announced that *P. ramorum* had been isolated from Douglas-fir in one area in Sonoma county, where it was killing tops and branches on small trees under heavily infested California bay laurel. They reported that the pathogen acts like a girdling insect, killing branches, but not whole trees. With the addition of coast redwood and Douglas-fir, there are now 17 known hosts of *P. ramorum*.

In response to the confirmation of redwood and Douglas-fir, APHIS and CDFA added only the affected plant parts of redwood and Douglas-fir to the quarantine, regulating only needles and twigs less than one-inch in diameter. Under this rule, Christmas trees and seedlings are now regulated. Canada added all plant parts to its quarantine, prohibiting shipment of redwood and Douglas-fir logs from California to Canada.

October 2002

Canada eased its regulation of *P. ramorum* by adding a certification option for nursery plants grown in uninfested counties in California. Before this change, the Canadian quarantine covered all of California, prohibiting shipment of all species of plants that were in contact with soil. As a result, all containerized plants were banned and bareroot plants had to be washed to be free of soil prior to shipment. The ban or wash rules are still in effect in the twelve infested counties, but growers in uninfested counties can now have plants and soils tested and certified as free of *P. ramorum*.

2002 By The Numbers

Training

Over the year, the California Oak Mortality Task Force (COMFT) trained over 600 people to be official *P. ramorum* samplers. Thousands of people attended lectures or meetings concerning various aspects of SOD.

Media Coverage

In 2002, over 200 newspaper articles or radio spots on SOD were published or aired worldwide. We have over 160 newspaper articles from this year in our archive. The COMTF website (www.suddenoakdeath.org) had over 60,000 hits. Over 20,000 SOD Pest Alerts have been distributed. Sudden Oak Death has become part of the greater California culture. A rock band named Sudden Oak Death now plays in bars and clubs near Sacramento. Their advertisements feature quotes and sayings from newspaper articles on SOD!

Panel: Mechanisms and Inheritance of Disease Resistance in Forest Trees

Detlev Vogler, Moderator

Program

Resistance to white pine blister rust in North American five-needle pines and *Ribes* and its implications. Paul J. Zambino (presenter) and Geral I. McDonald, Rocky Mountain Research Station, Moscow, ID.

Genetic resistance in Port-Orford-Cedar to the non-native root rot pathogen *Phytophthora lateralis*—2003 update. Richard Sniezko, Dorena Genetics Resource Center, Cottage Grove, OR.

Need for studying both host and pathogen in gene-for-gene systems. Thomas L. Kubisiak (presenter), C. Dana Nelson, Southern Institute of Forest Genetics, Saucier MS and Henry V. Amerson, North Carolina State University, Raleigh, NC.

Inherent and induced resistance to pitch canker in *Pinus radiata*. Thomas R. Gordon (presenter) and Christopher J. Friel, University of California, Davis, CA.

Resistance of pines to dwarf mistletoe. Robert F. Scharpf (retired) Pacific Southwest Research Station, Placerville, CA.

Stefan Zeglen Bruce Hostetler Brennan Ferguson Dave Thomas Terry Shaw
Eric Smith Jon Bell Paul Zambino
Fred Baker
Diane Hildebrand Mee-Sook Kim Jim Hoffman

Resistance to White Pine Blister Rust in North American Five-Needle Pines and *Ribes* and Its Implications

Paul J. Zambino and Geral I. McDonald

Abstract—Both R-gene and multigenic resistance to the introduced pathogen white pine blister rust (*Cronartium ribicola*) appear to be present in five-needle pines and *Ribes* of North America. R-gene resistance that confers immunity is well documented in some populations of three North American species of five-needle pines. Rust virulence factors have overcome two of these R-genes in local areas. Partial resistance in pines is due to the interaction of at least several genes and is sensitive to environment. Resistance in wild *Ribes* species may also be due to a combination of R-gene and multigenic resistance. R-gene resistance might be inferred from 1) dominant resistance in related Eurasian *Ribes* and 2) patterns of susceptibility in cross inoculations of *Ribes* clones with rust from different geographic sources. If R-gene resistance occurs in North American *Ribes*, additional undetected blister rust virulence factors may exist in North America. Either a "cost of virulence" or local adaptation might cause reduced rust aggressiveness in local populations, although local adaptation to increased aggressiveness is also conceivable. Reductions in rust aggressiveness might enhance, and increases in aggressiveness might erode the effectiveness of multigenic resistance in pines and *Ribes*. The future effectiveness of both R-gene and multigenic forms of resistance of both hosts may thus be dependant upon the combined effects of resistance traits artificially deployed or generated through natural selection; local or regional blister rust structure with differences in virulence, aggressiveness, and local adaptation; and diversity of environments. **Keywords—**R-gene, partial resistance, virulence, aggressiveness, mortality analysis

Overview

Blister rust was introduced to North America around the turn of the twentieth century. In concert with changes in forest management that it precipitated, it has been and continues to be the most significant factor in the loss of ecosystems in which five-needle pines have been historically important. This heteroecious rust fungus requires two different hosts (five-needle pines and *Ribes*) to complete its life cycle. The two predominant selectable forms of disease resistance found in plants—R-gene and multigenic resistance—have each been identified in both hosts of the white pine blister rust fungus in North America. This paper reviews the general operation of these mechanisms in better known rust pathosystems, their occurrence in five-needle pines and *Ribes*, and the implications of their presence and utilization toward natural or accelerated development of stable blister rust pathosystems that enhance ecological and timber values. Information on five-needle pine breeding programs that contributed most of our knowledge of blister rust resistance has been separately reviewed (McDonald and others, 2004 in press).

Characteristics of R-gene and Multigenic Resistance

R-gene resistance (often referred to as Major Gene Resistance or MGR) results from specific recognition between host and pathogen, with a resistance allele in the host controlling a specific response to pathogenic strains that carry an allele for avirulence at a virulence/avirulence locus (Takken and Joosten 2000). R-gene resistance is usually fully expressed in even juvenile tissue and is effective across different levels of inoculum and host environments. As predicted by gene-for-gene theory (Flor 1956; 1971), virulent2 races of a pathogen having different or mutant alleles at the avirulence locus overcome R-gene resistance. Thus, R-gene resistance is also known as race-specific resistance, vertical resistance, or gene-for-gene resistance. Resistance in the host and avirulence in the pathogen are both generally considered dominant traits with two readily discerned phenotypes. However, partially dominant expression is known for R-genes in cereal rusts (Roelfs 1988), and unlike the known MGR genes for resistance to blister rust, most R-genes in other rust pathosystems do not confer "all-or-nothing" immunity in resistant interactions, but rather, result in smaller sizes of individual infections than with susceptible interactions (Kolmer 1996; Roelfs 1988). Dominant epistasis is the usual expectation for a resistance phenotype of a host carrying multiple R-genes, so that the amount of rust development is restricted to that allowed by the single most

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2 To avoid confusion, we use the term virulence in this paper in the standard plant pathological context, meaning ability of a strain to avoid detection by a host carrying a specific gene for resistance. We use the term aggressiveness to include quantitative, non-specific traits that affect rates of infection, colonization, host utilization, disease and mortality. In contrast, virulence in the context of human and animal epidemiological literature specifically refers to the ability of a strain of a pathogen to induce mortality.

effective gene. Modifier genes in either the host (for example, suppressers of resistance or of hypersensitive cell death) or the pathogen (suppressers of avirulence) can mask recognition, alter the phenotypes of resistance, and change dominant/recessive behavior in crosses (Kolmer 1996; Roelfs 1988, Williams and others 1992; Yu and others 2001; Zambino and others 2000).

In contrast to R-gene resistance, multigenic resistance is quantitative, with complex genetic inheritance. Furthermore, the threshold level of resistance for effective disease control can vary with host physiology, environment, and aggressiveness of pathogenic strains. Multigenic resistance is least effective in young plants and non-hardened tissues, and is thus subject to breakdown under environments or cultural conditions that increase frequency of exposure to a pathogen or that maintain host tissues in a juvenile, succulent state; but relationships are not always predictable (Poteri and others 1997). This, along with the variation and different levels of expression that is usually found among progeny of test crosses under experimental conditions, can make assigning "resistant" and "susceptible" classes among progeny and assigning levels of resistance to parents difficult and subjective.

As shown in the cereal rust systems where it has been most intensively studied, some forms of multigenic resistance that are known as "adult plant resistance" can decrease initial infection, increase latent period between infection and symptom expression, and reduce rates of colonization and sporulation on non-seedling plants (Broers 1997; van der Gaag and Jacobs 1997). However, it must also be recognized that some forms of cereal adult plant resistance and "partial" resistance are not multigenic; but they are instead attributable to R-genes that have defined races of rust that can overcome their partial effectiveness. Furthermore, and contrary to the usual expectations of dominant epistasis, some such R-genes behave similarly to multigenic resistance when combined in a host, by having earlier and more complete expression of recognition, and thus generate more effective "partial" resistance than with single R-genes (Bender and others 2000; Roelfs 1988).

Differences in aggressiveness of pathogenic strains may also affect the landmarks of infection and rust development, thereby altering the effectiveness of multigenic resistance. "Defeated" R-genes can have a residual effect on resistance of hosts that carry them (Nass and others 1981). Avirulence genes serve various necessary functions in fungi, so a cost of virulence may be associated with overcoming R-gene resistance (Frank 2000; Knogge and Marie 1997). Experimental evidence for a cost of virulence has been provided by Thrall and Burdon (2003) and used to explain why strains of flax rust with unnecessary virulence traits are infrequent in host populations with few R-genes, even when in close proximity with host populations that possess higher numbers of R-genes. A similar cost of virulence may be associated with traits that overcome R-gene resistance of blister rust hosts, whether pine or *Ribes*. If this is true, then the geographic distributions of R-genes and multigenic resistance in pine and *Ribes* hosts and of virulence genes in rust are important for understanding the potential for blister rust to become a "naturalized" part of a stable pathosystem that will allow recovery of ecosystems once dominated by five-needle pines.

R-gene Resistance in Five-needle Pines

R-gene resistance to white pine blister rust is known in four species of five-needle pines and is identified by tan-colored needle spots that result from hypersensitive cell death, an indication of host reaction to the fungus. The *Cr1* locus is found in sugar pine (*Pinus lambertiana*) with an average frequency of the resistant allele at about f = 0.02, but ranging from near zero in the northern part sugar pine's range, to about f = 0.07 in some areas in the southern Sierra (Kinloch 1992). *Cr2* is found in western white pine (*P. monticola*) at very low allelic frequencies, predominantly in the western Cascades where it reaches f = 0.001 and the Sierra where it can reach f = 0.004 to 0.008. *Cr2* is as yet undetected among western white pine seedlings representing Rocky Mountain populations (Kinloch and Dupper 2002; Kinloch and others 1999, 2003). *Cr3* in southwestern white pine (*P. strobiformis*) occurs at frequencies of up to f = 0.05; it is unknown whether the same or a different gene is responsible for the even higher frequencies of hypersensitive reactions reported for some stands of the closely related species, limber pine (*P. flexilis*) (Kinloch and Dupper 2002). R-gene resistance has not yet been detected among families of whitebark pine (*P. albicaulis*) tested for resistance to blister rust (Kegley and others this proceedings, Zambino unpublished data).

Mendelian ratios among seedling progeny expressing versus lacking hypersensitive resistant needle spots identified *Cr1* resistance as the first resistance trait in a forest tree species under the control of a single dominant allele. This locus has also been genetically mapped using molecular markers (Devey and others 1995). An outbreak of rust among large numbers of resistant sugar pine carrying the *Cr1* trait at a location near Happy Camp, CA in the Klamath National Forest in northern California revealed that virulence trait *vCr1* (for virulence to *Cr1*) could become established after periods of intensive regional rust pressure when the "selective" resistant host is present (Kinloch and Comstock 1981). However, spread of *vCr1* has been minimal. Since its detection around two decades ago, strains carrying this virulence have only been found within a few kilometers of two sites—the Happy Camp location and a second site within Mountain Home Demonstration Forest in southern California, over 700 km

distant from the original site (Kinloch and Dupper 1987; Kinloch and Dupper 2002).

In contrast to the sugar pine *Cr1* resistance, selection of material carrying what is now known as *Cr2* resistance in western white pine had initially been based on quantitative comparisons of resistance among families, without recognition of the single-gene nature of the resistance. The outbreak near Champion Mine, OR, of a strain that overcome the resistance of many of the trees that had survived earlier epiphytotics (McDonald and others 1984) prompted new tests of seedling progeny of formerly resistant trees that compared rust inoculum containing versus lacking this so-called "Champion Mine Strain". These inoculations revealed the R-gene resistance now identified as *Cr2* (Kinloch and others 1999) and the rust virulence trait that defeats such resistance, now known as *vCr2*. Kinloch and others (1999) have also suggested a modifier or suppressor of *Cr2* resistance, based on lower than expected numbers of resistant progeny in some white pine crosses. In contrast to *vCr1*, rust strains carrying *vCr2* occur at many locations in the Cascades and the Happy Camp, CA location, where western white pine carrying *Cr2* resistance is found either naturally or in plantations (Sniezko 2002; Sniezko and others 2001).

Consistent with gene-for-gene theory (Flor 1956), the *vCr1* and *vCr2* virulence factors are only effective against *Cr1* and *Cr2* genes, respectively. Resistance of R-gene resistant southwestern white pine (*Cr3*) and limber pine has not as yet been overcome at any known location by a matching virulence trait in the rust (Kinloch and Dupper 2002). Lack of rust races that overcome resistance in southwestern white pine and limber pine has thus far prevented the independence of resistance in these two closely related host species from being tested.

Multigenic Resistance in Five-needle Pines

In contrast to the ease with which R-gene resistant and susceptible pine seedlings can be differentiated by hypersensitive-response versus non-reactive needle infections, other forms of resistance to blister rust can vary greatly in expression. Non-R-gene resistance limits the infection rate and subsequent damage, but resistance is not absolute. For this reason, non-R-gene resistance to blister rust is also variously known as "partial resistance" or "slow rusting resistance". The expression of partial resistance in various plant parts appears to be similar for western white pine, sugar pine, and eastern white pine (*P. strobus*) (Bingham 1983; Hoff and McDonald 1972; Hunt 1997; Kinloch and Byler 1981; Kinloch and Davis 1996; Patton 1972; Patton and Riker 1966, Riker and others 1953). Indications of resistance include—1) lack of or low frequency of spot infections on needles; 2) lack of stem infection after needle infection (either through lack of timely colonization through needles and fascicle bases or through premature shed of needles; McDonald and Hoff 1970); 3) inactivation of infections in stems accompanied by formation of a layer of phellogen known as "corking out" (Struckmeyer and Riker 1951) or "bark reaction" (Hoff 1986); and 4) latent infections, slow canker growth, slower mortality of seedlings with needle or stem infection, and "tolerant" responses to infection due to intermittent or decreased overall rates of colonization (Hoff 1984; Hunt 1997; Kegley and Sniezko 2004). Another type of expression termed "twig blight", has been reported in sugar pine (Kinloch and Davis 1986), may have been observed in eastern white pine by Riker and others (1953), and has also been independently observed in eastern white pine (Zambino, unpublished). In this resistance response, a rapid dieback of infected twigs occurs that includes uninfected tissue on either side of the infection, often extending basipetally to the next node.

Resistance may increase with age of seedlings and trees. With this situation, seedlings that would be killed if inoculated at an early age may have increased resistance and longer survival if inoculation is delayed. The same individuals may tolerate infection or overcome infection as older plants in field plantings (Kinloch and Davis 1986; Patton 1961; Riker and others 1953; Zsuffa 1953). Needle type and physiological age (for example, the simple, first formed "primary" needles versus mature "secondary" needles bound in fascicles) can greatly affect infection (Pierson and Buchanan 1938b). Frequency of substomatal vesicles produced by the rust after inoculation is higher and infections and spots more frequent in primary needles and in young expanding secondary needles than in older secondary needles (Patton 1961, 1967). Additionally, some "select" parent trees of sugar pine (Kinloch and Byler 1981; Kinloch and Davis 1986) and eastern white trees (Patton 1961, 1972; Patton and Riker 1966; Zsuffa 1981) that lack or have undetectable levels of resistance among offspring seedlings have a high degree of resistance among grafted scions exposed to the same inoculation conditions. Such mature clonal parents have been characterized as having "mature tree" or "ontogenic" (developmentally expressed) resistance. A portion of this increased resistance may be due to the act of grafting itself (Patton 1961; Patton and Riker 1966). Also, in the opinion of this paper's authors, this increased resistance could correspond to a slow and progressive increase in the effectiveness of low level resistance, in combination with induced or systemic acquired resistance after exposure to as yet uncharacterized stresses or challenges (for examples see Bonello and others 2001; Enebak and Carey 2000; Evensen and others 2000; Krokene and others 1999, 2000; Pei and others 2003).

Bingham and others (1960, 1969) and Becker and Marsden (1972) have estimated heritability of multigenic resistance

of western white pine, based on the occurrence of active cankers on seedlings two years after inoculation. Although differences were apparent in levels of total and additive heritability, these estimates predicted significant progress in obtaining generally effective levels of resistance after several cycles of selection; and this prediction appeared to be borne out by second generation testing (Hoff and others 1973). Partial ("slow-rusting") resistance in sugar pine (Kinloch and Byler 1981) and eastern white pine has also been suggested to be "polygenic" (Heimberger 1972; Zsuffa 1981).

A critical consideration is whether partial resistance phenotypes represent the expression of different mechanisms under different genetic control or multiple modes of expression for one or more common resistance mechanisms, under common genetic control. Though attempts have been made to determine heritability of some modes of resistance expression as individual traits (for example, Hoff 1986; Hoff and McDonald 1971, 1980; McDonald and Hoff 1970; Yanchuk and others 1994), difficulties are posed by the diverse modes of expression and the lack of families that have only one mode of expression. Hoff, in his 1986 analysis of the inheritance of bark reaction as an independent mechanism, acknowledged this problem: "…a high proportion of the seedlings could not be used for determining bark reaction inheritance because many expressed other resistance phenotypes." He further stated that bark reaction was most prevalent among controlled-cross progenies of parents that exhibited intermediate, rather than high expression of this trait. These results may imply that for these data or families, bark reaction may be an intermediate mode of expression of one or more resistance mechanisms. At higher levels of effectiveness (in other words, more contributing alleles or loci), these resistance mechanisms might prevent infection from reaching the stem in the first place, and might therefore be classified as needle shed or "needle spots only" resistance. That genetic mechanisms underlying bark reaction may also be common to other expressions of resistance is consistent with previous observations. Most highly resistant trees, which were derived from parents with superior field resistance and had been selected as highly resistant progeny after seedling screening, produced progeny with highly expressed resistance mechanisms that were "early-operating" and expressed in needles or needle fascicles (Hoff and others 1973).

Multiple genetic mechanisms may underlie heritable differences for at least some partial resistance phenotypes. For example, Woo and others (2001) found that a portion of western white pine families having low needle-spot numbers had an identifiable physical trait—stomatal openings that were narrower than other low-spotting or normal-spotting families. Other mechanical or physiological traits of western white pine (Pierson and Buchanen 1938b) and eastern white pine (Hirt 1938; Patton 1961) change with needle and seedling age and alter frequencies of needle infections; but whether family differences in resistance can reflect differences in the rate of these maturation phenomena has not been explored (Patton 1967). One post-infection, physiological explanation for differences between eastern white pine families in colonization rate, and perhaps also for numbers of "successful" needle and stem colonization events, has been revealed through histological tests of infected needles. These tests revealed greater production of phenolic compounds after infection, less hyphal colonization, and more hyphal disruption in the resistant families that show fewer/smaller spots and less crown damage than more susceptible families with phenolic compounds present at a greater distance from infection in the resistant families (Jurgens and others 2003).

Race-specific interactions are another mechanism proposed by McDonald and Hoff (1975) to account for low spot number for some selections of western white pine from populations from the Interior Northwest. McDonald and Hoff observed that numbers of red-plus-yellow spots that developed on individual, fully susceptible seedlings after experimental inoculations were approximately equal to the added averages from yellow-spot-only and red-spot-only seedlings. They hypothesized that numbers of red versus yellow susceptible spots in seedlings might actually reflect interactions of two R-genes, each effective against one of two putative, locally common strains of rust that cause red or yellow spots in seedlings susceptible to these strains. Unfortunately, lack of pure rust strains for inoculation, lack of western white pine controlled crosses and selfs as test material, and administrative redirection of the research program precluded further investigation of this hypothesis.

Premature needle-shed resistance may be another resistance phenotype that can result from different genetic mechanisms. This phenotype can occur after hypersensitive response in secondary needles of seedlings that carry R-gene resistance. It has been observed in MGR seedlings of sugar pine and southwestern white pine inoculated with wild type rust, in which case it was often preceded by "bleaching" of infected needles (Kinloch and Zambino, unpublished observations). Needle shed was also reported in western white pine families that can now be assumed to have been carrying Cr2 (McDonald and others 1984). But, needle shed also occurs in non-MGR coastal western white pine (see discussion of "Mechanism X" and "Mechanism Q", Sniezko and Kegley 2003a,b), in inland western white pine populations where R-gene resistance is not known to occur (McDonald and Hoff 1970), and in eastern white pine seedlings of families known to express high levels of multigenic resistance (Zambino, unpublished observations).

Whether R-gene or multigenic resistance is considered, it is impossible to correctly interpret expressions of resistance without accounting for effects of environment, physiological maturity, and virulence or aggressiveness of local rust variants. This need has resulted in regionally-based programs for testing R-gene and multigenic resistance in different environments against different sources of local rust. This approach is further supported by regional differences in—1) pine populations (Conkle 1996; Rehfeldt and others 1984); 2) distributions of R-gene resistance; 3) expression and effectiveness of partial resistance as affected by environment (examples reviewed separately McDonald and others 2004; Hunt 1997); and 4) occurrence of rust variants that defeat R-gene resistance but uncover forms of resistance normally masked in MGR seedlings (Kinloch and Davis 1996). The British Columbia program considers slow canker growth—a direct measure of effective colonization of host by pathogen—to be the most reliable indicator of broadly effective resistance for their high hazard environment (Hunt 1997, 2002), which may suppress the effectiveness of other resistance mechanisms. For example, resistance of western white pine developed for the Inland Northwest in Idaho that relies heavily on needle mechanisms has held up at relatively dry higher elevation inland sites in British Columbia, but not at coastal sites. It is unknown whether resistance breakdown at coastal sites could be attributable to differences in rust aggressiveness (Meagher 1991) or to environmental effects that alter needle retention in a way that could interfere with some needle-based resistance mechanisms. Hirt's (1939) description allows a comparison of this phenomenon in eastern white pine. An isolated breakdown in resistance of inland-derived materials has also occurred at Merry Creek, ID. At this high-hazard, inland site, the types of undergrowth vegetation on plots with the highest rates of infection may indicate the contribution of abundant soil nitrogen as an environmental aspect of resistance breakdown (McDonald and Decker-Robertson 1998), but the local occurrence of more aggressive strains has also been suggested (McDonald and others 1982). Also, although the bark reaction trait has been effectively used in some selection programs, notably in sugar pine in California (Kinloch and Davis 1996) and in western white pine and sugar pine for the Cascades (Sniezko and Kegley 2003b), the bark reaction symptom on western white pine in British Columbia has been suggested to be due in large part to infection or co-infection with other fungi, and not directly related to blister rust resistance (Hunt 1997).

One potential method for assessing levels of multigenic resistance is to monitor percentages of mortality over time in seedling families that have been inoculated at growth stages that ensure nearly 100 percent early stem infection and maintained under constant environment (Zambino and Michler 1999). Mortality can be considered the outcome of a race between a pathogen's colonization/utilization of host tissue versus host response and growth under that environment. Thus, mortality can be used as a surrogate for canker growth to indirectly assess internal colonization, cellular damage, and degree and timing of physiological resistance response. Research is underway at the University of Minnesota to revive operational resistance testing of eastern white pine (see Patton 1972) using similar mortality-monitoring methods.

Figures 1a,b,c (Zambino, unpublished) demonstrate features of mortality curves among eastern white pine seedlings that were heavily inoculated at two growth stages (Zambino and Michler 1999). These growth stages—uniformly mature, primary needles of 20-week seedlings versus needle expansion stage of secondary needles of 2-0 seedlings—have both been considered highly conducive to needle infection (Hansen and Patton 1977) but would differ in the physiological age of basal stem tissue. Within each experiment, time until mortality of the most susceptible individuals of a family provided an indication of that family's levels of multigenic resistance. Expectations that resistance in open-pollinated families had been inherited as a multigenic trait were reinforced by the even slower onset of mortality in seedlings derived from a cross between resistant parents (figure 1a). Differences among experiments in families' time until onset of mortality and final mortality demonstrate that resistance can be altered by host tissues and host physiology, in contrast with immunity-conferring R-gene resistance. Had R-gene resistance been operating, about half the seedlings from an open-pollinated resistant parent heterozygous at one gene or a fourth of seedlings from a resistant parent heterozygous at two loci would have been fully susceptible, and these ratios would have been constant from experiment to experiment.

Others (Heimberger 1972; Patton 1961) have commented that when very young materials are heavily inoculated, even the most resistant material dies, precluding data analysis. However, using mortality analysis, the consistent gap between the onset of mortality in resistant versus susceptible families in figure 1 still allows identification of resistant families (although differentiation still appears best if older seedlings are used). Also the same single-spore-derived rust strain was used throughout this set of experiments. In the future, different rust strains could be used to examine the effect of aggressiveness in the balance between pathogen colonization and host response; and to test whether part of the apparently multigenic resistance

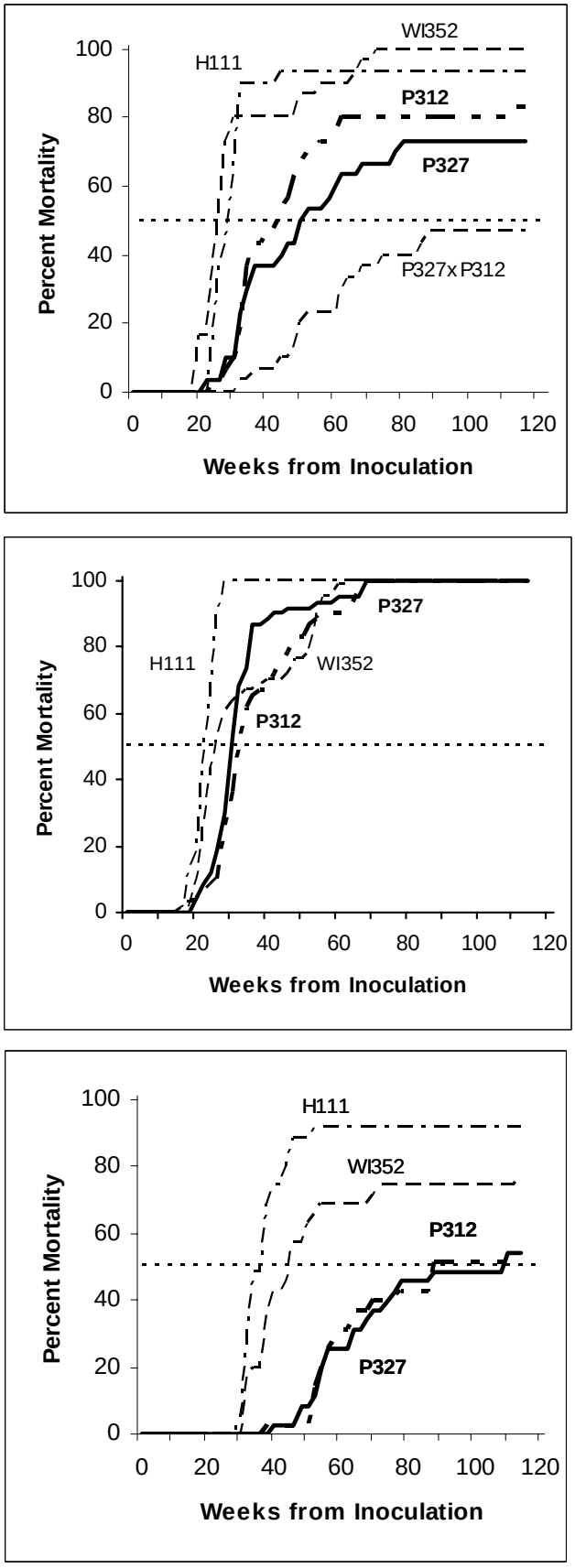

Figure 1—Time until mortality for seedlings of eastern white pine families—a) (top) and b) (middle) subset of families inoculated as 20-week-old seedlings trimmed to 2.0 cm of needle-bearing stem with only primary needles remaining (two experiments, 1 year apart); c) (bottom) subset of families inoculated as 16-month-old, 2-0 seedlings with secondary needles in late expansion. Seedlings were grown in the greenhouse before and after inoculation. All families, including the relatively susceptible H111 and WI352, had been selected as resistant mother-tree candidates from infected stands; mother trees for families P312 and P327 had been selected by Patton. Open-pollinated and controlled-cross (for P327xP312, figure 1a only) seed were provided by the Oconto River Seed Orchard, Nicolet National Forest, USDA Forest Service Region 9 Genetic Program.

could be due to partially effective R-genes that could be partially overcome by as-yet-undetected virulence factors. Effects of temperature and light regimes could also be tested.

R-gene and Multigenic Resistance in *Ribes*

Compared to five-needle pines, much less is conclusively known concerning mechanisms and frequency of rust resistance in wild North American *Ribes* species. Little incentive has existed for making selfs and crosses of *Ribes* hosts or making controlled inoculations using single-spore-derived rust strains comparable to similar work with pines. This is less true for commercial species, such as red and black currants that are valued as fruit and juice crops, and other ornamental species.

A dominant R-gene (*Cr*) occurs in cultivars Consort, Coronet, Crusader, Titania, Tiben, and Tisel of European black currant (*Ribes nigrum*), where it confers immunity without visible hypersensitive-response lesions. The source of resistance was a homozygous resistant clone of *Ribes pauciflorum* var. *ussuriense* (=*R. ussuriense*; Hunter and Davis 1943; review by Brennan 1996)—an eastern Eurasian species that occurs near the presumed center of diversity for rusts of five-needle pines (McDonald and others manuscript under review). Immunity conferred by this gene has held up under field conditions in Europe and North America, as well as in recent in vitro tests against single-spore-derived blister rust strains from regions and hosts across North America (Zambino 2000). However, there are also effective levels of multigenic resistance in some varieties of *R. nigrum,* as indicated by their range in rust resistance from extreme susceptibility to moderate resistance (Brennan 1996) and the large amounts of additive versus. non-additive genetic variance for rust resistance in diallel crosses (Zurawicz and others 1996).

The reported complete immunity of red currant Viking, a Norwegian cultivar derived from a cross of *R. petraeum* Wulf. x *R. rubrum* L and also known as Røt Hollandsk

Druerips and Holländische Rote (Hahn 1949) may be due to another single dominant gene, in this case from *R. petraeum*. Hahn (1938, 1949) reviewed literature demonstrating the complete lack of infection of this cultivar in extensive field plantings in Scandinavia, Holland, Germany, and Switzerland, and in field trials for susceptibility in Germany (Munich), Great Britain, Canada (Ottawa), and the United States (Maine, New Hampshire, Massachusetts, Connecticut, New York, and Oregon). Because susceptible seedlings have rarely been recovered among even open-pollinated seed derived from this cultivar, Hahn (1938) concluded that the cultivar was homozygous for resistance. Typical symptoms after inoculation in the field (Hahn 1936) or greenhouse (Hahn 1935) are complete immunity, or at most, a watery chlorosis followed by a hypersensitive fleck in very young leaves (Anderson 1939; Hahn 1943). More recently, in vitro and greenhouse inoculation tests by Zambino (2000) of available North American Viking clones produced sporulating infections delimited by slow necrosis at major veins that had been described as typical for red currant cultivars (Hahn 1939; Hennings 1902; Spaulding 1922). Moreover, leaf lobe shape and curvature of this material resembled commercial *R. sativum* red currents but differed from descriptions and photographs from published reports of the hybrid cultivar Viking (Anderson 1939; Hahn 1943). Thus, Zambino (2000) concluded that the test materials might have been misidentified at some time during the six decades of propagation since the last reported resistance tests of this cultivar. Hummer and Picton (2002) have also detected rare infections on artificially inoculated leaves, although infection has not been detected under field conditions at two locations in the western United States (Hummer 2000).

Some cultivars of the North American species *R. aureum*—the "golden" or "Colorado" currant prized as a landscape ornamental—have also been reported to have R-gene resistance (Hunter and Davis 1943). A staminate clone of *R. alpinum* was also reported to be immune (Hahn 1939). In tests that included different rust sources in conjunction with horticultural and wild North American *Ribes*, Anderson and French (1955) also identified a clonal line of *R. hirtellum* that produced large necrotic leaf spots within two weeks of inoculation with some rust isolates, but chlorotic lesions with others, suggesting a differential interaction with rust source. All five California and Oregon rust isolates from sugar pine produced necrotic lesions, whereas five rust isolates from eastern white pine from locations from New York to Minnesota and an isolate from western white pine from Oregon produced chlorotic lesions.

Resistance of Wild North American *Ribes*

Most of the historical record regarding susceptibility of North American *Ribes* that occur in natural habitats has attempted to rank the relative contribution of different species to spread or intensification of the blister rust epidemic on pine. Much less emphasis has been placed on interactions between individual *Ribes* genotypes (bushes and clonally propagated ramets) and individual rust isolates that would be useful for identifying mechanisms of resistance. An early study by Hahn (1928) identified differences in susceptibility among 21 North American *Ribes* species to both the exotic white pine blister rust and the North American pinyon blister rust (*C. occidentale*). Greenhouse-grown whole plants were separately inoculated with these rust species using mixtures of urediniospores representing local collections. *R. triste* was the only species that appeared to be immune to white pine blister rust, whereas other *Ribes* supported rust development to different degrees. Hahn identified some immune plants in some species and differences between host species in their reaction to the two pathogens. However, he suggested that no significant within-species differences were evident among the 2 to 37 plants chosen to represent each *Ribes* species in their reaction to each of the pathogens. Studies by both Kimmey (1938) and Mielke and others (1937) that relied upon artificial inoculations of clones under field conditions also regularly identified immune clones within otherwise susceptible species. The highest proportion of apparently immune clones reported by Mielke and others (1937) was about 15 percent, in a North American black currant, *R. hudsonianum* var. *petiolare*. A small number of additional clones of *R. hudsonianum* var. *petiolare* and other North American species were noted as having high resistance, but lacking immunity (Mielke and others 1937).

It is noteworthy that among western North American species, such apparently high numbers of immune or nearly-immune clones occurred in *R. hudsonianum* var. *petiolare*, although *R. hudsonianum* var. *petiolare* has also shown the greatest potential ability to support urediniospore and teliospore production among western species (Hahn 1928; Kimmey 1938; Mielke and others 1937). The generally high susceptibility of *R. hudsonianum* var. *petiolare* is understandable, as it is very closely related to the highly susceptible (Kimmey 1938) European black currant, *R. nigrum*. These two species were significant contributors to early spread of blister rust in the West (Lachmund 1934; Mielke and others 1937). These species were also interfertile in controlled crosses (Jandzewski 1907; reviewed in Keep 1962) and were indistinguishable in a study of currant and gooseberry species based on chloroplast DNA restriction sites (Messinger and others 1999). Differences between the most- and least-heavily infected bushes were less dramatic in other species, but relatively resistant bushes with trace infection were also found in *R. viscosissimum* and *R. inerme* (Mielke and others 1937).

The Need to Identify Interactions Among *Ribes* Clones and Rust Sources by Cross Inoculations Under Constant Environment

Both immune and non-immune expressions of resistance to blister rust appear to occur in North American *Ribes*, yet as with pines being examined for multigenic resistance, environment and developmental stage can also greatly affect the expression of resistance. Leaves are immune when immature (Harvey 1972; Spaulding 1922), become highly susceptible after full expansion, and then decrease in receptivity to infection with age (Lachmund 1934; Pierson and Buchanan 1938a; Spaulding 1922). Susceptibility of aging leaves can be extended if shoot growth is interrupted by induced dormancy (Harvey 1972). Previous work has also demonstrated that infection was greater in the less "hardened" plants that grow in full or partial shade than in open-grown plants in full sun (Hahn 1928, Kimmey 1938, Mielke and others 1937). Type of inoculum also has an effect. Significant infection by urediniospores can occur in *Ribes* leaves that would be 1 to 2 weeks too mature for aeciospore-initiated infection (Pierson and Buchanen 1938a). McDonald and Andrews (1981) also noted that disks from 14- to 21-day-old leaves of *R. hudsonianum* var. *petiolare* will develop fewer infections after inoculation with aeciospores than with urediniospores. These results could be due to leaf maturation interfering with infections that colonize leaf tissue slowly. Aeciospores usually divide their stored resources among multiple germ tubes, leaving fewer resources for the successful penetrating germ tube, in contrast with urediniospores which produce a single, stout, and more rapidly growing germ tube. Also, time from inoculation until uredinia appear is usually about 2 to 3 days slower with aeciospore inoculum than with urediniospore inoculum (Zambino, unpublished observation).

The influences of environment and mixed rust strains preclude the use of previously reported symptom severity for differentiating R-gene resistance from multigenic resistance in *Ribes*. Two independent tests are needed to identify types of resistance—1) the reaction of host tissues of different clonally propagated individuals at their peak of susceptibility should be identified in cross-inoculation tests against diverse pure-genotype rust sources under a common environment; and 2) monogenic versus oligo- or multigenic patterns of inheritance should be determined using *Ribes* progeny tests. Three potential types of resistant interactions might be recognized from cross-inoculation tests—1) reversals in relative susceptibility of two clones when exposed to two different strains of rust may indicate R-genes interacting with rust races carrying different virulence/avirulence traits; 2) when resistance increases or decreases due to rust strain occur among clones but the relative ranking of clones is preserved for a quantitative attribute of infection (for example, number of infections, latent period to spore production, or amount of urediniospore or teliospore production), then hosts may differ in background levels of multigenic resistance and may be responding to differences in aggressiveness or overall fitness of rust strains; and 3) instead of race-specific resistance, apparent immunity of some *Ribes* clones to a subset of rust strains could indicate an effectiveness threshold for a quantitative resistance trait interacting with levels of aggressiveness. In this situation, the aggressiveness or colonization ability of a rust strain may be insufficient to overcome the level of partial resistance of the host under the test environment. Diagnosis of this third type of resistant interaction may be aided by determining whether the non-infecting strain has less than average development on other *Ribes*.

Although inheritance of rust resistance is not well studied for wild *Ribes*, the cross-inoculation approach has been employed in attempts to identify/detect differences among *Ribes* clones and rust races. Anderson and French (1955) tested resistance of unreported numbers of clones of several North American *Ribes* species using blister rust collections from different hosts and geographical regions. Although these rust collections cannot be considered as single genotypes, differential interactions reflecting differences in rust sources were reported to occur among some clones of *R. cynosbati* and *R. lacustre*. However, *R. virburnifolium* was uniformly immune, whereas *R. hudsonianum* var. *petiolare, R. cereum*, and *R. americanum* were uniformly susceptible. Of *Ribes* species that displayed variable levels of rust resistance, resistance of even the best clones fell short of complete immunity. Reversals in resistance among some of Anderson and French's (1955) clones when exposed to different rust collections perhaps indicated R-genes; the occasional infections could have been due to R-genes that confer less than full immunity or to a mixture of strains in the inoculum.

All three types of resistant interactions may be represented in cross-inoculation studies using North American *Ribes*. In an attempt to identify *Ribes* host resistance, rust aggressiveness, and environmental factors important for understanding the behavior of rust epidemics, a method was developed by which leaf disks of different clones at physiologically susceptible stages could be uniformly inoculated with rust of different sources and spore types, and held in different environments while tracking development/spore production of uredinia and telia (McDonald and Andrews 1980, 1981, 1982). Rust aeciospores collected from white pine from four locations (Champion Mine, OR; Still Creek, OR; Priest River, ID; and Merry Creek, ID) was used to inoculate 21- to 24-day-old leaf disks of 50 clones representing four *Ribes* species prevalent in the Northwest (*R. hudsonianum, R. inerme, R. lacustre*, and *R. viscosissimum*; McDonald 2000; a discussion of the relative importance of these species in blister rust epidemiology is provided by Kimmey 1938). Infection parameters examined included numbers of

infections per unit leaf area, infection efficiency per spore, and time until appearance of uredinial pustules.

Previous analysis of the data either averaged over clones (McDonald 2000) or illustrated strain-specific and location-specific resistant interactions between a variety of geographic sources of rust and clones of *R. hudsonianum* var. *petiolare* from one location (McDonald 1996). Results indicated that 1) the Champion Mine aeciospores ranked at or near the bottom for infection efficiency for *Ribes* species collected from all sites; 2) the Merry Creek inoculum was among the most effective inoculum sources for *Ribes* species and clones from Idaho, but ranked low in effectiveness as an inoculum source for *Ribes* from the Cascades; and 3) some clones of *R. hudsonianum* var. *petiolare* and *R. viscosissimum* were immune to some or all geographic collections of rust (McDonald 2000).

An illustrative subset of the original data is presented in figure 2 in reaction norm format, which displays the "response" of rust source (in terms of infection density and incubation period until urediniospores appear, respectively) under the "environments" posed by clones of two *Ribes* species (*R. hudsonianum* var. *petiolare* from Merry Creek, and *R. viscosissimum* from Priest River). The best case for a source-specific resistant interaction may be found in figure 2a. The Still Creek inoculum had been second only to the Merry Creek inoculum in infection levels on Merry Creek clones H5, H7, and H8 of *R. hudsonianum* var. *petiolare*, but had dramatically lower infection on clone H1. Thus, the stark contrast on clone H1 between the high levels of infection from Merry Creek and Priest River inocula versus the low level of infection from Still Creek and Champion Mine inocula might represent differences in resistance to strains of the rust carrying different avirulence factors. In contrast, the "immunity" of clone H2 against all rust sources provides little information to allow addition interpretation. However, the relative order of infectivity or aggressiveness (Merry Creek > Still Creek > Priest River > Champion Mine) found in clones H5 and H8 had been preserved in the "nearly immune" clone H7. Thus, it cannot be determined whether clone H2 represents a generally effective R-gene, or alternatively, a somewhat higher level of multigenic resistance than was observed in clone H7. The absence of infection of Priest River clone V1 of *Ribes viscosissimum* by selected rust sources may represent a high resistance threshold in this *Ribes* clone (figure 2b). In this example, time until urediniospore appearance of even the most successful rust sources was relatively slow, suggesting that Champion Mine and Priest River inoculum sources may have lacked the aggressiveness to colonize/develop before leaf disks reached maturation levels that prevented rust development.

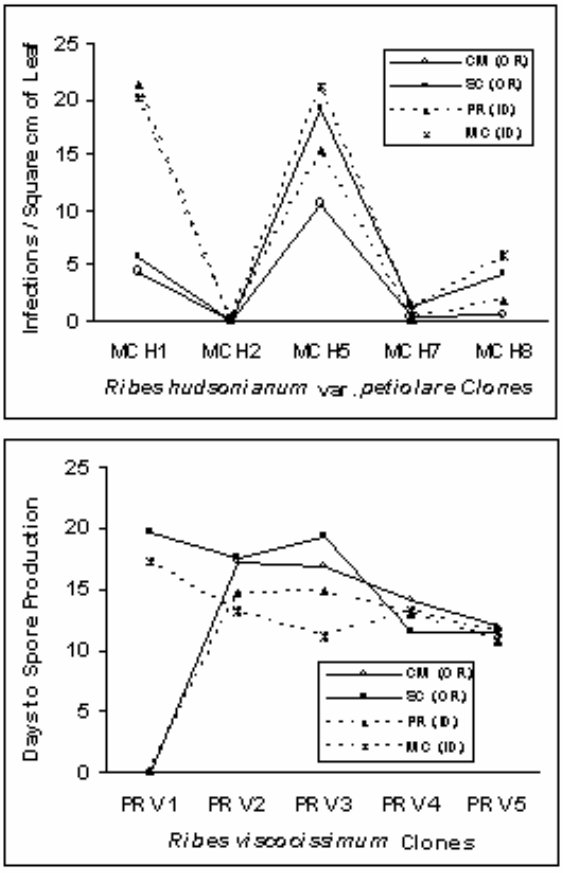

Figure 2—Examples of three types of interactions among Ribes clones in cross inoculation studies with rust aeciospores from Champion Mine, OR (CM), Still Creek, OR (SC), Priest River, ID (PR), and Merry Creek, ID. a) (top) Infections per square cm that developed on inoculated, detached leaf disks (McDonald and Andrews 1981, 1982, 1982) of *Ribes hudsonianum* var. *petiolare* clones originating from Merry Creek, ID (McDonald, 1996). b) (bottom) Days until urediniospores were produced on inoculated, detached leaf disks of clones of *Ribes viscosissimum* originating from Priest River, ID. (Lack of infection is indicated by data points at "0 days"; McDonald, unpublished).

Implications of Aggressiveness on Effectiveness of Multigenic Pine Resistance

As well as identifying differences in resistance among *Ribes* clones, results of Anderson and French (1955), McDonald and Andrews (1982), and McDonald (1996) imply regional differences in rust aggressiveness or virulence to *Ribes*. To assess how these differences may influence the stability and usefulness of multigenic resistance in pine, one needs to determine—1) how the tentatively identified differences in aggressiveness on *Ribes* relate to differences in aggressiveness on pine, and vise

versa; 2) whether differences in aggressiveness are due to costs of virulence factors that overcome R-genes in pine or *Ribes*; 3) whether changes in aggressiveness are related to local adaptation; 4) whether the potential for future changes in the rust pathogen will generally favor increased or decreased aggressiveness; and 5) the magnitude of differences in pine infection and mortality that might be attributed to differences in rust aggressiveness.

Little experimental evidence has been obtained regarding the interaction of host resistance in five-needle pine and *Ribes* with rust aggressiveness. The Champion Mine inoculum was of low aggressiveness on *Ribes* as determined by McDonald and others (1982). Subsequently, McDonald and others (1984) reported similar inoculum to have a longer incubation period on susceptible pine. These results suggest that lower aggressiveness on one host may correspond to some aspects of lower aggressiveness on another. Because the 1984 study was completed before the identification of the *vCr2* virulence factor and *Cr2*-resistance-carrying western white pine families, inoculum for neither study had been specifically tested for *vCr2*, although aeciospores had been obtained from trees that may be presumed to carry *Cr2*. Kinloch and others (1999) had compared Happy Camp inoculum (which can be presumed to have a high frequency of *vCr1* in addition to *vCr2*) to other sources of *vCr2* for their ability to infect families of western white pine seedlings carrying *Cr2*. Their finding that Happy Camp inoculum produced the fewest spots and fewest infected seedlings of any inoculum that carried *vCr2* was attributed to the low *vCr2* frequency at Happy Camp at the time of sampling. Time to appearance of symptoms was not reported. *Cr2* western white pine and *Cr1* sugar pine are the only pine hosts at the Happy Camp inoculum source location and occur as separate blocks in close proximity, although *Cr1* sugar pine is by far the predominant pine host represented. The low *Cr2* frequency despite the close proximity of *Cr1* and *Cr2* hosts may be indirect evidence of selection against strains carrying *vCr1* and *vCr2* in combination. As previously mentioned, *vCr1* is of limited geographic distribution (Kinloch and Dupper 1987; Kinloch and Dupper 2002). This limited distribution has been attributed to white pine blister rust in western North America being a genetically fragmented meta-population (Kinloch and others 1998); however, costs of virulence on environmental fitness or aggressiveness on one or both hosts should also be considered. Although Zambino (2002) was able to infect *R. nigrum* cultivars that lacked the *Ribes CR*-gene with strains derived from aeciospores produced on *Cr*-gene carrying sugar pine and western white pine from Happy Camp and Champion mine, respectively, infections with such strains frequently appeared to develop slower and produce fewer uredinia compared to other North American sources of blister rust inoculum (Zambino, unpublished). To date, no tests have compared aspects of infection and colonization of single-genotype rust strains on pine versus *Ribes* hosts; however, such tests are being developed (Zambino, unpublished).

Models and observations based on other host–parasite interactions may provide insights into changes that might occur in white pine blister rust aggressiveness at some localities. Zimmer (2003) stated that according to observations on animal and human pathogens, "…a pathogen can evolve to become harmless, more deadly, or anything in between, depending on the forces guiding natural selection." Examples of evolution from high to lower aggressiveness are believed to occur most frequently when a pathogen is not in equilibrium with its host, such as when an imported pathogen encounters a new, highly susceptible population. To understand this trend, one must consider a pathogen's evolutionary tradeoffs between "…how fast a pathogen breeds and how easily it can infect new hosts" (Zimmer 2003) and the tradeoff between longevity and fecundity (Frank 1996). Interacting factors include—1) the population structure of both host and parasite in relation to their capacity for long or short range dissemination (Haraguchi and Sasaki 2000); 2) the association between aggressiveness and transmission success (Levin 1996); 3) the susceptibility of the host population and its density (Day 2003; Levin 1996); 4) whether infection of an individual characteristically occurs by a single infection with a single strain, by co-infection by several strains, or by super-infection in which a more aggressive strain can replace or eliminate a less aggressive strain on a host (Mosquera and Adler 1998); and 5) whether infections are lethal or sublethal (Schjorring and Koella 2002). Our interpretation of models of aggressiveness evolution holds that in general, high virulence will be favored when a highly susceptible population is being colonized by an efficiently disseminated pathogen; when aggressiveness translates into abundant short-term fecundity (given that abundant susceptible hosts are present); and when infection is either by single infection or by super-infection that results in rapid mortality. Lower aggressiveness can evolve when host density is low and/or intermittent and infection uncertain, so that pathogen longevity is favored over fecundity; where population structure exists in association with more localized dissemination of host and pathogen; where either co-infection or induced resistance to subsequent infection can occur; and on hosts where infections can be sublethal allowing greater longevity of individual infections.

Characteristics of the blister rust pathogen and five-needle pines during the blister rust epidemic in North America suggest that high pathogen aggressiveness and fecundity would have been favored early in the epidemic. However, rust aggressiveness/fecundity would be expected to decrease over time, eventually reaching levels that are selectively responsive to local host population structure, host density, levels and types of resistance, and

environmental signals. Blister rust that reached North America from Europe was probably of high aggressiveness, having spread and intensified rapidly in Europe on eastern white pine in nurseries and small, scattered plantations and on the even more widely planted European black currant (Moir 1924). Aggressiveness and high fecundity would have been initially favored upon initial introduction to the extensive stands of susceptible pines in North America. Mortality was high—over 95 percent in some stands of eastern and western white pine—so that uncolonized hosts would eventually have become scarce. Intermittent regeneration of new five-needle pine cohorts from survivors might have favored those slow-growing cankers that had persisted longer due to higher host resistance/lower pathogen aggressiveness, compared to cankers caused by strains with high fecundity coupled with aggressiveness or lethality to hosts. This process would be more pronounced if hosts infected with less aggressive strains could develop induced or systemic acquired resistance that would limit subsequent infection by aggressive rust strains that cause rapid host mortality.

Is induced resistance to blister rust a feature of North American five-needle pines? Infection records were recently re-examined from diverse stands that developed in northern Idaho within a generation after the arrival of blister rust. This analysis revealed that a significant proportion of the infected trees developed only one or a few cankers (McDonald and others in press). Induced resistance may also operate in very young seedlings. Kinloch and Comstock's (1981) original report of the *vCr1* virulent race described an experiment where sugar pine seedlings carrying *Cr1* were inoculated with wild-type rust (lacking *vCr1* virulence) and subsequently developed hypersensitive resistant spots. These inoculated plants were held under 24-hr photoperiods until secondary needles developed, and were then inoculated a second time, with rust carrying *vCr1*. Despite the normal increase in susceptibility that continuous illumination usually causes in sugar pine seedlings (Kinloch 1980), these previously exposed seedlings developed fewer susceptible needle spots than did susceptible seedlings that had not been previously exposed. This response may represent an example of induced or systemic acquired resistance to white pine blister rust, thereby supporting the premise that rust strains with low aggressiveness could become established and persist in forest trees.

If reduction in rust aggressiveness does occur, what would be the effect on pine resistance developed under either natural or artificial selection? From the outbreaks that occurred in established stands of *Cr1* sugar pine and *Cr2* western white pine, it appears that costs of virulence are not of sufficient magnitude to prevent trees with MGR as a sole source of resistance from rapidly succumbing when challenged with *vCr1*- or *vCr2*-carrying strains, respectively (Kinloch and Comstock 1981; Kinloch and Dupper 1987; Kinloch and others 1999; McDonald and others 1984). However, one would expect that lower aggressiveness would affect the balance between tissue colonization and non-specific host response, and thereby enhance the effectiveness of partial resistance. Even small changes in effectiveness of partial resistance may be critical for pines with marginal resistance, when grown under moderate rust hazard. In fact, we may be reaping the benefits of reduced aggressiveness already—the breakdown of *Cr1* resistance in sugar pine at the Happy Camp location has enabled seedlings carrying *Cr1* to be tested for additional resistant traits that may be oligo- or multigenic (Kinloch and Davis 1996) and similar programs are in place for testing the partial resistance of western white pine carrying *Cr2* by exposing them to rust strains carrying *vCr2* during screening or under field conditions (Sniezko and Kegley 2003b, Sniezko and others 2004). It remains undetermined whether the apparent levels of multigenic resistance among seedlings selected for their *Cr* resistance have been enhanced by lower aggressiveness of *vCr1*- and *vCr2*-carrying strains.

It is important to note that one must never assume that lower aggressiveness will be the rule, disregarding the specificity that rust may develop during adaptation to locality. Aggressiveness may even be indirectly affected as rust adapts its life cycle to optimize its infection of pine and *Ribes* hosts under specific climates and microclimates. McDonald (1996) has suggested that rust infection of pine in some areas of the southern Sierra may be more likely to occur during spring weather patterns than in fall. This adjustment would necessitate early production of telia, which has been considered a hallmark of host stress, low pathogen aggressiveness, and/or moderate plant resistance in many rust pathosystems (Waters 1928). Local fluctuations in *Ribes* and pine hosts following thinning, burning, regeneration, restoration efforts, and fluctuation in types and levels of resistance might also ensure that aggressiveness will continue to fluctuate, depending on the local situation. For restoration, use of R-gene plus multigenic resistance may be favored in those areas where both occur. In theoretical models, greater non-specific resistance stabilizes the gene frequency dynamics of specific defenses (Frank 2000). However, where remnant stands occur, one may expect that local adaptation to local rust and the occurrence of local population structure would support utilizing these remaining trees for regeneration whenever possible. Thus, utilization, development, and deployment of pine resistance will remain an activity that will be aided by knowledge of types and effectiveness of resistance in pine populations, for management by regeneration and restoration efforts at the local level.

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Genetic Resistance in Port-Orford-Cedar to the Non-native Root Rot Pathogen *Phytophthora lateralis*—2003 Update

Richard A. Sniezko

Abstract—Genetic resistance to *Phytophthora lateralis* in Port-Orford-cedar (POC) provides a management tool for restoration and reforestation in areas heavily impacted by this non-native pathogen. Since it began in 1997, an operational program to develop resistant populations of POC has made rapid progress in some breeding zones. Seed is now available, but additional selections from natural stands are needed for other breeding zones. In greenhouse tests, the best resistant seedling families have 50 to 100 percent survival versus no survival for the most susceptible families. Families also vary in time to mortality in greenhouse testing. In young field validation trials, resistant families are showing good potential, but more time is needed for assessing durability of resistance. A few field selections from 1989 and 1990 confirmed as resistant in greenhouse testing have been revisited and continue to thrive. Investigations into the nature of the resistance mechanisms and their inheritance are underway. Future needs of the program are discussed.

Introduction

Since 1952, a non-native, invasive pathogen, *Phytophthora lateralis*, has been spreading throughout the native range of Port-Orford-cedar (*Chamaecyparis lawsoniana*). This root rot kills seedlings as well as large trees, particularly in riparian areas (Casavan and others 2003; USDI-BLM and USDA-FS 2004). Some management tools are available for slowing the spread of *P. lateralis* (Goheen and others 2000; Goheen and others 2003a; USDI-BLM and USDA-FS 2004). Use of genetic resistance may be key in efforts to restore heavily impacted areas.

The first indication in Port-Orford-cedar (POC) of genetic resistance to *P. lateralis* was noted in the 1980s (Hansen and others 1989; Sniezko and others 2003c). Since then, greenhouse and field studies have confirmed genetic variation in susceptibility to *P. lateralis*, the low frequency of resistance in natural POC populations, and some understanding of the inheritance of resistance (Sniezko and Hansen 2003; Sniezko and others 1996; Sniezko and others 2000; Sniezko and others 2003a; Sniezko and others 2003b; Sniezko and others 2003c; Sniezko and others this proceedings). An examination of some of the underlying mechanisms of resistance is also underway (Eun-Sung Oh, personal communication)

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This paper briefly summarizes my presentation from the genetic resistance panel at this conference (with some updates). It covers aspects of the operational breeding program, what is known about the resistance, and questions for the future. Further information is available from cited papers and at www.fs.fed.us/r6/dorena.

Resistance Program Overview

In 1997, the USDA Forest Service (FS) and USDI Bureau of Land Management (BLM), in cooperation with Oregon State University (OSU), began an operational program to develop resistant populations of POC for use by land managers. Breeding zones have been established based upon common garden tests (Kitzmiller and Sniezko 2000; Kitzmiller and others 2003; Jim Hamlin and Jay Kitzmiller, personal communications) (see figure 1 for breeding blocks). Elevation bands within breeding blocks have been used to define breeding zones.

The first stage of the operational resistance program involved selecting thousands of candidate trees to test for resistance. Most of the earliest selections were made in Oregon in high disease areas. Parents confirmed as resistant are used to establish seed orchards to provide genetically diverse seedling populations with resistance to *P. lateralis*. A major emphasis of the program is to strive to maintain broad genetic diversity in restoration populations; this differs fundamentally from many horticulture and crop breeding endeavors (in which crop uniformity is often paramount). It is unlikely there is sufficient resistant POC in the highest hazard areas to provide large trees for the future without restricting the genetic variation. The resistance program brings together naturally occurring resistant trees from the same breeding zones.

Since 1997, over 10,000 field selections have been tested using the stem dip method (see Bower and others 2000 for summary of the first 7000 selections). The top ten percent of these candidates (approximately) undergo a second phase of testing using rooted cuttings (root dip test). Approximately 500 of the parents have been through this second phase of testing, and about 100 of these appear to be resistant (100 percent survival); an additional 160 some parents show about 50 to 90 percent survival. Further investigation of these parents is underway. Most of the early selections have been in the most northwestern breeding block (BB1 in figure 1).

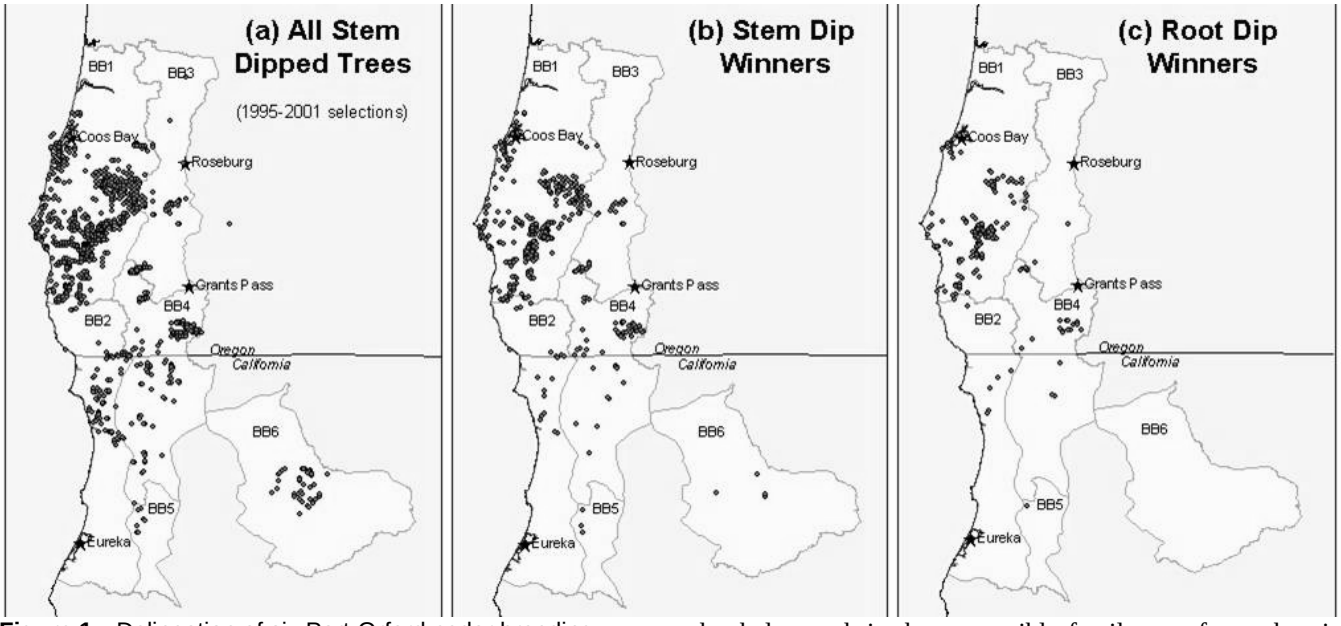

Figure 1—Delineation of six Port-Orford-cedar breeding blocks and locations of (a) all candidate trees Stem Dip tested, (b) top ranked trees from first phase of testing (stem dip testing), and (c) top ranked trees in the second phase of testing (root dip inoculation; progeny had 50 to 100 percent survival in test of 6 rooted cuttings). Note: only 50 percent of stem dip winners have been tested through 2003).

Containerized seed orchards have been started for several breeding zones, and the first seed was produced in Fall 2002. Seed has already been sown for restoration and reforestation activities on federal and private lands. Control pollinations are being used to generate full-sib families for evaluation using the root dip test.

The number of resistance mechanisms and their inheritance is under investigation. At this preliminary stage, two groups of resistant families appear evident from greenhouse tests—1) families that show high survival and 2) families that show longer time to mortality ('slow dying'). In greenhouse testing, some parents show very high survival (50 to 100 percent) as rooted cuttings or as full-sib families versus no survival for many of the most susceptible parents (table 1; and Sniezko and others, this proceedings). In greenhouse testing, time-to-mortality varies by 50 percent or more among families, with a very few families showing much slower mortality. Further confirmation is underway (figure 2, also Sniezko and others, unpublished data). Traits such as 'high survival' may have immediate utility in field plantings while more information is needed on the 'slow dying' response (and further breeding may be needed).

The underlying nature of resistance is currently under investigation. Preliminary results from a study at OSU showed that 24 hours after root inoculation with zoospores, a susceptible family had more cysts than the resistant family tested (Eun-Sung Oh, personal communication). The hyphal growth in the susceptible family was faster than in the resistant family following stem inoculations (mycelium was used to inoculate, and the stem was examined four weeks later). Fewer hyphae in the resistant family suggested that there may be a defense response in resistant families against *P. lateralis* (Eun-Sung Oh, personal communication).

Table 1—Percent mortality in seedling families at ten months in the greenhouse root dip trial, results from 2000 test

	Male Parent						
Female Parent	CF1	CF2	510049	510008	118569	117344	**Mean**
118573		58	50			33	47
117490		0	6[a]			0	2
510042	67[a]			71[a]	100[a]	63[a] 92	79
118562	38[a]		72[a]	42	89[a]	54	59
510041	67	25				56	49
510044	42	61	83[a]		94[a]	61[a] 56	68
OP	39[a]						
Mean	51	36	53	56	94	46	

[a] Reciprocal cross

Field tests have been established (primarily since 2000) to examine field resistance and its durability. In general, the survival in field tests correlates well with greenhouse tests (for example, see figure 3; Sniezko and others 2000). There are exceptions and further investigation is needed. The numerous phenotypic field selections made since the program began (over 10,000, many now confirmed as resistant or susceptible) provide an opportunity for monitoring in the field. Several candidate trees (such as 510015, 510005, and 117490—selected in high disease hazard areas prior to 1991 and shown to be resistant in greenhouse testing) have been revisited and found to be alive and healthy (Chuck Frank and Leslie Elliott, personal

communications). Further investigation of the status of confirmed resistant parents is planned. How different field environments (for example, soil temperature and sustained flooding of low areas) influence effectiveness of resistance is currently unknown.

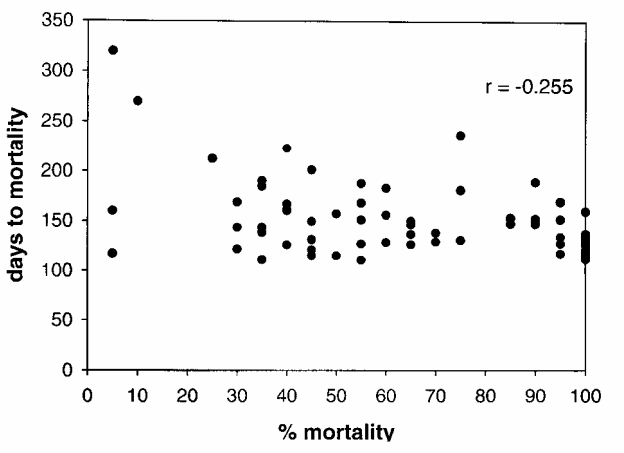

Figure 2—Mortality rate (days to mortality versus percentage mortality for Port-Orford-cedar families in a 2001 greenhouse resistance test.

P. lateralis has shown relatively little genetic variation (Goheen and others 2003b; McWilliams 2000). Coupled with the relatively low spread and population sizes of *P. lateralis*, the evolutionary potential of a pathogen such as *P. lateralis* may be low. Therefore, resistance on most sites may have a high likelihood of being durable (McDonald and Linde 2002).

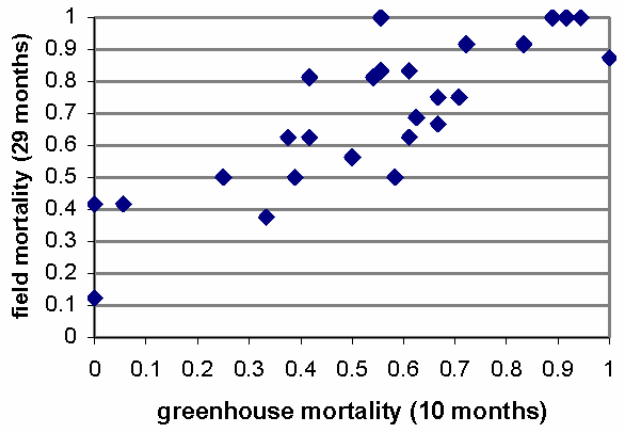

Figure 3—Proportion mortality of 26 families in 2000 greenhouse test (OSU root dip) and field site (Camas Valley). (r = 0.84)

Federal land managers are currently evaluating options in managing POC in *P. lateralis*-infested areas, and resistant seedlings may play a key role on some sites (USDI-BLM and USDA-FS 2004). In addition, private landowners are very interested in the availability of resistant POC, and the joint U.S. Forest Service–BLM effort is currently the only source for seed.

Future Needs

1. Additional selections to increase the genetic base of resistance in some breeding zones. Some breeding zones require evaluation of more than 1000 additional candidates.

2. Completion of second phase (root dip inoculation and assessment) of resistance testing for remaining candidates.

3. Establishment of seed orchards for all breeding zones in which resistant seed is needed. Production of resistant seed with a broad genetic base for each zone.

4. Further elucidation of resistance mechanisms, their underlying nature, and their inheritance.

5. Further evaluation of field plantings. This will be key to discerning whether resistance is durable. Are the resistance mechanisms exhibited in the greenhouse trials equally effective in the field? The resistant material consistently has higher survival than the susceptible material. However, survival of the resistant material is sometimes less than in controlled greenhouse testing. It should be noted that these sites are often visited only annually, and it can be difficult to definitively discern the cause of mortality on young seedlings. Within sites currently classified as high hazard, there may be differences in mortality. Some sites have standing water for sustained periods, and these may be the highest hazard especially to young seedlings. Is foliage on very small seedlings also an infection court, thus bypassing root defense mechanisms?

6. Examination of the susceptibility of very young resistant seedlings in the field. Small resistant seedlings or rooted cuttings may be more susceptible (first few years after planting) than larger resistant trees.

7. Monitoring the durability of resistance. Field trials will eventually give good data on this, but the 1000's of parent trees selected can also serve a monitoring function. Some of the oldest confirmed resistant trees have recently been revisited and are still alive. In the future, additional selections will be visited.

8. Further summary of the resistance data to refine estimates of the frequency of natural resistance and geographic trends in resistance.

9. More knowledge about *P. lateralis* and its genetic variation and the geographic origin of this pathogen.

Maintaining genetic diversity is a major focus of the resistance breeding program. A proposal is in preparation to investigate the levels of genetic variation in the resistance populations (Kolpak and Sniezko, personal communication).

10. Planting strategies for using resistant Port-Orford-cedar. Do resistant trees 'harbor' *P. lateralis*?

Summary of Program

Most Port-Orford-cedar trees are very susceptible to *Phytophthora lateralis*, but useable levels of natural resistance to this non-native pathogen exist. POC is highly amenable to a program to quickly evaluate resistance and produce resistant seedlings. Seed production can be accomplished on very young trees in pots (figure 4) (Elliott and Sniezko 2000), and this permits rapid updating of orchards as new selections are made or as breeding increases the level of resistance. Cooperation among FS, BLM, OSU and others coupled with the silvics of POC have led to rapid early progress in developing resistant populations. Continued progress is expected. Monitoring of field validation plantings will provide information on the durability of genetic resistance. Few, if any, operational resistance programs for forest trees have made such rapid progress.

Figure 4—Pollination bags on Port-Orford-cedar in a containerized seed orchard.

Acknowledgments

Numerous individuals in the USDA Forest Service, USDI BLM, and Oregon State University have contributed immensely to the progress of the resistance program. State, county, private landowners, as well as National Parks have contributed candidates for resistance testing and/or sites for field validation of resistance. Jodie Sharpe, Lee Riley and Scott Kolpak provided key support with summaries, graphs and analyses used. Angelia Kegley and Leslie Elliott provided support in editing and reviewing this document. A special thanks to both Everett Hansen and Don Goheen for their continual inputs and insights.

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Need for Studying Both Host and Pathogen in Gene-for-Gene Systems

Thomas L. Kubisiak, C. Dana Nelson, and Henry V. Amerson

Fusiform rust caused by the fungus *Cronartium quercuum* f. sp *fusiforme* is the primary disease of southern pines in the southeastern U.S. Economic losses are estimated at approximately $28M annually. Data collected over the past ~20 years or more suggests that the fusiform rust–southern pine pathosystem largely conforms to a gene-for-gene system (Powers and others 1977; Nelson and others 1993; Wilcox and others 1996; Stelzer and others 1997). In other words, the presence or absence of a stem gall results from the interaction between both host and pathogen. Infection is the character of this interaction. It is not simply a character of just the host or the pathogen. Resistance and pathogenicity, respectively, designate the character of the host and pathogen, although infection (presence or absence of a gall) is a measure of the interaction of these two characters.

Despite this knowledge, efforts to manage this pathosystem have focused almost exclusively on breeding for resistance in the host. The approach taken has been to screen both open-pollinated and full-sib families of the host with a broad source or bulk inoculum (Anderson and Powers 1985). Although this approach has made it possible to identify trees or families potentially harboring large numbers of resistance genes, control of the attacking pathogen's genetic constitution has not been possible and the genetic variability existing within the pathogen population is not known. As a consequence, little information is available regarding the nature of the resistances currently being deployed.

In an attempt to better understand the nature of these resistances, a concerted effort has been made to identify and 'tag' specific resistance genes with DNA markers (Wilcox and others 1996). To date, as many as eight different resistance genes (Fr1 to Fr8) have been mapped (Henry Amerson unpublished data). The associations between resistance loci and marker loci have been observed to be robust and repeatable, and these markers are being confidently used to identify resistant progenies within these defined host pedigrees.

Although resistance genes have been successfully mapped in many host species, few corresponding genes have been mapped in their pathogens. Only relatively recently were avirulence genes in the wheat stem rust fungus *Puccinia graminis* mapped (Zambino and others 2000). Mapping resistance genes in the host is only half the picture. To effectively manage gene-for-gene pathosystems, it is essential that the corresponding genes in the pathogen (avirulence genes) be identified and tagged with molecular markers. Once markers tightly linked to pathogen avirulence alleles have been identified and their usefulness confirmed in natural populations, that is, linkage disequilibrium of the markers is confirmed, these markers could be used to more effectively manage these pathosystems by directly estimating avirulence allele frequencies in natural populations of the pathogen hence allowing for more informed decisions regarding host resistance gene deployment.

Cooperative research between the USDA Forest Service's Southern Institute of Forest Genetics in Saucier, Mississippi and the Department of Forestry at North Carolina State University is currently underway to map the corresponding avirulence genes in the fusiform rust pathogen. Crosses between single-spore isolates of the fungus are being conducted to produce lines heterozygous for specific avirulence genes. These fungal lines are being used to challenge host families segregating for the corresponding resistance genes. DNA markers within the host are being used to identify the selecting (resistant) and non-selecting (susceptible) progenies. Fungi from infected trees within these two progeny sets (resistant and susceptible) are being screened with DNA markers to identify markers associated with avirulence.

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Inherent and Induced Resistance to Pitch Canker in *Pinus radiata*

Thomas R. Gordon and Christopher J. Friel

Pitch canker, caused by *Fusarium circinatum*, was originally described as a disease affecting plantation-grown pines in the southeastern U.S., where it remains a chronic problem. Pitch canker was first recognized in California in 1986 (McCain and others 1987). Although many native California pines are susceptible to pitch canker, *Pinus radiata* (Monterey pine) has been by far the most heavily damaged. Individuals within *P. radiata* vary in their susceptibility to this disease, as a result of both inherent genetic and induced resistance.

The first indication that some Monterey pines were resistant to pitch canker was the presence of disease-free trees in areas where pitch canker was well established. Unaffected trees could be differentiated from those showing symptoms of pitch canker by comparing their response to artificial inoculations. Disease-free trees generally showed a slower rate of lesion expansion than those that were visibly susceptible. Through vegetative propagation it was possible to show that relative susceptibility to pitch canker was determined primarily by the host genotype rather than circumstances under which it was growing (Gordon and others 1998). Thus, the absence of infections in heavily infested areas offers a good indication of resistance to pitch canker. However, it is also true that genetically susceptible trees can remain un-infected for many years, not-withstanding close proximity to diseased trees. The reasons for this variable lag period are unknown.

An assessment of the relative susceptibility of individual trees is routinely accomplished through mechanical inoculations and subsequent measures of lesion length at the site of inoculation. Incubation periods can be as short as three weeks in a growth chamber, but may require three months or more under field conditions. This reflects the influence of temperature on growth of the pathogen. A constant moderate temperature (such as 25° C) is near optimal for the pathogen, whereas cooler temperatures result in slower growth. In either case, time course studies show that after a sufficient incubation period, inoculated branches sustaining long lesions will eventually be girdled, and those with short lesions will not. Girdling kills the branch distal to the point of inoculation, which produces the dieback symptoms typical of pitch canker. Thus, the shorter the lesion at the time of measurement, the lower the probability the tree in question will sustain girdling lesions.

The extent of lesion development is influenced by the host genotype, but potentially by other factors as well. Thus, multiple inoculations of a tree (one inoculation on each of several branches) may yield short lesions on some branches, suggestive of resistance, but one or more long lesions as well. The reasons why some inoculations fail to produce long lesions on trees that are genetically susceptible have not been identified, but may include differences in the physiological status of individual branches.

Although it is clear that Monterey pines differ in susceptibility and that these differences remain apparent in vegetatively propagated clones, the heritability of disease resistance has yet to be established. This question has been addressed, in part, by collecting seed from known resistant and susceptible trees and assessing the susceptibilities of their progeny. Such studies show that both resistant and susceptible trees produce seed that gives rise to seedlings with a wide range of susceptibilities. In both cases, the distribution of reaction types is at least superficially similar to what is found among individuals within most native and planted stands of Monterey pine. If further testing substantiates these observations, one would have to conclude that heritability of resistance to pitch canker is low. However, only seeds from open-pollinated cones have been tested. Progeny of controlled crosses may reveal different estimates of heritability.

The extent to which pitch canker develops in a tree is obviously influenced by the genetically determined susceptibility of that individual. However, susceptibility is not a static characteristic and may change over time. Specifically, trees subjected to repeated inoculations over a period of two years supported progressively lower levels of lesion development (Bonello and others 2001). Thus, trees may manifest systemic induced resistance in response to infections by the pitch canker pathogen. This phenomenon has been confirmed under controlled conditions, but may also occur naturally. In particular, the occurrence of systemic induced resistance is suspected where heavily-infected trees go into remission. Disease remission has been observed in a number of monitoring plots (Gordon and

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others 2001) that were first established in 1992 (Storer and others 2002).

The physiological mechanisms responsible for resistance to pitch canker have not been identified, but may involve effects of resin on the pathogen. Resin is known to have a primary role in defense against insects and pathogens that attack pines. Thus, the ability to tolerate anti-microbial components of resin, such as monoterpenes, may be a perquisite for pathogenesis on pine. Evidence to support this hypothesis comes from experiments showing differential inhibition of nonpathogenic strains, relative to those that are pathogenic on pine, in the presence beta-pinene and limonene (Friel and Gordon, unpublished data). Studies are underway to determine if the resin composition of trees is predictive of their susceptibility to pitch canker.

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Resistance of Pines to Dwarf Mistletoe

Robert F. Scharpf

Introduction

Last year's WIFDWC in Powell River, British Columbia emphasized the high level of interest and importance we place on research and development of resistance to forest pathogens in western North America. In those few days alone, seven papers or posters were devoted to some aspect of forest disease resistance. Interestingly, however, none of the presentations involved resistance to dwarf mistletoes (*Arceuthobium*), one of the most widespread and damaging pathogens in the West. Because of this omission, I think it is appropriate at this meeting in Grants Pass to discuss what is known about resistance to this damaging group of pathogens, mainly because southern Oregon and northern California contain one of the world's greatest concentrations of dwarf mistletoe taxa (Hawksworth and Wiens 1996). More than a dozen species out of about 40 in North America are found in this rather small geographic area. In addition, most of the studies on dwarf mistletoe resistance have been conducted in these states. A summary of most of this work can be found in Hawksworth and Wiens 1996; Scharpf and Roth 1992, Scharpf, Kinloch and Jenkinson 1992 and Shamoun and DeWald 2002. Unfortunately, relatively little research and development is currently under way. I will summarize two or three examples of past work on resistance in pines and briefly discuss some of the on going studies.

Expressions of Resistance

I can think of no other forest pathogen–host combination that shows more variation and expression of resistance than the dwarf mistletoes and their hosts. In an effort to simplify some of these relationships, Hawksworth and Wiens developed "susceptibility classes" based on the percentage of trees infected within 6 m of heavily infected hosts (table 1). Many dwarf mistletoe species not only have primary hosts, but also secondary, occasional, and rare hosts, as well. Some dwarf mistletoes infect only a single host species, whereas others can parasitize several genera. For example, *A. laricis* primarily infects *Larix* and *Tsuga*, but also occurs on *Abies*, *Pinus* and *Picea*. Although Hawksworth and Wiens termed the relationship "susceptibility classes", it could just as well been called "resistance classes".

Table 1—Classes of host susceptibility to *Arceuthobium*

Class	Host	Infection Level
I	principal	>90 percent
II	secondary	90–50 percent
III	occasional	50–5 percent
IV	rare	<5 percent
V	Immune	0

From Hawksworth and Wiens (1996).

Several other host–parasite reactions, I believe, are also expression of resistance. Excessive swelling of host tissue, restricted endophytic growth of the parasite and lack of mistletoe shoot production have been called an "incompatible host–parasite relationship" by Hawksworth and Wiens. I believe these reactions are expressions of resistance. The wide variation in broom development and morphology also suggests a possible resistance response in some hosts. Hypersensitive reactions to the penetrating seeds have been observed in some host–parasite combination indicating a very specific-resistance mechanism. Also, the formation of a suberized layer at the infection site indicates an active resistance response to infection.

Several investigators have made observations of what appears to be obvious resistance in the field. "Susceptible" hosts with no infections have been reported growing within heavily infected stands. In these cases, the only logical explanation for the absence of infection is resistance. Ponderosa pine in Oregon has been reported to express "juvenile susceptibility." Among the true firs of California, young red firs appear to be more resistant to infection than larger trees.

Field studies in Oregon also provide strong evidence for resistance within individual, grafted selections of ponderosa pine. In one case, it appeared that the endophytic system of the parasite failed to grow across a graft union between an infected rootstock and the scion of a resistant candidate. In another case, a forked tree that developed from a susceptible rootstock; and, after 22 years, a resistant scion showed no infection on the scion but numerous infections on the rootstock.

Although the field observations and past studies have received little additional attention, it appears that several mechanisms of resistance to *Arceuthobium* may be present. In order to keep this paper reasonably brief, I will discuss only three studies involving resistance of pines to dwarf mistletoe.

In: Geils, B. W. comp. 2004. Proceedings of the 51st Western International Forest Disease Work Conference; 2003 August 18–22; Grants Pass, OR. Flagstaff, AZ: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station.

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Jeffrey Pine, California

The first study discusses a case in which resistance was observed in a plantation of Jeffrey pines at the Institute of Forest Genetics, Placerville, California. (Scharpf and Parmeter 1967). The plantation was established in 1940 to study the growth of pines from different geographic locations in the Sierra Nevada. Randomly spaced, 16-tree blocks constituted the plantation. Unknowingly, the plantation was located adjacent to a stand of ponderosa pine infected with dwarf mistletoe. Fifty years later, many of the Jeffrey pines were heavily infected with dwarf mistletoe but others much less so. Results of a study indicated that the higher elevation seed sources were the most severely infected; the low elevation sources were much less infected; and the mid-elevation sources showing moderate infection. In a subsequent test to confirm these observations, a young plantation of Jeffrey pines from the same seed sources was inoculated in 1980 and 1981 with a local source of seeds of dwarf mistletoe (*A. campylopodium*) on ponderosa pine (Scharpf, Kinloch and Jenkinson 1992).

In 1980, 3000 inoculations were made on 50 trees from each of 3 seed sources for a total of 9000 inoculations. In 1981, the same trees were inoculated with a total of 4500 seeds. After five years, results of the study confirm the earlier work by Scharpf and Parmeter (1967) in that the low elevation, the Foresthill source of Jeffrey pine showed significantly fewer trees infected and fewer infections per tree than the other seed sources tested (table 2). Therefore, it appears that different geographic populations of Jeffrey pines express different levels of resistance to *A. campylopodium*. Unfortunately, the factors that determine resistance among different populations remain unknown.

Table 2—Dwarf mistletoe seed retention and infection of Jeffrey pine inoculated in 1980 and 1981

Jeffrey pine seed sources: Site and time of collection [a]	Trees infected in 1985	Seeds on branches the spring after inoculation [b]	Infections in 1985–1986 from seeds on branches
		percent	
High Meadows, Fall 1980	50.0	12.7	21.8
South Lake Tahoe, Fall 1980	24.0	8.7	14.5
Foresthill, Fall 1980	8.0	6.0	4.4
High Meadows, Fall 1981	46.0	34.6	10.0
South Lake Tahoe, Fall 1981	44.0	28.2	7.8
Foresthill, Fall 1981	20.0	26.2	3.3
Laguna Mt., Fall 1981	48.0	41.3	9.1

[a] Fifty trees per source were inoculated, except for 25 trees from Laguna Mountain.
[b] Inoculation totaled 3000 seeds per source in 1980, and 1500 seeds per source in 1981, except 720 for Laguna Mountain source.
Adapted from Scharpf and others (1992)

Ponderosa Pine, Oregon

Another study that shows a very convincing case for resistance was that conducted in Oregon by Roth in the 1960s (Scharpf and Roth 1992). Thirty resistant candidate ponderosa were selected from various locations in Oregon, and 12 to 15 grafts made from each candidate on nursery grown rootstock. Over 400 of these grafted trees were then out planted in central Oregon from 1967 to 1969 in a random block design in a heavily infested stand of ponderosa pine. The final data on the plots was collected 20–22 years later in 1989. Summaries of the results are as follows:

- A wide variation in resistance was found among the grafted selections ranging from 100 percent infection of some selections to no infection of others.
- The mean number of infections varied widely among the clones. Also the greatest number of infections was found in clone trees with the greatest percentage of trees infected.
- Ungrafted "field run" seedlings from three forests were heavily infected, showed no evidence of resistance and experienced heavy mortality before the end of the test.

The results of these tests in ponderosa pine correspond closely with those in Jeffrey pine in that there was a wide variation in resistance among trees from different geographic locations and that resistance persisted after many years of exposure to the disease.

Ponderosa Pine, California

The last study I want to discuss is that currently being conducted by Paul Stover and Dennis Ringnes at the U.S. Forest Service, Tree Improvement Center, Placerville, California. Details of this study appear in the mistletoe committee report in these proceedings. Based on early observations by Dr. W. W. Wagener on possible resistance in eastside ponderosa pine, guidelines were established to select and test resistant candidate pines. From 1993 to 1996, field crews selected 109 resistant candidate trees from several locations in northern California. In 1997, seedlings from open pollinated resistant candidates, control crosses and susceptible trees were planted in a random block design at the nearby Badger Hill Tree Orchard. In 2001–2002,

each tree was inoculated at the rate of 10 seeds per tree; and in 2002–2003, the inoculation rate was 20 seeds per tree.

Although it is to soon for any meaningful results to be obtained, the investigators are optimistic that useful information will result from the tests. Infections are developing from the inoculations that we hope will provide sufficient data for a statistically sound determination of resistance among the selections and crosses.

Conclusions

So what are some of the conclusions we can draw from past and present work on dwarf mistletoe resistance?

- Resistance has been observed in the forest by several investigators, and resistance is probably much more common than we realize.
- There is wide variation in resistance within and among conifer species, and variation in resistance within species from different geographic areas.
- Resistance is expressed in many ways, and probably is the result of a complex multigenic process.
- Lastly, we know very little about the mechanisms and inheritance of resistance in conifers to dwarf mistletoe.

So why is there so little research being conducted on resistance to dwarf mistletoes? After all, they are among the most damaging and widespread pathogens in western North America.

- Managers, in general, see little need for widespread use of resistance in the forest against these diseases. Current thinking suggests that most dwarf mistletoes can be managed at tolerable levels through silvicultural or other means. Use of resistance mainly seems applicable only in high use, high value areas.
- Development of resistance is considered to be an expensive long-term commitment. Few research institutions and scientists are willing to devote the time and resources necessary to achieve meaningful results.
- Understanding and developing resistance is perceived to be a high risk, very complex area of research with the chance of success questionable at best. Current evidence suggests that resistance is likely a complex multigenic process, and we do not have the technology or resources to obtain results and provide answers.

So where do we go from here?

- Field workers should continue to report mistletoe resistant candidates they observe in the field. Where appropriate, then candidates should be tagged and located for future examination.
- We should protect the currently known resistant candidates, particularly valuable ones like the ones on the “Roth Pringle Butte Plots”.
- We should continue to propagate and field test candidates as resources allow.
- A centralized database of known resistant candidates should be established. This will allow investigators ready access to test material when resources and technology allow for further research.
- Investigators should keep abreast of new developments in the area of genetics and biotechnology that may apply to testing and development of resistance in dwarf mistletoes.
- A multidisciplinary approach to research and development efforts should be considered as the most efficient way to obtain the desired results.

Acknowledgement

Thanks to Cindy Collins at the Institute of Forest Genetics for her help in preparing this paper.

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Lew Roth Walt Thies John Pronos Alan Kanaskie John Browning Borys Tkcaz
Kathy Lewis Rona Sturrock Ellen Goheen Brian Geils Joan Webber Mike McWilliams

Nursery Pathology Committee Report August 18, 2003, Central Point, OR

Diane Hildebrand, Acting Chair

The 22nd Annual Nursery Pathology Workshop was held in conjunction with WIFDWC on Monday, August 18, 2003, from 12:30 pm to 5 pm. Ten participants included state, federal, and industrial forest pathologists involved in forest tree nurseries, and other interested individuals. Thanks to Katy Marshall for organizing and hosting the workshop this year at J. Herbert Stone Nursery.

The Western Nursery Pathology Workshop was made an official WIFDWC committee, the Nursery Pathology Committee. We will continue to meet on the Monday afternoon in conjunction with WIFDWC each year. Bob James is the Chair of the committee.

Meeting

News and Notes

Tom Landis—Volume 5 of the Container Manual is being scanned onto CD in Adobe PDF and MS Word. Working on a two-CD version with high quality graphics and pictures. Tom will have completed 30 years with the USDA Forest Service on December 17, 2003, and is planning to retire. He hopes to contract back to continue working on Forest Nursery Notes and volume 7 of the Container Manual.

Diane Hildebrand—The results of the first phase of the USFS Alternatives to Fumigation project (1993 to 1998) will be published as a PNW General Technical Report hopefully next year. Co-authors are Diane Hildebrand, Jeff Stone, Bob James, and Susan Frankel.

Discussion

Faith Campbell (American Lands Alliance), and others—Old-fashioned methods of back-crossing American chestnut with resistant chestnuts from other countries is looking successful. Resulting hybrid so far is 90 percent American chestnut and is still resistant to chestnut blight.

Katy Marshall—Sporadic outbreaks of cypress canker on Port-Orford-cedar at Dorena Genetic Resource Center are caused by *Seiridium cardinale*. Using methods described by Art McCain in 1977, and assistance and advice from Jeff Stone (OSU) and Jean Williams-Woodward (University of Georgia), I designed an inoculation trial to test the effectiveness of several fungicides. Artificial inoculation of seedlings with mycelial plugs followed by ninety days incubation was most successful. Fungicides tested were Cleary 3336, Daconil, and Champ (tri-basic copper). Cleary 3336 was most effective.

A monitoring study of 2+0 DF in Field K at Stone Nursery showed that falldown in survival of some seedlots of 2+0 DF survival after outplanting was probably not due to hidden root disease. There were very few seedlings with healthy tops but diseased roots in samples I took at packing (after freezer storage and after district cooler storage). However, the proportion of seedlings with visually healthy roots that were colonized by *Cylindrocarpon* increased during cooler storage and after outplanting. These may have been saprophytic or potentially pathogenic species of *Cylindrocarpon*, so their role and possible effect on seedling roots was not clear. At the end of the study, almost the same proportion of randomly sampled healthy and dying seedling roots were colonized by *Fusarium* or *Cylindrocarpon*. About half of the seedlings had unusually pale foliage on the upper half of the stem. Nutrient analysis of foliage from one seedlot indicated that the pale needles were very low in nitrogen. This might also have contributed to poor performance.

Corky, decayed roots associated with *Cylindrocarpon destructans* has been causing problems on bareroot western white pine at J. Herbert Stone Nursery. In some seedlots there is a high percentage of cull. Disease appears to be most severe in beds adjacent to irrigation lines, suggesting that over-watering may predispose seedlings to disease. Similar corky root symptoms are associated with the nematode *Xiphenema* in Douglas-fir. However, these symptoms have not been found on Douglas-fir at Stone Nursery.

John Browning—1. Weyerhaeuser is in second round of testing metam-sodium and chloropicrin as alternative fumigants.

2. Late spring fumigation with MBC resulted in transplant mortality, when incomplete de-gassing of the fumigant occurred prior to transplanting. Gas testing in the soil was not an adequate indicator of when it was safe to plant! Growers pushed to get transplants into the ground by mid-May (although fumigation was delayed until late April 2003).

3. DF 1+1 stunting during the first growing season after fallow was associated with high nematode counts (fungal

feeders). These nematodes could be found on surface sterilized roots of both seedlings (1+0) and seedlings after transplanting. The importance of nematodes in transplant disease complexes has yet to be determined.

4. Interactions between glyphosate (Roundup) and *Cylindrocarpon didymum* were examined in a greenhouse study. Seedlings treated with glyphosate (140 ppm product) were damaged (80 percent mortality), but seedlings treated with both glyphosate and *Cylindrocarpon* had less damage. Seedlings treated with *Cylindrocarpon* were smaller than control seedlings, which were the largest in the four treatments. The pathogenicity of *Cylindrocarpon didymum* is uncertain, and it may be functioning merely as a saprophyte on root tissue. The inoculation established that macroconidia are a potent source of root infection, and that the organism demonstrated the ability to colonize new root tissue during the experiment.

Bob James—Provided a written summary of his nursery work, which follows with annotations from the meeting.

Current Projects: 1. Tests to evaluate alternatives to pre-plant soil fumigation with methyl bromide/chloropicrin (MBC) at the Lone Peak Nursery, Draper, UT (Utah Department of Natural Resources)

2. Tests to bare fallowing with periodic cultivation and chloropicrin (only) to the standard MBC fumigation for their effects on production of bare root bitterbrush seedlings

3. Tests to evaluate effects of various *Brassica* green manure crops, meal amendments and formulations of the biocontrol agent *Trichoderma harzianum* (locally developed) on production of bare root white pine seedlings at the USDA Forest Service Coeur d'Alene Nursery and on ponderosa pine seedlings at the USDA Forest Service Lucky Peak Nursery, Boise, ID (In cooperation with the University of Idaho).

4. Two tests completed at Coeur d'Alene and one at Lucky Peak Nurseries, a fourth test planned assessing *Brassica* meal amendments at Coeur d'Alene.

5. Tests to evaluate alternative heat treatments to sterilize styroblock containers and forest nursery soil (In cooperation with the USDA Forest Service Missoula Technology & Development Center).

6. Initial tests completed assessing dry heat (including radio frequency waves) on containers and soil treatment tests are underway

7. Molecular characterization of populations of *Fusarium oxysporum* from forest tree nurseries (In cooperation with the USDA Forest Service Rocky Mountain Research Station, Moscow, ID).

8. Work beginning in the fall of 2003 to compare isolates from different conifer seedlings (healthy and diseased) as well as those from forest nursery soil; comparisons also between pathogenic and non-pathogenic isolates

9. Etiology and control of Douglas-fir stem and branch cankers and stem dieback at the Webster Nursery (Washington Department of Natural Resources, Olympia, WA)

10. Fungal associates to be identified and nutrient manipulation effects on cankering to be determined. Isolated mostly *Phoma eupyrena*. Cracks in stem epidermis sometimes get infected, then get breakage and dieback.

11. Evaluation of the efficacy of T-22 formulations of *Trichoderma harzianum* (used to be called SoilGuard) to control root diseases (primarily seed-borne *Fusarium*) of container-grown seedlings at several nurseries in the Pacific Northwest (In cooperation with the Southern Forest Experiment Station, Moscow, ID).

12. Several container nurseries in Washington and Idaho to evaluate commercial formulations of T-22 (applied through irrigation system, shortly after emergence) to reduce impact of damping-off and root disease on different conifer and non-conifer seedling species.

Recent Evaluations: 1. *Fusarium* blight of container-grown ponderosa pine seedlings (Montana State Nursery, Missoula). Blight started on scattered secondary needle fascicles and moved to main stems. One third of crop lost in a few weeks. Mostly *F. avanaceum*, *F. sporotrichiodes*, and *F. proliferatum*. Roots healthy but contained *F. proliferatum*. Controlled by fungicides, for example, Cleary's.

2. *Fusarium* root disease of bare root 1-0 western white pine and Douglas-fir seedlings (USDA Forest Service Nursery, Coeur d'Alene, Idaho).

3. *Cytospora* blight of bare root red oak seedlings (USDA Forest Service Bessey Nursery, Nebraska).

4. Root disease of thinleaf alder seedlings (Lone Peak Conservation Nursery, Draper, Utah). Too wet, but drying winds cause mortality if don't keep wet.

5. Stem blight of golden willow cuttings (Montana State Nursery, Missoula). In cold storage, *Venturia* (shepherd's crook).

6. Epiphytic root fungal growth on container-grown white pine seedlings (Pelton Reforestation Ltd., Alberta, Canada).

7. Storage mold associated with treatments of container-grown western red-cedar seedlings with the animal

repellent Plantskydd® (blood-based) (USDA Forest Service Nursery, Coeur d'Alene, ID). Can't dip seedlings before storage.

8. Effects of the animal repellent Plantskydd® on colonization of styroblock containers with potentially-pathogenic fungi.

9. Etiology of whitebark pine container seedling mortality (USDA Forest Service Nursery, Coeur d'Alene, ID).

10. *Phoma* tip blight of eastern and western white pine seedlings (Dorena Tree Improvement Center, OR).

11. *Cylindrocarpon destructans*-associated root disease of container-grown western white pine tree improvement stock (USDA Forest Service Nursery, Coeur d'Alene, ID). Not sure if *Xiphenema* involved.

12. *Thelephora terrestris* causing stunting of container-grown Douglas-fir seedlings (USDA Forest Service Nursery, Coeur d'Alene, ID). Growth of fruiting body clogs drain hole of container.

13. Contamination of Douglas-fir seedlots with potentially-pathogenic fungi (Idaho Department of Lands); post-stratification.

14. Root binding of container-grown ponderosa pine seedlings and associations with potentially-pathogenic organisms (CalForest Nursery, Etna, CA); kept too long in containers.

15. Cotyledon blight of container-grown western larch seedlings (Potlatch Nursery, Lewiston, ID); seed-borne *Fusarium*.

16. Rapid mortality of container-grown bitterbrush seedlings (USDA Forest Service Lucky Peak Nursery, Boise, ID); heat damage.

17. Stem cankers of container-grown Douglas-fir seedlings (Washington State University Extension- Puyallup, WA).

18. Leaf spot and stem dieback of container-grown aspen seedlings and *Rhytisma* tar spot of container-grown maple seedlings (Reggear Tree Farm, McCall, ID).

19. Container-grown conifer seedling diseases and assays of Douglas-fir seed for potential pathogens (Hawaii Agriculture Research Center, Hilo, HI).

Sudden Oak Death (SOD)

Gary Chastagner—The recent detection of *Phytophthora ramorum* on ornamental plants at several commercial nurseries in Oregon and Washington has generated a lot of concern within the Pacific Northwest (PNW) nursery and Christmas tree industries. Douglas-fir is one the current regulated hosts of this pathogen and a shoot dieback on grand fir Christmas trees at a site in California that is adjacent to a mixed wooded area containing highly susceptible bay laurel trees has recently been confirmed to be caused by this pathogen.

Although no *P. ramorum* has been detected in any Christmas tree fields or conifer nurseries in the PNW, these industries are at risk of potential disruptions in the shipment of trees and nursery stock if efforts to eradicate this pathogen are unsuccessful and the pathogen spreads into other areas in western Oregon and Washington. During this past year, a cooperative project involving Washington State University and Oregon State University was initiated to determine the susceptibility of various conifer species to *P. ramorum* and identify fungicides that are effective in protecting conifers from this pathogen.

In an effort to better understand the potential impact this pathogen might have on the Christmas tree and conifer nursery industries, a series of inoculation studies were conducted to determine the potential susceptibility of foliage and shoots from 25 conifers and nine sources of intermountain Douglas-fir to *P. ramorum*. Symptoms associated with *P. ramorum* infections included needle blight, a shoot blight resulting from needle infections, and stem lesions resulting from the growth of the pathogen from infected needles/shoots into the 2002 wood. Growth stage had a significant effect on susceptibility. Douglas-fir shoots that were inoculated just after bud break were much more susceptible than those that were inoculated 2 to 8 weeks later. Twenty of the 25 conifers tested, including many of the important species that are used as Christmas trees throughout the world, were susceptible to *P. ramorum*. Some true firs were highly susceptible.

The sensitivity of *P. ramorum* to 20 fungicides that are commonly used to manage *Phytophthora* diseases in agricultural and nursery crops has also been determined under laboratory conditions. The effect of each fungicide on mycelial growth and germination of zoospores was assessed by comparing the extent of growth or the percentage of germinated spores on fungicide-amended media to growth and germination on un-amended media. These studies indicate that a number of the tested fungicides were very effective in inhibiting *P. ramorum* mycelial growth and zoospore germination. This included systemic materials like dimethomorph as well as contacts like maneb. Mycelial growth was much more sensitive to some fungicides than were spores (for example, mefenozam), while spore germination was more affected than mycelial growth with others (for example, chlorothalonil). Additional studies are in progress to determine the effectiveness of these materials

in protection Douglas-fir seedlings from infection by *P. ramorum*.

Susan Frankel—Occurrence of SOD in nurseries is major problem. There are questions about burying plants rather than burning them, and about the California practices of using redwood and Douglas-fir sawdust as growing medium, as well as natural sand and gravel. Bob Linderman in Corvallis and McDonald at Berkeley are testing growing media and longevity of inoculum.

Tour of J. Herbert Stone Nursery____

Thanks to Ken Wearstler, Nursery Manager, Steve Feigner, Culturist, and Dave Knight, Equipment Operator, for the nice tour of J. Herbert Stone Nursery. The highlight of the tour was the plantings using Q-plugs. This is a new and promising production technique for conifers at Stone Nursery. The Q-plug technology was developed for the vegetable transplanting industry. The Q-plug is smaller than a mini-plug and the potting medium includes a spongy polymer that holds the peat and other particles together. Seedlings are started in the Q-plugs in a commercial container greenhouse in January, when the commercial greenhouses are usually empty. In March, the Q-plugs are transplanted to the nursery beds. Transplanting the Q-plugs requires a special machine that loosens the plugs from the styroblock, and then a carousel transplanter designed to handle the small plugs. Plugs are sown in nine rows per bed. Seedlings grow in the field for one full summer and are lifted in winter. The resulting seedling will be larger than a 1+0, with a good root to shoot ratio. This stock type will be an improvement over the 1+0, especially for Douglas-fir. Q plugs might also be used to produce a large 2+0.

Recent Reports and Publications___

James, R.L. 2002. Biological control of *Fusarium oxysporum* and *Fusarium proliferatum* on young Douglas-fir seedlings by a nonpathogenic strain of *Fusarium oxysporum*. USDA Forest Service, Northern Region, Forest Health Protection. Report 02-2. 14 p.

James, R.L. 2002. Effects of preplant soil treatments on *Fusarium* and *Trichoderma* populations and fungal root colonization of 1-0 nondiseased ponderosa pine seedlings – USDA Forest Service Lucky Peak Nursery, Boise, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Report 02-3. 9 p.

James, R.L. 2002. Effects of preplant soil treatments on *Fusarium* and *Trichoderma* populations and fungal colonization of 2-0 nondiseased western white pine seedlings – USDA Forest Service Nursery, Coeur d'Alene, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Report 02-8. 11 p.

James, R.L. 2002. Effects of spring applications of dazomet on root diseases and performance of Douglas-fir and western white pine transplants – USDA Forest Service Nursery, Coeur d'Alene, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Report 02-9. 18 p.

James, R.L. 2002. Investigations of potential disease-causing organisms associated with production of container-grown bitterbrush seedlings – USDA Forest Service Lucky Peak Nursery, Boise, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 146. 9 p.

James, R.L. 2002. Root disease of 1-0 bare root ponderosa pine seedlings – Lone Peak Nursery, Draper, Utah. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 147. 8 p.

James, R.L. 2003. Accumulation of root pathogens on bare root Douglas-fir and Engelmann spruce seedlings induces foliar chlorosis after three growing seasons at the USDA Forest Service Nursery, Coeur d'Alene, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 148. 14 p.

James, R.L. 2003. Comparing three methods of assaying soil *Fusarium* and *Trichoderma* populations for integrated pest management in forest nurseries. USDA Forest Service, Northern Region, Forest Health Protection. Report 03-3. 14 p.

James, R.L. 2003. Diseases in forest nurseries: implications for forest managers. Western Forester (Society of American Foresters) 48(5) (In press).

James, R.L. 2003. Fungal associates of corky root syndrome on bare root 2-0 western white pine seedlings – USDA Forest Service Nursery, Coeur d'Alene, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 150. 7 p.

James, R.L. 2003. Fusarium blight of container-grown ponderosa pine seedlings – Montana State Nursery, Missoula, Montana. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 149. 14 p.

James, R.L. 2003. Stem lesions and dieback of Douglas-fir seedlings – Washington Department of Natural Resources, Webster Nursery, Olympia, Washington. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 152. 14 p.

James, R.L. 2003. Storage mold on conifer and hardwood seedlings – University of Idaho Research Nursery, Moscow, Idaho. USDA Forest Service, Northern Region, Forest Health Protection. Nursery Disease Notes No. 151. 5 p.

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Rust Committee Report
August 19, 2003, Grants Pass, OR

Brian Geils, Acting Chair

The Rust Committee breakfast was held on August 19, 2003 with good attendance and interesting reports of recent, current, and pending activities.

Discussion

Brian Geils—An extended review and synthesis on white pines, *Ribes*, and blister rust is being compiled for submission as a special issue of Forest Ecology and Management. Contributed articles will cover history, pathology, and management in North America;.

Richard Sniezko—Rust Busters meet at Dorena prior to this year's WIFDWC for a review of resistance work, cooperative projects, and mutual interests. The meeting included a field trip to several "historic sites" and much worthwhile discussion. With so much work being by many folks in various locations, there is a need for agreed terminology. A writing group is being organized.

Det Vogler—The next party conference for the IUFRO Rusts of Forest Trees is planned for July of 2006 at Placerville. There will be presentations and field trips.

John Schwandt—The Forest Service report, *Managing for Healthy White Pine Ecosystems in the United States to Reduce the Impacts of White Pine Blister Rust*, is published (http://www.fs.fed.us/foresthealth/publications/WPBRRepo rt.pdf).

Blakely Lockman—A database is being compiled to map the extent and condition of whitebark and limber pine and to identify critical information gaps.

Stephan Zeglen—Parks Canada hosted a Whitebark and Limber Pine Workshop on February 18–19 at Calgary, AB (a proceedings is available). The purpose of the workshop was to promote communication, coordination, and research for the protection of whitebark and limber pines from such threats as white pine blister rust.

Paul Zambino—A workshop has also been held in eastern Canada for a similar purpose.

Faith Campbell—White pines are being imported.

Horticultural use of *Ribes*—There was a brief discussion on the extent to which cultivated *Ribes* were being planted for various purposes and the possible concerns.

Don Goheen John Kliejunas John Dale Sue Hagle Judy Adams Susan Frankel Bill Woodruff Det Vogler Annette Mix

Hazard Tree Committee Report
August 19, 2003, Grants Pass, OR

John Pronos, Chair

The Hazard Tree Committee luncheon meeting was held on August 19, 2003 and had 32 people in attendance. We discussed the three topics that are summarized here.

Status of the North American Tree Failure Database (NATFD)

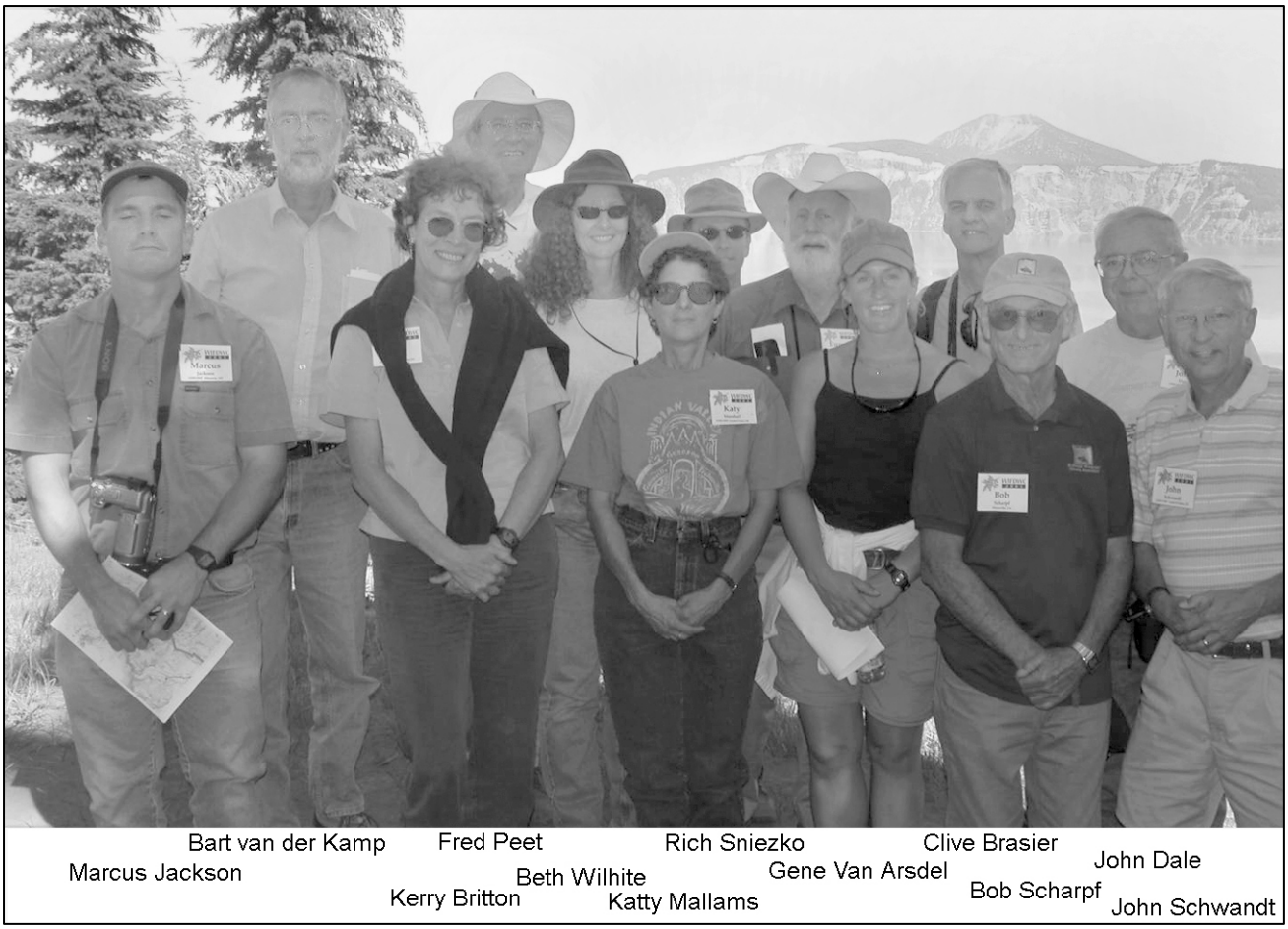
Efforts to create the NATFD began in January 2003, with a meeting in Sacramento, CA. The twelve people who were present represent the program's Steering Committee. Eight of the committee members were from the Forest Service and the other four represented the private sector, primarily the International Society of Arboriculture. A website and field form were completed under the direction of Judy Adams, and are currently being evaluated. After all comments concerning these two products are received, the Steering Committee will meet to discuss the proposed changes. After changes are made, the revised Internet and field forms will be made available for review by state/urban forestry contacts. The ISA representatives are planning to have a training session on the NATFD at the upcoming Tree Care Industry Association annual meeting in Baltimore next November. Once people have completed this training, they will be able to submit tree failure data on the Internet.

Next Western Hazard Tree Workshop

Planning has begun to hold the 4th Western Hazard Tree Workshop in Colorado in 2004. Jim Worrall has agreed to handle local arrangements and the field trip. Although dates have not been set, the workshop will be held in either the Montrose/Ouray or Durango areas. A group will meet in Denver sometime in October 2003 to plan the indoor and outdoor programs.

Hazard Trees for Forestry Images

University of Georgia personnel who maintain the Forestry Images website have agreed to include images of hazard trees and tree defects. They accept either 35 mm slides or digital images. John Pronos will gather photos from the Hazard Tree Committee members and forward them to Forestry Images. Images will be put into one of the following defect categories: root, butt, bole/trunk, branch, top, whole tree, or multiple defects. The due date for submitting photos to John is September 14, 2003.



Bart van der Kamp Fred Peet Rich Sniezko Clive Brasier John Dale
Marcus Jackson Beth Wilhite Gene Van Arsdel
Kerry Britton Katty Mallams Bob Scharpf John Schwandt

Dwarf Mistletoe Committee Report August 20, 2003, Grants Pass, OR

Katy Marshall, Chair

Thirty-two people attended the committee breakfast on August 20th. The Chairperson suggested that someone volunteer to be the next committee chair. No one spoke up immediately, but later in the week Fred Baker volunteered for the position. An email message was sent after the meeting to canvas all committee members on the e-mail list. All responses were in favor, so Fred Baker will assume the duties of Chairperson with planning for WIFDWC 2004.

Discussion

Forest Insect and Disease Leaflets

B. Mathiasen reported on the status of dwarf mistletoe-related FIDLS (Forest Insect and Disease Leaflets). Almost all of them have now been revised. The two ponderosa pine dwarf mistletoe FIDLs have been combined into one. The text and images are at the Washington Office where they will be printed. John Pronos took the lead on revising the gray pine dwarf mistletoe FIDL. It will also be published by the WO. Pinyon pine dwarf mistletoe FIDLs are available from Mary Lou Fairweather and Dave Conklin. Fred Baker is working on the eastern dwarf mistletoe FIDL. Jerry Beatty is working on the western white pine/sugar pine dwarf mistletoe FIDLs. Lodgepole pine dwarf mistletoe may be the next one to revise. The Regions can also publish FIDLs using the current format. Revision of the *Phoradendron* FIDL will wait until J. Kuijt has completed work on the taxonomy.

Verbal Reports

During the round robin, attendees reported on their dwarf mistletoe-related projects. As usual there were more people than time. The following items were mentioned that were not submitted for the report:

J. Muir—Funding for the western hemlock dwarf mistletoe and tree growth model has been approved. He is interested in the effects on succession in western hemlock/western red-cedar forests.

J. Adams—The dwarf mistletoe spread model TDP (Technology Development Project) is progressing slowly. The model is intended to account for dwarf mistletoe spread in uneven-aged stands.

G. Filip—Working with H. Maffei on removing Douglas-fir dwarf mistletoe brooms from old growth trees in campgrounds. No indication yet whether it has improved tree vigor. They did not remove brooms from trees with DMR > 4.

B. Geils—Working with Kailen Mooney, PhD student at University of Colorado on insect communities associated with dwarf mistletoe in ponderosa pine. Continuing work on deposition the Forest Pathology Herbarium–Fort Collins, Mistletoe collection (assembled by Hawksworth and Wiens). Most of the collection is now with the herbaria at University of California Berkeley (UC) and Smithsonian National Herbarium (US). The remaining work includes cataloging the *Phoradendron* and dispersing the numerous duplicates. When the catalog is complete, he will publish a report documenting the disposition of all assessed sheets (over 5000).

A. Kanaskie—Working on dwarf mistletoe "enhancement" to create marbled murrelet nesting platforms in coastal western hemlock forest.

B. Mathiasen—Has two new graduate students. They will work on prescribed fire and dwarf mistletoe, and southwestern dwarf mistletoe and bark beetles on ponderosa pine. Continuing work on taxonomy of western hemlock dwarf mistletoe, dwarf mistletoes on Brewer spruce, and species in Central America.

H. Merler—Working on hazard ratings for lodgepole pine dwarf mistletoe-infected stands to revise reforestation contracts. Concerned that rapid increase of dwarf mistletoe in young stands is going undetected.

L. Roth—Continuing work at Pringle Butte. Concerned about dwarf mistletoe research and control being continued beyond one person's career, and with agency commitment to reducing impacts of dwarf mistletoes. Convinced that resistant genes do exist although the level of heritability is not yet known.

B. Scharpf—Working as consultant on project with D. Ringnes (see VII. Genetics). Handbook of Pests of California Conifers, published by UC Berkeley, covering insects, diseases and animal damage, will be available through amazon.com.

B. van der Kamp—Has two students still working on mycoparasites of western hemlock dwarf mistletoe. A promising species is *Neonectria* (a weak canker fungus). It kills mistletoe infections completely by killing the host

tissue. The only difficulty is that it requires a wound as an entry court.

D. Vogler—Continuing work on NASA study related to mycorrhizae.

Submitted Reports

I. Taxonomy, Hosts and Distribution

Dan Nickrent, Southern Illinois University, Carbondale, IL; M. García and M. Martín, Real Jardín Botánico, CSIC, Madrid, Spain; R. Mathiasen, Northern Arizona University, Flagstaff, AZ—Maximum parsimony analyses were conducted on two data partitions (separately and combined): nuclear ribosomal ITS sequences for all 42 currently recognized species of *Arceuthobium*; and chloroplast trnT-L-F sequences for 34 New World species. The trnT-L-F sequences, which vary widely in length depending upon taxon, contain three times less phylogenetic signal than ITS, although homoplasy for this partition is lower. Several of the clades obtained from analysis of nuclear ITS sequences are also recovered using trnT-L-F sequences such as *A. guatemalense* and *A. pendens*, the *A. rubrum* group, the *A. vaginatum* group, and the *A. campylopodum* group. A manuscript reporting our results has been submitted to the American Journal of Botany.

Robert Mathiasen, Northern Arizona University, Flagstaff, AZ—A manuscript reporting several years of work on the host range of western hemlock and mountain hemlock dwarf mistletoes in the Pacific Northwest has been submitted to Forest Science. Sixty mixed conifer stands in California, Oregon, Washington, or British Columbia infested with western hemlock or mountain hemlock dwarf mistletoe were sampled to compare host susceptibility. Temporary circular plots were established around dominant, severely infected principal hosts. More than 14,000 trees were sampled in 712 plots.

John Muir and Don Norris, BC Ministry of Forests, Forest Practices Branch, Victoria—We are continuing fieldwork to test a possible subspecies of *A. americanum* in southeastern British Columbia. We expect to undertake a detailed comparison of shoot lengths, diameters and numbers. So far, we have observed several weeks' differences in anthesis and seed dispersal periods, plus some differences in the extent of systemic branch growth and size of stem swellings for the supposedly different subspecies. The subspecies also exhibits genetic differences based on the amplified fragment length polymorphism analyses reported by Jerome and Ford in Molecular Ecology 11:387–405; 407–420. 2002.

II. Physiology and Anatomy

III. Life Cycles

IV. Host-Parasite Relations

James T. Hoffman, USDA Forest Service, Region Four, Boise, ID—Field data are being collected from sixteen permanent plots in 45-year-old lodgepole pine stands in eastern Idaho. The 100-tree plots represent four replicates of four thinning regimes established in 1983. Plots have been re-measured every five-years. Objectives of the study are to determine:

The effects of pre-commercial thinning on growth of infected lodgepole;

The changes in dwarf mistletoe incidence and intensity over time;

The effects of dwarf mistletoe parasitism on long-term growth and mortality of lodgepole pines.

The Intermountain Region has a system of 60 permanent mistletoe plots in a variety of host types. The primary function of these plots is to gather data for model validation, but some plots are also intended to examine dwarf mistletoe spread rates in clearcuts, spread dynamics after thinning treatments, and spread from overstory to understory in the wake of a bark beetle outbreak.

V. Effects on Hosts

S. Kenaley, R. Mathiasen, and C. Daugherty, Northern Arizona University, Flagstaff, AZ—Although we initiated a two-year field study to examine the effects of dwarf mistletoe infection on cone and seed production of ponderosa pine and pinyon pine in northern Arizona, poor cone and seed production during Fall 2002 has caused us to shift this research to a new topic. Because northern Arizona is undergoing a bark beetle outbreak due to the several dry years, we are now examining the relationship between bark beetle attack and level of dwarf mistletoe infection in ponderosa pine. Eight study areas have been selected during March and April with varying levels of dwarf mistletoe infection. Dwarf mistletoe infestations in these sites have been identified, and trees within each study area have been selected for monitoring in relation to bark beetle activity over the next several months. Trees with DMRs of 0 to 6 have been tagged and will be monitored for attacks.

VI. Ecology

T. Parker, R. Mathiasen, and C. Chambers, Northern Arizona University, Flagstaff, AZ—We compared attributes of breeding bird communities in 19 northern

Arizona ponderosa pine stands infested with different levels of southwestern dwarf mistletoe. Of the 16 bird species we investigated, we detected a positive correlation between mean DMR, witches' broom volume, or snag density and bird abundance for four bird species, a negative correlation for five species, and no relationship for seven species. Three of the four species that were positively correlated with dwarf mistletoe severity or snag density were cavity-nesting birds. As part of this research, we developed a witches' broom rating system for use in ponderosa pine. We are preparing two manuscripts for publication based on the results of our work.

D. Russell, Bureau of Land Management, Medford (Oregon) District—During a ponderosa pine release study it was noted that many pine trees had formerly had dwarf mistletoe in their lower crowns. The infection had died out during stand development and ingrowth of Douglas-fir that increased stand density. The mistletoe had likely been prominent in the pine during the younger, open-growth phase, but was now of little evidence in old dead brooms. Thinning of the overall stand had lowered basal area from 160 to 100 ft^2 surrounding individual pine, and increased tree vigor indices by 60 percent in five years, although this is still to low compared to historic stand levels on a site like this. Average pine age was about 155 years. Current crown percent is also low, approximately 35 percent. Evidently, ingrowth of Douglas-fir adjacent to pine decreased dwarf mistletoe development as well.

Bob Tinnin, retired from Portland State University, OR—Sharon Stanton (Dept. of Biology at Portland State University) is investigating the effects of fire on ponderosa pine that is either infected or not with dwarf mistletoe. She will be examining test plots in Oregon and Arizona before and after prescribed burns looking at such things as fuel loads, fire performance, and fire effects on the pine. She will also examine wood from broomed and normal branches to see whether infection changes the fuel quality of branch wood. This work is being done in cooperation with the US Forest Service and the National Park Service.

VII. Genetics

Dennis Ringnes, USDA Forest Service, Region Five Genetics Resource Program, Camino, CA— Identification and genetic assessment of resistance to *Arceuthobium campylopodum* in ponderosa pine. Six ponderosa pine were selected for resistance to dwarf mistletoe from within a 38 year-old plot established to monitor the rate of disease spread (Wagner 1965). All six parents were grafted and established in the Badger Hill Breeding Arboretum in 1965 as part of a continuing effort in ponderosa pine tree improvement in Region 5. As these grafts began to produce cones, controlled pollination was done between the clones. In 1993, Paul Stover, Central Zone Geneticist, inquired with PSW about any future plans for these clones. This resulting project was proposed to develop efficient techniques and methodology for the selection and testing of ponderosa pine parents for resistance to dwarf mistletoe.

From 1993 to 1996, guidelines for selecting trees for potential resistance to dwarf mistletoe were developed and 109 candidates were selected on the eastside of the Plumas and Lassen National Forests, along with ten highly susceptible controls. Cones and scion wood were collected from most of these selections. In 1997, seedlings from 14 resistance candidates, 10 susceptible controls, and 20 control-crosses were outplanted at Badger Hill. The seedlings were planted in five test blocks, five seedlings per block (cross 366x302 only had seven plantable seedlings). Seedlings were planted in a five tree row plot design in Block 1 and were randomized in the other four blocks. In the winter of 1999–2000, the seedlings in Block 1 were inoculated with five dwarf mistletoe seeds per seedling. The other four blocks were inoculated during the following winter. Dwarf mistletoe seeds were placed at a needle fasicle on first year wood. Two additional inoculations have been done, applying 10 seeds per seedling in the winter of 2001–2002 and 20 seeds per seedling in 2002–2003.

Evaluation of the seedlings for dwarf mistletoe infections began with Block 1 in 2002. Infections were recorded and any plants were stripped from the branches to avoid unintended seed dispersal. In July 2003, all five blocks were evaluated for infections from the initial inoculations. Jay Kitzmiller ran a simple, one-way ANOVA on the data and provided the following input:

On the surface, the data appears to be taking some shape. However, the numbers have little meaning without, at minimum, standard errors of the means, and more desirable, a full analysis looking at normality of data, need to transform the counts or percent variation due to crosses, blocks, crosses by blocks, inoculation events (years), and unexplained error. At this point I think you are getting good separation between the extremes only, the best and the worst. I would have little confidence in those in-between the extremes at this point. It appears to me (without any analysis, other than the one-way ANOVA below), we will need nearly twice the data numbers in order to discriminate among the crosses and determine inheritance. Some data seem troublesome, for example 364x366 versus the reciprocal; also L-136-EL-C appears to be an outlier in its group; also 364 as female vs. male is highly variable. These kinds of things will need to be looked at closely to see if there is any reasonable explanation for them. Note the following table of simple one-way ANOVA (comparing group means) shows highly significant differences among groups, and fortunately they have very similar variances but with more than a two-fold difference in means.

ANOVA: Single Factor

SUMMARY

Groups	Count	Sum	Average	Variance
Grp 1	14	55.83333	3.988095	8.244206
Controls	10	74.07246	7.407246	7.724497
Grp 2	20	58.89147	2.944573	8.014835

ANOVA

Source of Variation	SS	df	MS	F
Between Groups	134.6517	2	67.32587	8.39074[a]
Within Groups	328.977	41	8.02383	
Total	463.6287	43		

[a] P-value=0.000882; F-critical=3.225679

More extensive analysis will be possible when the 2001–2002 inoculation cycle (ten seeds per seedling) is evaluated next year. Additional infections may also be found from the first inoculation cycle next year as well. During the summer of 2005, evaluation of the 2002–2003 inoculation cycle (20 seeds per seedling) will strengthen the analysis even more.

VIII. Management

James T. Hoffman, USDA Forest Service, Region Four, Boise, ID—Silvicultural Control, Dwarf mistletoe suppression efforts, including overstory removal, girdling, and sanitation thinning, will be conducted on 1,060 acres in the Intermountain Region (R-4) in 2003.

Brian Geils, RMRS, and Mary Lou Fairweather, FHP, Flagstaff, AZ—We re-measured the permanent plots Frank Hawksworth and Paul Lightle established at the South Rim of Grand Canyon in the 1950s. All areas had received some type of management including thinning, pruning, and/or prescribed burning. Although this is intended to be the close-out of the 1950s study, some of these plots will be used in a new study of dwarf mistletoe and prescribed fire interactions.

Simon Shamoun, Pacific Forestry Centre, and Bart van der Kamp, University of British Columbia, BC—Project Title, Biological and Genetic Control Approaches for Management of Dwarf Mistletoes. The overall objectives of the project are to survey and collect fungal hyperparasites and to understand their biology, ecology and explore their potential use as biological control agents for dwarf mistletoes. Currently the focus of this research program is on biological control of western hemlock and lodgepole pine dwarf mistletoes. Most recently, research efforts are underway to explore the use of genetic control method for management of western hemlock dwarf mistletoe.

Field trials. Virulent isolates of two candidate fungi for biological control of hemlock dwarf mistletoe (*Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc., a parasite of dwarf mistletoe shoots and fruit, and *Neonectria neomacrospora* (Booth & Samuels) Mantiri & Samuels, a parasite of mistletoe infected hemlock bark and possibly the endophytic system) were selected and tested for efficacy in a field trial at Spider Lake near Parksville. Conidia were produced in culture and applied in either a Stabileze formulation or in a sucrose-gelatin solution in late August. Mistletoe shoot, fruit and swelling characteristics were recorded before treatment and at 0.5, 1, 2, 3, 4 and 5 months after treatment. *C. gloeosporioides* reduced the current seed crop by more than 50 percent with virtually no difference between formulations. Analysis of shoot loss associated with *C. gloeosporioides* is in progress. *N. neomacrospora* produced sporodochia on the bark of wounded and inoculated DM swellings at significantly greater frequency than on parallel control treatments. It also caused a marked reduction in the number of healthy dwarf mistletoe shoots five months after treatment. Further work involving culturing and dissection is required to elucidate the mode of infection of *N. neomacrospora* infection.

Genetic resistance strategy for management of western hemlock dwarf mistletoe. Early results suggests levels of resistance to western hemlock dwarf mistletoe within western hemlock clones in conifer plantations in British Columbia. This study compares the susceptibility of hemlock from a broad range with the aim of identifying geographic trends, and partitioning of variation between provenance and family within provenance. This is intended as a preliminary trial to guide screening of hemlock seed orchard parents from Oregon, Washington and British Columbia with the purpose of allowing the creation of mistletoe-resistant seedlots. Six individuals from each of five open-pollinated families from ten sources were potted at age 1-year from seed, inoculated with the *A. tsugense* seeds collected from Woss, BC in the second year, and assessed for susceptibility at age four-years. Growth of mistletoe was not correlated with the growth of the host plant. Analysis of variance revealed significant variation in height at age-four by provenance, but no trends in susceptibility to infection were detected. Duncan's multiple range test identified one provenance as different for this trail. This research venture is a collaborative research effort with Charlie Cartwright, Hemlock Breeder, BC Ministry of Forests, Victoria, BC.

Biological control of lodgepole pine dwarf mistletoe. This project was part of a Ph.D. work conducted by Tod Ramsfield who completed his research work on May 24, 2002, under the direction of Drs. Bart van der Kamp and Simon F. Shamoun. Dr. Ramsfield joined Forest Research Institute as a Research Scientist (Molecular Forest Pathologist) at Rotorua, New Zealand.

Funding for this research project for the fiscal year of 2003–2004 continues by the provincial research initiative, BC Forestry Innovation Investment (FII): Biological Control of Hemlock Dwarf mistletoe. Don Robinson (ESSA Technologies, Vancouver, BC) has joined the project to develop a spatial statistical model to predict the outcome of mistletoe treatment with biological control agents. The model will also be enhanced to simulate biocontrol treatment effects on mistletoe survival and reproduction.

IX. Surveys

R. Mathiasen and C. Daugherty, Northern Arizona University, Flagstaff, AZ—Our manuscript submitted to the Western North American Naturalist reporting the results of our roadside survey for pinyon pine dwarf mistletoe (*A. divaricatum*) in the pinyon-juniper woodlands of the Coconino National Forest in northern Arizona has been accepted. It is scheduled for publication in the November issue. We surveyed 220 km of roads representing pinyon pine woodlands in 24 townships. Our results estimate that only about 12 percent of the area surveyed is infested with pinyon pine dwarf mistletoe. We are continuing our distribution study of pinyon pine dwarf mistletoe using GIS techniques and plan additional field work next summer.

John Muir, BC Ministry of Forests, Forest Practices Branch, Victoria—We are searching coastal western hemlock second-growth stands for suitable infestations of *A. tsugense* to measure spread and intensification. I have been trying to find stands that represent a range of factors such as stand age, tree species composition, and ecological features that appear to influence risk ratings. We also need suitable stands ideally with long-term data measurements to test projections of the detailed spatial mistletoe model being developed with Don Robinson, ESSA and Brian Geils, USFS, Flagstaff.

One of the major difficulties has been to find infected stands that demonstrate that hemlock dwarf mistletoe can have major impacts in second-growth forests. Recently, thanks to several suggestions of colleagues, I encountered two outstanding examples of stands with major infestations. Both stands are approximately 100 years age. One developed from selection logging (high grading) during the late 1880–90s. The other developed from a program of continuing selective cutting to remove defective Douglas-fir and other tree species, with no regard to dwarf mistletoe sanitation. Both stands appear to represent "new forest" conditions that are expected from new practices that restrict clearcutting and promote retention of numerous live trees. Both stands now have patches of lightly to severely infected trees, with gaps or stand openings developing in some of the most severely infected patches. Both are outstanding examples of how severe impacts of *A. tsugense* can develop in coastal forests on good sites. The major drawback is that both sites are in parks, presenting major difficulties in surveying and determining impacts!

Marcus Jackson, USDA Forest Service, Northern Region and Brennan Ferguson, State of Montana—We are re-measuring larch dwarf mistletoe plots established in 1991 (Taylor and others 1993) and re-measured in 1996 (Taylor and Marsden 1997) on the Flathead Indian Reservation. Taylor and others (1993) stated the study objectives:

1. Quantify the spread and intensification of dwarf mistletoe in western larch with and without overstory removal and precommercial thinning.

2. Quantify the growth effects due to dwarf mistletoe in infected western larch with and without overstory removal and precommercial thinning.

3. Provide visual demonstration of the treatment effects on stand growth and development.

4. Provide data for the validation of dwarf mistletoe models for stand conditions similar to those found in this study.

Little more than half of the 16 plots were re-measured in July 2003, before crews were not allowed further access to the area due to increased fire danger. We hope to complete re-measurement later this summer or fall.

Taylor, J., T. Reedy, and T. Corse. 1993. Permanent plots for studying the spread and intensification of larch dwarf mistletoe and the effects of the parasite on growth of infected western larch on the Flathead Indian Reservation, Montana. USDA For. Ser. Rpt. FPM-93-2. Forest Health Protection, Northern Region, MT. 13 p.

Taylor, J., M. Marsden. 1997. Permanent plots for studying the spread and intensification of larch dwarf mistletoe and the effects of the parasite on growth of infected western larch on the Flathead Indian Reservation, Montana: Results from the 5-year re-measurement. USDA For. Ser. Rpt. FHP-97-5. Forest Health Protection, Northern Region, MT. 5 p.

K. Marshall and D. Goheen, Southwest Oregon Forest Insect and Disease Service Center and D. Russell, Medford District, Bureau of Land Management—We completed data analysis of our retrospective case study of dwarf mistletoe broom development in mature Douglas-fir trees in southwest Oregon. The objectives of the project were to examine the relationship between broom characteristics and BVR (Broom Volume Rating), determine how long the trees had had the current level of infection and understand the sequence of initiation and spread of the brooms in crowns. We collected data from 331 Douglas-fir dwarf mistletoe brooms in 30 trees that were felled in conjunction with a timber sale on land managed by the Medford District, BLM.

Results of the data analysis showed a significant positive relationship between broom size and age. However, a few brooms grew large very quickly. For example, one Type-2 broom reached 789 ft^3 (about 9 ft on a side) in 17 years and one Type-3 broom reached 1287 ft^3 (about 11 feet on a side) in 13 years. This suggests that under the right conditions, brooms usable for nesting by wildlife could be developed in relatively short time. There was no significant relationship between the size of brooms and BVR, although average broom size decreased as the number of brooms increased. This suggests that managing for heavily infected trees with large numbers of brooms might not necessarily provide brooms that would be useful to wildlife. Type-3 brooms were significantly larger than Type-2 brooms, which were significantly larger than Type-1 brooms. Type-2 and -3 brooms were also significantly older than Type-1 brooms. This suggests that conditions that favored initiation of Type-2 and -3 brooms occurred earlier in the lives of the trees than conditions that favored Type-1 brooms. We hope the complete report will be available next year.

X. Modeling

John Muir, Jim Goudie, Ken Poulson, BC Ministry of Forests, Victoria, BC; Don Robinson, ESSA Technologies Ltd, Vancouver; Brian Geils, RMRS, Flagstaff AZ; Alan Thomson, Pacific Forestry Centre, Victoria, and Hamish Kimmins, UBC, Vancouver BC—We recently submitted a research proposal to develop a detailed model for A. tsugense to predict spread and impacts in second-growth western hemlock coastal forests. The approach is to use a spatial growth model (tree and stand simulator, TASS) that includes detailed equations on foliage and branch growth for western hemlock developed by Jim Goudie, to test and measure dwarf mistletoe effects. A detailed spatial model for dwarf mistletoe spread will be incorporated to predict spread and intensification under various ecological, site and stand factors. The detailed model should enable us to predict impacts of various new practices such as variable retention silviculture under a wide variety of conditions to determine stand and forest level impacts on growth and yield.

We suspect that many of the recently instituted forestry practices will maintain or exacerbate impacts of *A. tsugense*. However, with the current scarcity of tree growth data from long-term measurement of plots with dwarf mistletoe, the effects of current practices are almost impossible to demonstrate with any certainty or confidence. Mistletoe effects usually are not evident until young trees are at least 20 years age or older, and it is unlikely that trees of this age would be treated to mitigate impacts. If we can develop a mistletoe model as a component of a tree growth model that is widely accepted in BC, we should be able to estimate impacts of *A. tsugense* under variable conditions. Hopefully, these will provide a quantitative basis for determining effects and potential benefits of various forestry practices in infected stands. We also intend to use the model as input for larger-scale ecological models to predict effects on long-term forest succession and other ecological processes.

XI. Miscellaneous

Mary Lou Fairweather, FHP, Flagstaff, AZ—On the Mistletoe Center website visitors can view the most frequently asked questions page and submit their own question if they so choose. I am at the receiving end and just wanted to let you know that most of the questions pertain to true mistletoes. Although most people would like to lessen the infection in their trees, others would like to inoculate their trees and grow mistletoe for production and distribution.

Simon Shamoun—IUFRO working party 7.02.11 "Parasitic Flowering Plants in Forests" is continuing it's activities. Dr. Simon Francis Shamoun is the Coordinator for this group. This working party is preparing its web site under the IUFRO web site where it will post activities including future international meetings. For more information, please, contact Dr. Simon Shamoun at: SShamoun@pfc.cfs.nrcan.gc.ca; Phone: (250) 363-0766; Fax: (250) 363-0775.

Bob Mathiasen—continues his heroic quest for a male dwarf mistletoe plant exceeding one meter in length. The largest specimen he has found so far measures 92.8 cm!

Root Disease Committee Report
August 22, 2003, Grants Pass, OR

Blakey Lockman, Acting Chair

The Root Disease Committee met for breakfast on Friday, August 22, 2003. Over 30 people participated, which is remarkable since we met on the last day of WIFDWC following the evening banquet!! Blakey Lockman agreed to run the meeting and take notes in Ellen Goheen's absence.

Agenda Items

Update on Literature Review of Fertilization and Root Diseases

Ellen Goheen took the bull by the horns on getting this item rolling from last year's WIFDWC. She has purchased time from a librarian to do a search from a list of keywords. Presently, there are 400 pages of citations (lots of duplicates!). The list of citations is being narrowed down and added as appropriate. The goal is to have a draft of the annotated bibliography/literature review by this fall. Abstracts will be included. We applaud Ellen for making this happen!

Black Stain Project

Walt Thies is leading a project looking at interactions between black stain root disease and season of prescribed burn. The study location is near Burns, Oregon. There are spring and fall burns, 24 units with 4 different burns, and 5400 trees. An ecologist has done pre-burn observations, and post-burn plants will be recorded. They are trapping insects to try and determine the vector. Walt is doing post-mortem sampling. There are lots of folks working on this study. There will be 5-year and 15-year fires to follow. Walt has no extra dollars but is opening study up to others if they want to come and take data. There are also cow exclosures within the study area that will be in place for two years.

Round Robin

Terry Shaw—Involved in developing root disease model for European use of *H. annosum* model. There are number of challenges, not the least of which are language barriers. Terry is trying to get Ellen over to Vienna to do a workshop on the western root disease extension.

Greg Filip—Presently testing the western root disease extension in eastern Oregon; it seems to be "underestimating" mortality.

Bill Woodruff—Looking at black stain in California, specifically thinning and under-burning. Smoldering fires kill the roots. Also testing 3 thinning regimes—30 ft^2/acre, 60 ft^2/acre, and 90 ft^2/acre. Collecting beetles and sending to Matheo Garbellotto and Bill Otrosina for DNA analysis.

Bob Edmonds—Looking at fire and fire surrogates (thinning). Has a study in place with three repetitions of three treatments. Working with Paul Hessburg to look at post-fire insects.

Alex Woods—Looking at *I. tomentosus* and modeling. Trying to incorporate spore spread into the model. Will be looking at a 1996 stump trial where spruce was planted around stumps and then mapped.

Rona Sturrock—Working on *Phellinus weirii* resistance program. Evidence of genetic based resistance in coastal Douglas-fir. Seeing a great degree of variability between different isolates of *P. weirii*. Looking at terpene chemistry in general defense mechanisms of Douglas-fir.

Kristen Fields—Isolated *Armillaria gallica* from central Oregon. Not previously known to exist there!

Kathy Lewis—Studying *I. tomentosus* and soil moisture. Finding it more often at mid-slope. Looking at spruce beetle and root disease to quantify the root disease. Looking at *I. tomentosus* spread mechanisms and management. Also looking at *H. annosum* on Kenai.

Eric Smith—The western root disease extension of FVS is now linked with the westwide bark beetle extension. Working with Sue Hagle to improve the link.

Brennan Ferguson—Working with population structure of *Armillaria ostoyae* in western Montana. Recommending looking at Rick Kelsey's work on root disease making trees more attractive to bark beetles.

Bill Jacobi—In the midst of an *Ips* outbreak. On the old black stain plots, the *Ips* beetles are not picking out black stain trees.

Scott Kolpak—(Dorena Tree Improvement Nursery) Looking at resistance to *P. lateralis*. Wanting to map resistance.

Judy Adams—Working with the FFE model (Fire, Fuels Extension). It will be incorporated into the Basic FVS training session.

Brian Geils—Moving to a new project. Interested to know where root disease in thinned ponderosa pine might be predicted to become severe using information on soil moisture, soil types, etc.

Dave Schultz—High diversity of root disease in his area of California. Black stain around Mt. Shasta now gone, but now experiencing problems with *H. annosum*. Still recommending treating ponderosa pine stumps 16 to 18 inch diameter, and true fir stumps >=16 inch diameter. *H. annosum* found in 14 inch diameter pine stumps, but not causing disease centers. Not seeing *H. annosum* associated with shear stumps in pine—too small?

Don Goheen—Gave us some insight into the Port Orford Cedar EIS.

Hadrian Merler—Duncan Morrison and Mike Cruickshank are excavating stumps and hauling them from the site, which has caused a scare among some local folks! Reaching a large number of forest managers in their training sessions (about 400!).

We ran out of time before we could get to the last few people, but we almost made it all the way around the room. Our apologies to anyone who really wanted to say something and didn't get a chance to!

Business Meeting Minutes
51st WIFDWC 2003 Grants Pass, OR

Bart van der Kamp, Acting Chair for Everett Hansen

Previous Minutes—2002 Business Meeting Minutes are approved as circulated (moved by Walt Thies).

Treasurer Report—John Schwandt. We had 92 people register for this year's meeting (including 8 students, 7 retirees, and 77 regular members). We also had eight spouses/guests join us. For those that need it for travel, our Federal Tax Id. number is: #91-1267879. The following is a summary of transactions for the WIFDWC account.

Combined WIFDWC and Hazard Tree Committee Account (US$)

Transaction	Income	Expense	Balance
Total Account Balance as of 11/30/2002 (reported in previous Proceedings):			**5,293.33**
2003 WIFDWC meeting—Grants Pass, Oregon			
Registration	18,740.		24,033.33
Meeting setup, hospitality		760.(	23,273.33
Hotel costs: rooms, breaks, banquet, lunches		10,100.(	13,183.11
Field trip transportation & BBQ lunch		3,600.(	9,583.11
Outside speaker expenses		194.(	9,389.09
(For 2003 meeting alone, as of 12/31/2003, Income-Expenses =$4,289.78)			
Other Account Activity			
Awards		90.(	9,299.09
Printing/binding/mailing of 2002 Proceedings		1,485.8	7,813.27
Sale of old Proceedings	40.		7,853.27
Bank Interest/dividends/service charges	32.	12.(	7,873.61
WIFDWC Balance as of 12/31/2003:			**7,873.61**
Hazard Tree Committee (Balance as of 11/30/2002):			2,999.54
Hazard Tree Committee Meeting—Sacramento		578.8	2,420.66
Total Account Balance as of 12/31/2003:			**10,294.27**

Future Meetings—The 53nd WIFDWC in 2005 is to be hosted by Fred Baker and Jim Hoffman in western Wyoming. For the 54th WIFDWC in 2006, Alex Woods offered to host the meeting in Smithers, BC. The offer was accepted by acclamation.

Nominations

Nominating Committee—John Muir and others presented the slate for the 2004 Executive Committee. The nominees are Ellen Goheen for Chair; Blakey Lockman for Secretary; and John Schwandt for Treasurer. For the next meeting (a joint session with WFIWC in San Diego from April 26 to 30) John Klijunas will serve as preliminary contact for local arrangements. Judy Adams to remain as Webmaster. There being no other nominations, the chair declared the slate appointed by acclamation.

Awards Committee—As the most senior member, Greg Filip becomes committee chair. Don Goheen is added as a new American member, and a Canadian member will be appointed by the new Chair.

Old Business

Past Proceedings

Fred Baker reported on status of the project to publish past WIFDWC Proceedings on compact disk (CD). The Utah State University Library has offered to handle distribution, but final negotiations and approval are needed. Alternative pricing schemes for members and others were discussed. Terry Shaw offered the motion that Fred Baker and Treasurer John Schwandt develop a fair pricing schedule and arrange for distribution through Utah State University. The motion was debated and passed.

New Business

Honorary Life Members

Several retired members were noted as not having been formally recognized as Honorary Life Members (HLM). Mike McWilliams nominated Bob Gilbertson for HLM; Bob's nomination was approved. With discussion on the definition of "member in good standing" and need for standard procedures, further nominations for HLM were held. The motion was offered and passed that the Awards Committee review definitions and procedures for determining member status; that they report and recommend what actions should be taken.

Program

Interim Program Chair Alan Kanaskie asked for additional ideas and suggestions for organizing a program at a joint WIFDWC–WFIWC meeting (given the different formats of the two groups).

Resolutions Committee

In discussion, we recognized it may be useful to have a special committee to accept, prepare, and propose special resolutions on a timely basis for consideration of the membership. Bill Jacobi proposed (Diane Hildebrand seconded, and motion passed) that a committee be formed with an American member, a Canadian member, and a invitation to a Mexican member), that such committee would develop procedures for drafting and proposing special resolutions. Ellen Goheen announced that upon assuming the Executive Chair, she would appoint Terry Shaw as the Resolutions Committee Chair and direct him to include a Canadian member and if possible a Mexican member.

Western Plant Board

Susan Frankel proposed a motion that the Chair contact the Western Plant Board to develop a relationship between WIFDWC and the Western Plant Board and present issues of concern (such as movement of infected nursery stock causing forest diseases) to the Western Plant Board. The WIFDWC chair would also invite participation of Western Plant Board leaders in WIFDWC and encourage working together on areas of mutual concern. The motion passed.

Nursery Pathology Committee

Diane Hildebrand proposed recognition of a Nursery Pathology Committee and that it be chaired by Bob James. Motion passed.

There being no other business, the meeting was adjourned.

WIFDWC Outstanding Achievement Award

Award Criteria

Based on a vote at the Business Meeting, October 2, 1998

Purpose—recognize outstanding achievement in the field of forest pathology in western North America. The award will recognize the individual that has contributed the most to the field of forest pathology in Western North America.

Award—The award winner will be announced at the banquet. The awardee will present a keynote address at the following year's WIFDWC. A list of winners will be printed in the Proceedings. The winner will receive: a framed certificate and some sort of gift to be determined by the award committee. They will also become keeper of the social achievement award* hat, tie, etc., for one year or until the award is given again.

Selection Process—The award will be given annually. An Awards Committee composed of three WIFDWC members will determine the awardee. The Awards Committee will be selected annually by the WIFDWC Executive Committee. The Committee will be comprised of a representative from each of the following—a university researchers, a public agency employee, and one member-at-large. One member should be working in Canada.

Nomination Procedures—WIFDWC members may nominate another member for the award. they may not nominate themselves. An individual may only nominate one person per year. There is no formal nomination form but the following guidelines are provided (printed in the Proceedings, included in a WIFDWC mailing, and available on request from the chairman of the award committee):

- short introductory letter,
- narrative of nominees qualifications, educational background, work history, etc.
- letters of support from other individuals and organizations, and
- copies of a few of nominee's publications.

Nominations are due three months prior to the starting data of the next year's conference and should be sent to the chairperson of the Awards Committee.

The Awards Committee may decide not make an award if suitable candidates are not nominated.

Based on vote at the Business Meeting, August 16, 2000

Awards Committee—committee members serve a three-year term, with one old member leaving each year and one new member elected at the business meeting; committee members were elected for terms ending in 2000, 2001, and 2003 to establish the staggered replacement scheme.

Based on Discussion at the Business Meeting, August 16, 2000

Committee Recommendation—recipient should be a current or active member of WIFDWC, still active in forest pathology; awards should not be separated into research and non-research; recognizing two recipients in 2000 was a unique situation (not to be repeated) since the award had just been established and not given for the initial year.

Based on Business Meeting, October 9, 2002

Committee Chairperson—the most senior committee member automatically becomes the chairperson.

Awards Committee

Year[a]	Chair	Members	
1999	J. Byler	W. Littke	B. van der Kamp
2000	W. Littke	B. van der Kamp	R. Sturrock
2001	B. van der Kamp	R. Sturrock	G. Filip
2002	R. Sturrock	G. Filip	"unknown"
2003	G. Filip	D. Goheen[b]	S. Zeglan[c]

[a] Year elected
[b] Elected to replace "unknown"
[c] Appointed by Chair to filling remaining position.

* The social achievement award retired in 1997.

2003 Recipient: Everett M. Hansen

Nomination Narratives

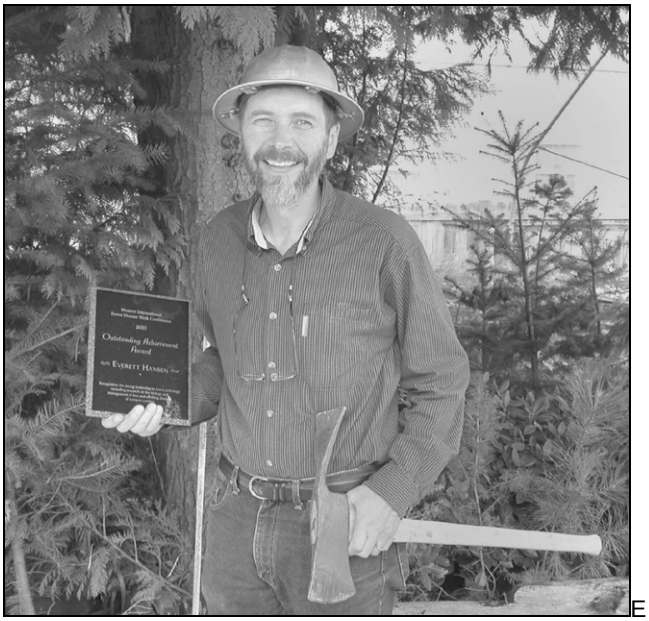
Everett Hansen is Professor of Plant Pathology at Oregon State University, Corvallis, Oregon. He holds joint appointments in the College of Agriculture, Department of Botany and Plant Pathology and the College of Forestry, Department of Forest Science. Everett moves easily between the worlds of teaching, basic research, and applied research. His research has covered all aspects of forest pathology including the biology and management of a variety of forest tree diseases and diseases of trees in nurseries. He has a special interest in population biology of forest fungi and is viewed as a world expert in this field. Everett has trained many graduate students, most of whom hold positions in forest pathology research and extension throughout the United States and in Canada. He has a gift for inspiring students to probe deeply into basic biology of forest pathogens while not losing sight of the practical challenges of management.

Everett Hansen has provided strong leadership in forest pathology research and management in the Pacific Northwest for close to thirty years. His work has been critical to our understanding of laminated root rot, black stain root disease, forest nursery pathogens, white pine blister rust, Swiss needle cast, Port-Orford-cedar root disease, and sudden oak death, among others.

Many very successful researchers are successful because they are narrowly focused and become the expert in that field. Not so with Everett—is success and brilliance come from his breadth, and his ability to link the highly focused, reductionist world of science, with the messy and highly variable real world. Everett sometimes complained about his inability to focus on one aspect of his research program—but I have seen that as a real strength.

Everett's research program has changed with the times—dramatically so. He has gracefully moved from classic studies in *Phellinus weirii* biology that used pulaskis and malt agar, to classic studies of *Phytophthora ramorum* biology involving molecular techniques. He comfortably spans old and new technology and ensures that technology is a means to an end, not the other way around.

As a graduate supervisor, Everett is exceptionally inspiring. He works hard, he recognizes the efforts of his students and willingly gives his valuable time. He is inclusive and brings his graduate students into his international world of research and interactions with other scientists. For all his success, he is pretty down to earth and accessible. He can laugh at himself and applaud others.



Everett Hansen accepting the 2003 Outstanding Achievement Award in front of Cold Frame 1, an important source of major gene resistance in Port-Orford-cedar to *Phytophthora lateralis.*

Past Outstanding Achievement Awards

Year [a]	Location	Recipient	Achievement Summary
2000	Waikoloa, HI	Lew Roth	For pioneering work on Phytophthora lateralis, Armillaria, and dwarf mistletoes, and for inspiration and leadership of a generation of plant pathology students and colleagues.
2000	Waikoloa, HI	Duncan Morrison	For long-standing contributions to forest pathology research, especially in root diseases and tree hazards.
2001	Carmel, CA	Robert Gilbertson	For contribution to the taxonomy and identification of wood-inhabiting basidiomycete fungi.

[a]No nominations received for 2002.

Bylaws of the Western International Forest Disease Work Conference

Passed by Vote of the Assembly at the Business Meeting October 2, 1998

Article I Objectives

The Western International Forest Disease Work Conference (WIFDWC) was formed in 1953 to provide a forum for information exchange among forest pathologists in western North America. The primary objectives of the organization are:

To exchange information on forest pests and related matters through periodic meetings and other appropriate means,

To promote education, research and extension activities in forest pathology,

To sustain and improve the health of western North America's forests.

Article 2 Membership

Membership is open to individuals who are engaged in forest pathology related endeavors in western North America. These include but are not limited to: research, survey, management, teaching or extension activities pertaining to tree diseases, forest health, or deterioration of forest products.

Western North America is defined as Canada: British Columbia, Yukon, Alberta, Manitoba, Saskatchewan; United States: Washington, Oregon, California, Idaho, Nevada, Utah, Arizona, Montana, Wyoming, Colorado, New Mexico, North Dakota, South Dakota, Nebraska, Kansas, Alaska, Hawaii, Guam, the Commonwealth of the Northern Marianna Islands and other Pacific Islands in Micronesia; and all of Mexico.

Membership is established after attending one Western International Forest Disease Work Conference. Members must attend another Western International Forest Disease Work Conference within 5 years or their membership is no longer valid.

~~Honorary members are WIFWDC members who have retired from continuous employment in the field of forest pathology. A list of honorary members will be published in the Proceedings of each meeting.~~ [replaced with text below at 2000 Business Meeting].

Honorary Life Membership will be automatically awarded to those members of WIFDWC (as defined above) who have attend at least 5 previous meetings of WIFDWC, and have retired from active forest pathology endeavors. Newly retired members who met this criteria should notify the current WIFDWC Chairperson of their status. Other members who have retired but do not meet the attendance criteria, or other outstanding contributors to the field of Forest Pathology, may request, or be proposed for Honorary Life Membership, by members present at an annual business meeting.

A list of Honorary Life Members will be published in the Proceeding of each meeting.

A 50% or more reduction in the registration fees for Honorary Life Members, to include a copy of the Proceedings, should be considered by the Executive Committee, as per Article7, amended in 1999. [as amended and passed at 2000 Business Meeting]

Article 3 Officers

WIFDWC officers will include a chairperson, secretary, treasurer, and historian. The chairperson and secretary will be elected by majority vote of the membership at the annual business meeting. If there is no majority, an acting chairperson will be appointed by the current chairperson. The tenure of the chairperson and secretary begins at the conclusion of the WIFDWC meeting where they were elected and ends when all business from their year's WIFDWC is completed. The treasurer, and historian will be elected every five years, to serve for the following 5 years.

Executive Committee

The Executive Committee may invite non-member speakers to the annual meeting and pay their travel expenses from conference registration fees [by motion passed in 1999, wording by B. Geils, 2004].

Duties of the Chairperson

At each WIFDWC, the chairperson will run the general and business meetings. The chairperson will appoint an interim program chairperson at the start of each WIFDWC to gather suggestions and opinions to guide the conference in the planning of next year's conference. The chairperson will also appoint three members to serve as the "railroad committee" to nominate candidates for next year's chairperson and secretary (and every fifth year, treasurer and historian). The chairperson may appoint members to assist in conducting the affairs of the Conference including but not limited to: local arrangements chairperson and program chairperson. The chairperson, secretary, treasurer and other appointees together form the executive committtee. The chairperson may also appoint ad hoc committees and their chairpersons as deemed necessary to assist in carrying out the mission of WIFDWC.

In the event that the chairperson cannot carry out their duties, the previous chairperson will carry them out. If other members of the executive committee cannot or will not carry out their duties the chairperson may appoint a replacement.

Duties of the Secretary

The secretary shall maintain the membership and mailing lists. The secretary shall send out meeting notices to the membership, take minutes at the business meeting, and compile and distribute the Conference proceedings.

The secretary will query all honorary life members to determine if they want to receive a free copy of the proceedings and only those responding in the affirmative will receive a copy. [by motion passed in 1999, moved from Article 9]

Duties of the Treasurer

The treasurer shall receive all payments, be custodian of WIFDWC funds, keep an account of all moneys received and expended, and make commitments and disbursements authorized by the chairperson. At the annual business meeting the treasurer shall make a fall report covering the financial affairs of WIFDWC. All funds, records and vouchers in the treasurer's control should be subject to inspection by the executive committee.

Duties of the Historian

The historian will keep a complete set of WIFDWC proceedings and answer any inquires as needed. The historian will contact the WIFDWC secretary and provide the address for mailing the archival copy of the proceedings.

Compensation

Officers will not be compensated for their services.

Non-liability of Officers

The officers shall not be personally liable for the debts, liabilities or other obligations of WIFDWC.

Article 4
Decision Making Process_____

The business meeting will be run by Roberts Rules of Order. Meetings are open to the public and non-members may participate in meetings. Only members may vote.

Decisions will be made by majority, with each member granted one vote. Votes may be called for at the annual business meeting. A quorum is reached when more than 25 members are present.

Article 5
Finances_____

Expenditures

The chairperson may authorize expenditures of WIFDWC funds. Checks, orders for payment, etc. may be signed by the treasurer, or other person designated by the chairperson. The executive committee may detemine which and how many, outside speakers they want to invite, and travel costs for such speakers can be paid from registration fees.

Contracts

The chairperson may authorize any officer or agent of WIFDWC to enter into a contract on behalf of WIFDWC. Unless so authorized, no person shall have any authority to bind WIFDWC to any contract.

Gifts

The chairperson or the treasurer may accept on behalf of the WIMWC any contribution, gift, or bequest. Commercial sponsorship of conference special events is not allowed.

Fiscal year

The WIFDWC fiscal year shall begin on the first of January and end on the last day of December.

Article 6 Bylaws

Amendments

Changes to bylaws shall be presented to all WIFDWC members for review. The by-laws maybe amended by a 2/3 majority vote, queried at a business meeting.

Article 7 Meetings

Frequency

The WIFDWC endorses holding annual meetings but will, on vote of the membership, change the time of any particular meeting when circumstances dictate that such action be taken.

Date

WIFDWC endorses holding meetings in late Summer but will, change the interval between any two meetings when circumstances dictate that such an action be taken.

Registration

Registration will be reduced by half, if possible, for graduate students and Honorary Life Members. It will be at the discretion of the WIFDWC Executive Committee for each meeting to offer a further reduction in fees to graduate students and Honorary Life Members and to offer a further reduced fees to others such as retired professional and visitors. [added by motion passed in 1999]

Article 8 Committees

The following are standing committees of WIFDWC: Hazard Trees Committee, Dwarf Mistletoe Committee, Root Disease Committee, Rust Committee, ~~Disease Control Committee~~ [disbanded 2002], *Nursery Pathology Committee* [established 2002].

Article 9 Proceedings

Papers for each year's proceedings must be submitted to the secretary by November 1 of the year of the meeting.

Distribution of proceedings is made to all paid registrants and honorary members who have indicated a desire to receive them and will be made available to others at cost. ~~The secretary will query all honorary life members to determine if they want to receive a free copy of the proceedings and only those responding in the affirmative will receive a copy.~~[by motion passed in 1999, moved to Article 3]

Select Motions and Decisions

1998

Outstanding Achievement Award—established.

1999

Honorary Life Members—members added and provisions discussed (see 1996 Proceedings for historic retrospective on HLM).

Assisting Outside Speakers—amendment passed.

Website—Committee Reports and Meeting synopsis by the Chairperson would be posted; web committee (Baker, Muir, and Adams) formed.

2000

Outstanding Achievement Award—staggered committee established and recommendations made.

Joint Meetings with WFIWC—motions passed to meet in 2004, have dual program chairs, form a planning committee in 2001 for the joint meeting.

2001

Disease Control Committee—proposal to reorganize tabled.

2002

Standing Committees—motion passed to disband Disease Control and establish Nursery Pathology Committee.

Bill Jacobi Holly Kearns Blakey Lockman Dave
Jenna Daniel

Mike Schmaker Dave Schultz Don Graham Greg Filip

Past Annual Meeting Locations and Officers

Meetings and Officers, 1953–1989

Annual	Year	Location	Chairperson	Secretary – Treasurer	Program Chair	Local Arrangements
1	1953	Victoria, BC	R. Foster			
2	1954	Berkeley, CA	W. Wagener	P. Lightle		
3	1955	Spokane, WA	V. Nordin	C. Leaphart	G. Thomas	
4	1956	El Paso, TX	L. Gill	R. Davidson	V. Nordin	
5	1957	Salem, OR	G. Thomas	T. Childs	R. Gilberston	
6	1958	Vancouver, BC	J. Kimmey	H. Offord	A. Parker	
7	1959	Pullman, WA	H. Offord	R. Foster	C. Shaw	
8	1960	Centralia, WA	A. Parker	F. Hawksworth	J. Parmeter	K. Shea
9	1961	Banff, AB	F. Hawksworth	J. Parmeter	A. Molnar	G. Thomas
10	1962	Victoria, BC	J. Parmeter	C. Shaw	K. Shea	R. McMinn
11	1963	Jackson, WY	C. Shaw	J. Bier	R. Scharpf	L. Farmer
12	1964	Berkeley, CA	K. Shea	R. Scharpf	C. Leaphart	H. Offord
13	1965	Kelowna, BC	J. Bier	H. Whitney	R. Bega	A. Molnar
14	1966	Bend, OR	C. Leaphart	D. Graham	G. Pentland	D. Graham
15	1967	Santa Fe, NM	A. Molnar	E. Wicker	L. Weir	P. Lightle
16	1968	Couer D'Alene, ID	S. Andrews	R. McMinn	J. Stewart	C. Leaphart
17	1969	Olympia, WA	G. Wallis	R. Gilbertson	F. Hawksworth	K. Russell
18	1970	Harrison Hot Springs, BC	R. Scharpf	H. Toko	A. Harvey	J. Roff
19	1971	Medford, OR	J. Baranyay	D. Graham	R. Smith	H. Bynum
20	1972	Victoria, BC	P. Lightle	A. McCain	L. Weir	D. Morrison
21	1973	Estes Park, CO	E. Wicker	R. Loomis	R. Gilbertson	J. Laut
22	1974	Monterey, CA	R. Bega	D. Hocking	J. Parmeter	
23	1975	Missoula, MT	H. Whitney	J. Byler	E. Wicker	O. Dooling
24	1976	Coos Bay, OR	L. Roth	K. Russell	L. Weir	J. Hadfield
25	1977	Victoria, BC	D. Graham	J. Laut	E. Nelson	W. Bloomberg
26	1978	Tucson, AZ	R. Smith	D. Drummond	L. Weir	R. Gilbertson
27	1979	Salem, OR	T. Laurent	T. Hinds	B. van der Kamp	L. Weir
28	1980	Pingree Park, CO	R. Gilbertson	O. Dooling	J. Laut	M. Schomaker
29	1981	Vernon, BC	L. Weir	C.G. Shaw III	J. Schwandt	D. Morrison R. Hunt
30	1982	Fallen Leaf Lake, CA	W. Bloomberg	W. Jacobi	E. Hansen	F. Cobb J. Parmeter
31	1983	Coeur D'Alene, ID	J. Laut	S. Dubreuil	D. Johnson	J. Schwandt J. Byler
32	1984	Taos, NM	T. Hinds	R. Hunt	J. Byler	J. Beatty E. Wood
33	1985	Olympia, WA	F. Cobb	W. Thies	R. Edmonds	K. Russell
34	1986	Juneau, AK	K. Russell	S. Cooley	J. Laut	C.G. Shaw III
35	1987	Nanaimo, BC	J. Muir	G. DeNitto	J. Beatty	J. Kumi
36	1988	Park City, UT	J. Byler	B. van der Kamp	J. Pronos	F. Baker
37	1989	Bend, OR	D. Goheen	R. James	E. Hansen	A. Kanaskie

Bylaws were amended in 1989 to split the office of Secretary–Treasurer.

Meetings and Officers, 1990–2003

Annual	Year	Location	Chairperson	Secretary	Treasurer	Historian	Program Chair	Local Arrangements	Web Coordinator
38	1990	Redding CA	R. Hunt	J. Hoffman	K. Russell		M. Marosy	G. DeNitto	
39	1991	Vernon, BC	A. McCain	J. Muir	K. Russell		R. Hunt	H. Merler	
40	1992	Durango, CO	D. Morrison	S. Frankel	K. Russell		C.G. Shaw III	P. Angwin	
41	1993	Boise, ID	W. Littke	J. Allison	K. Russell		F. Baker	J. Hoffman	
42	1994	Albuquerque, NM	C.G. Shaw III	G. Filip	K. Russell		M. Schultz	D. Conklin T. Rogers	
43	1995	Whitefish, MT	S. Frankel	R. Mathiasen	K. Russell		R. Mathiasen	J. Taylor J. Schwandt	
44	1996	Hood River, OR	J. Kliejunas	J. Beatty	J. Schwandt		S. Campbell	J. Beatty K.Russell	
45	1997	Prince George, BC	W. Thies	R. Sturrock	J. Schwandt		K. Lewis	R. Reich K. Lewis	
46	1998	Reno, NV	B. Edmonds	L. Trummer	J. Schwandt	D. Morrison	G. Filip	J. Hoffman J. Guyon	
47	1999	Breckenridge, CO	F. Baker	E. Michaels Goheen	J. Schwandt	D. Morrison	J. Taylor	D. Johnson	J. Adams
48	2000	Waikoloa, HI	W. Jacobi	P. Angwin	J. Schwandt	D. Morrison	S. Hagle	J. Beatty	J. Adams
49	2001	Carmel, CA	D. Johnson	K. Marshall	J. Schwandt	D. Morrison	A. Kanaskie	S. Frankel	J. Adams
50	2002	Powell River, BC	B. van der Kamp	H. Maffei	J. Schwandt	D. Morrison	F. Hennon	S. Zeglen R. Diprose	J. Adams
51	2003	Grants Pass, OR	E. Hansen	B. Geils	J. Schwandt	D. Morrison	H. Merler	E. Michaels Goheen	J. Adams

Bylaws passed in 1998 WIFDWC identify officers as chairperson and secretary elected at annual business meeting and treasurer and historian, elected every fire years.

Standing Committees and Chairs, 1994–2003

Committee	Chairperson	Term
Hazard Trees	J. Pronos	1994–2003
Dwarf Mistletoe [a]	R. Mathiasen	1994–2000
	K. Marshall	2001–2003
Root Disease	G. Filip	1994–1995
	E. Michaels Goheen	1996–2003
Rust	J. Schwandt	1994
	R. Hunt	1995–2003
Disease Control [b]	B. James	1995–2002
Nursery Pathology [b]	B. James	2002–2003

[a] F. Baker to succeed Marshall in 2004.
[b] Disease Control disbanded and Nursery Pathology established in 2002.

WIFDWC Honorary Life Members

Honorary Life Membership_____

Honorary Life Members (HLM) are WIFDWC members who have retired from continuous employment in the field of Forest Pathology. As of 2000, HLM status is granted to those members who have attended at least five previous meeting, retired, and notified the current WIFDWC Chair. Other retired members or contributors to the field of Forest Pathology may request or be proposed by members at an annual meeting HLM status.

An HLM list is published in the Proceedings. Honorary Life Members are entitled to a 50 percent reduction in registration fees (or more, at discretion of the Executive Committee) and the offer of a free copy of the Proceedings.

Honorary Life Members*

1956
Don Buckland (?D)

1959
Norman T. Engelhart (1967M)
John Hunt (1967M)

1960
Hans N. Hansen (1960D)
Albert Slipp (1959D)
Charles Waters (1960D)

1965
Lowell Farmer
Harold Offord (1992M)
Willhelm G. Solheim (1978D)
Willis W. Wagener (1969D)
Ernest Wright (1985M)

1966
Jesse L. Bedwell (1969D)
Warren J. Benedict (1992M)
Lake S. Gill (1969D)
John C. Gynn (1965M)
Homer Hartman (?D)
James W. Kimmey (?D)
James L. Mielke (1978D)
Virgil Moss (1998D)
Conrad Wessela

1967
John E. Bier (1967D)
Paul D. Keener (1967D)

1968
Thomas W. Childs (1998M)
Ross W. Davidson (1992M)
John Hansbrough (?D)
Clarence R. Quick (1988D)

1970
D. Reed Miller (1998M)

1971
Brenton Howard (?D)

1972
Thomas S. Buchanan (1990M)
Hubert H. Bynum (1972M)

1973
Stuart R. Andrews (1979M)
Paul Lightle
Wolf Ziller (?D)

1975
Richard T. "Dick" Bingham
David Etheridge
Ray E. Foster (2002M)
C. Donald Leaphart (1981D)
Jack Roff (?D)

1976
George Harvey (1985M)
Alex Molnar
Nagy Oshima (1976M)
Phil Thomas (?D)
Bratislav Zak

1977
Ed Andrews
Neil McGregor (?D)

1979
Lewis Roth

1980
Linnea Gillman
Donald Graham
Al Tegethoff

1981
Clarence C. Gordon (1981D)
Lee Paine

*Members are presented by year HLM status was conferred; deceased members are designated by year of memorial (M) or death (D).

1983
Elmer Canfield (2001D)
Dwight Hester (1985M)
Gordon Wallis

1984
Paul Aho
Mike Finnis
Charles G. Shaw (1998M)
Larry Weir (2000M)

1985
Robert Bega (?D)
Tommy Hinds (?D)
Thomas Laurent

1986
Oscar Dooling (1987M)
Jerry Riffle
James Trappe
John Woo (1998D)

1987
John Hopkins

1989
Alvin Funk
Neil Martin

1990
William J. Bloomberg (?D)
Richard B. Smith
Roy Whitney

1991
Frank G. Hawksworth (1993M)
Otis Maloy
John "Dick" Parmeter
Robert F. Scharpf
Stuart Whitney
Ed Wicker

1992
Roy Bloomstrom (1992M)
Charles H. Driver
Bob Harvey
Vidar Nordin

1993
Fields Cobb
John Laut
Arthur McCain
Earl Nelson

1994
Norm Alexander
Roger Peterson
Ralph Williams

1995
David W. French (2000M)
Ray Hoff
Tom Nicholls
E. Mike Sharon
Richard S. Smith

1996
James Ginns
Kenelm Russell
Jack Sutherland

1997
Tom McGrath
Pritam Singh
James L. Stewart
Allen Van Sickle

1998
Art E. Parker (1998D)

1999
John Hart
Eugene P. Van Arsdel
Ed Wood

2001
James Byler
David Johnson

2002
Leon Lamadeleine
John Palmer (?D)
Art "Doc" Partridge
Michael Srago

2003
Robert L. Gilbertson

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