Proceedings Of The Fifty-Third Western International Forest Disease Work Conference

Snow King Resort
Jackson, Wyoming
August 26–29

Many Thanks to Mister Megaphone, James Hoffman, your illustrious organizer par excellence. Photo by Angwin

Thanks everyone for your help, from your Region Four Host Team: Left to right, John Guyon, Laura Dunning, Valerie DeBlander and Phil Mocettini. Photo by Schwandt

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Ogden, Utah
April 2006

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1:00 - 5:00  Nursery Pathology Meeting, Chair Bob James--Jackson Room
5:00 - 7:00  Registration--Lobby
6:00-9:00  Social--Lobby

Tuesday, September 27, 2005

6:45-8:30  Breakfast Buffet for Group--Rafferty’s
8:00-12:00  Registration--Mezzanine level
8:30-8:45  Opening Welcome, Mary Lou Fairweather--Grand Room
8:45-9:30  Keynote Address: Bob James, 2004 Outstanding Achievement Award Winner
9:30-10:00  Break
10:00-10:30  Introductions
10:30-12:00  Regional Reports
12:00-1:30  Hazard Tree Committee Lunch, Chair John Pronos--Rafferty’s, then TBA
1:30-3:00  Panel: Current Issues in Forest Pathology, Moderator Ellen Goheen

Early Warning for Forest Health Threats: National and Regional Perspectives
Borys Tkacz, National Program Manager, Forest Health Monitoring, USDA Forest Service,
Forest Health Protection and Jerry Beatty, Center Director, Western Wildland Environmental
Threat Assessment Center, USDA Forest Service, Pacific Northwest Research Station

Caught in the Crossfire: Translating Phytophthora ramorum into Quarantine Policy
Susan J. Frankel, Sudden Oak Death Research Program Manager,
USDA Forest Service, Pacific Southwest Research Station

Towards a Better Biological Understanding: Seeking the Origin of Phytophthora ramorum
Ellen Michaels Goheen, Plant Pathologist, USDA Forest Service, Forest Health Protection

3:00-3:30  Break
3:30-5:00  Panel: Canker Diseases Near and Far, Moderator Diane Hildebrand

Nectria Canker of Radiata Pine
Margaret Dick, New Zealand Forest Research

Beech Bark Disease
Martin MacKenzie, USDA Forest Service Northeastern Area

Nectria Canker of Red Alder
Craig Cootsona, University of Washington

Fusicoccum arbuti on Pacific Madrone
Marianne Elliott, University of Washington
5:00-7:00  Poster Session Setup, Moderator Kristen Fields (klfields@fs.fed.us, 541-383-5587)
7:00-9:00  Poster Session, Reprint Swap, and Ice Cream Social--Teton Room

Wednesday, September 28, 2005

6:45-8:30  Breakfast Buffet--Rafferty’s
8:00-10:00  Panel: White Pine Blister Rust, Moderator Brian Geils

Epidemiology for Hazard Rating of White Pine Blister Rust
Eugene Van Arsdel (presenter), Brian Geils, and Paul Zambino, USDA–FS, Rocky
Mountain Research Station, Research Plant Pathologists

White Pines and Blister Rust in Western North America: Impacts, Spread, and Restoration
John Schwandt, USDA–FS, Northern Region, John Kliejunas, USDA–FS, Pacific Southwest
Region Blakey Lockman, USDA–FS, Northern Region (presenters), and John Muir, retired
from BC Ministry of Forests, Forest Pathologists.

An Assessment of White Pine Blister Rust in High Elevation White Pines in California
Patricia Maloney (presenter), University of California Davis, and Joan Dunlap, USDA–FS,
Placerville Nursery

Applications of Molecular and Genomic Techniques for the Identification and Characterization
of Rust Fungi and Other Forest Pathogens
Detlev Vogler, USDA–FS, Institute of Forest Genetics, Research Geneticist/Plant
Pathologist

Modeling the Potential Distribution of White Pine Blister Rust in the Central Rocky Mountains
Holly Kearns (presenter), USDA–FS, Northern Region, Forest Pathologist and William
Jacobi, Colorado State University, Professor

10:00-10:30  Break

10:30-12:00  Panel: Aspen, Moderator Mary Lou Fairweather

Browse History of Aspen and Implications for Response Following Wolf Reintroduction in the
Northern Winter Range of Yellowstone National Park
Roy Renkin, Management Biologist, Yellowstone National Park

Impact of the 2001-2003 Drought on Productivity and Health of Western Canadian Aspen
Forests
Ted Hogg, Research Scientist, Canadian Forest Service, Natural Resources Canada

Are the Changes in Aspen Forests in the Western United States a Forest Decline?
John Guyon, Forest Pathologist, USDA-FS, Forest Health Protection, Ogden Field Office,
Intermountain Region

12:00-1:30  Dwarf Mistletoe Committee Lunch, Chair Fred Baker--Timberline One Room

1:30-3:00  Panel: Past and Current Research on the Biology of Armillaria ostoyae in the Southern Interior
of British Columbia, Moderator Duncan Morrison

Fifteen Years of Research on Armillaria ostoyae: A Legacy of the Western Root Disease Model
Project and FRDA II
Duncan Morrison, Research Scientist (and adjunct professor), University of British
Columbia

Resistance in Western Redcedar against Armillaria ostoyae: Host Response to Infection and
Mortality Incidence in Juvenile stands in the Southern Interior of BC
Michelle Cleary, PhD Candidate, University of British Columbia
WIFDWC 2005 Program

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Determination of A. ostoyae Infection Events in Roots of Juvenile Douglas-fir, Subsequent Fungal Spread and Effects on Tree Growth
Mike Cruickshank, Research Scientist, Pacific Forestry Centre, Canadian Forest Service, Natural Resources Canada

3:00-3:30 Break

3:30-5:00 Panel: Forest Health Success Stories, Moderator Will Littke
Root Disease Research in Support of Forest Management: Some Recent Examples
Walt Thies, Research Plant Pathologist, USDA Forest Service, Pacific Northwest Research Station
Forest Pest Surveys: Changing Forestry in Manitoba
Fred Baker, Professor, Utah State University
Pest Management Prescriptions and Eagle Habitat Management
Will Littke, Weyerhaeuser Forestry

6:00-9:00 No-host reception and banquet--Grand Room
Special Presentation: Wolf Recovery in Wyoming-Reintroduction to the Present
Mike Jimenez, US Fish and Wildlife Service

Thursday, September 29, 2005

6:45-8:15 Breakfast Buffet for Group--Rafferty’s
Root Disease Committee Breakfast, Chair Ellen Goheen--Rafferty’s, then TBA
8:20-5:00 All Day Field Trip (group photos will be taken on the field trip)--Lobby

Friday, September 30, 2005

8:00-9:30 Business Meeting, Moderator MaryLou Fairweather--Lodge Room at the Pavilion
9:30-10:00 Break
10:00-11:30 Student Panel, Moderator Holly Kearns (hkearns@fs.fed.us, 208-765-7493)
Impacts of Magnesium Chloride Based Dust Suppression Products on the Health of Roadside Vegetation
Betsy Goodrich, Colorado State University
Classifying Blister Rust Cankers on Western White Pine: Can Canker Characteristics Predict Tree Mortality?
Amy Eckert, Oregon State University
Ecology of Fungal Endophytes in Douglas-Fir and Ponderosa Pine Roots in Eastern Washington
Amy C. Ramsey, University of Washington
Technology Transfer in Forest Science.
Amanda Crump, Colorado State University
Role of Armillaria spp. in Canopy Gap Formation in the Western Hemlock Zone of the Hoh River Valley, Olympic National Park
Nicholas J. Brazee, University of Washington
11:30-12:00 Discussion and Closing Ceremony
Welcome

Good morning and welcome to Jackson, Wyoming for the 53rd Western International Forest Disease Work Conference. I would like to extend a special welcome to those of you attending your first WIFDWC and also to retirees who made the trip this year. We encourage everyone to take part in the exchange of ideas and have a great time interacting with each other. New this year we will have everyone introduce themselves later this morning, before the Regional Reports.

The planning committee has done an excellent job of putting this event together and I would like to thank the individuals who put a tremendous amount of work into making this meeting happen:

First to our Local Arrangements Chair, our planner extraordinaire Jim Hoffman, who set up these lovely accommodations and sweated through the details and waited patiently for you all to register.

Thanks to Kelly Burns, Program Chair, for setting up an excellent agenda.

Thanks to John Guyon for taking over as secretary after Hadrian Merler stepped down. He hopes to receive all presenters’ papers by November 1st. Also, thanks to John Guyon for working with Jim on local details and fieldtrip planning.

And now we would like to give a special thanks to two veteran WIFDWC planning committee members, who do the same duties year after year. First, our Treasurer- John Schwandt. John’s most important attribute was that he constantly reassured Jim that we have enough money.

And thanks to our Webmaster and WIFDWC guru- Judy Adams. Judy not only makes sure all our updates get posted on the website, but she had all the practical answers for us during our conference calls because of having been involved with so many WIFDWC committees.

And now Jim Hoffman would like to honor a few of the Special Committee Chairs.

Remembrance

Before proceeding any further we would like to have a moment of silence for those members who died since our last meeting in San Diego.

Announcements

The first annual reprint exchange will occur at the poster session this evening. I hope you brought an extra suitcase. Brian Geils has brought copies of the 2003 proceedings to sell.

The CD of WIFDWC proceedings is also available.

Blakey Lockman is the interim program chair for the 2006 meeting in Smithers, Canada. Please give her suggestions for next year’s program.

The railroad committee has been recruited and will be looking for you/for someone to fill the 2006 WIFDWC chairperson and secretary positions.

A document about the recent loss of blister rust research positions with Rocky Mountain Research Station, at the lab in Moscow, Idaho, has been included in your information packet. One position is a research plant pathologist studying how the invasive blister rust functions in the northern Rocky Mountains. The second position is a research plant geneticist, studying how western white pine and whitebark pine respond to the rust. The third scientist has worked on canopy gaps. All three scientists were told their work is “mature science”, and therefore expendable. However, the Moscow lab has just recently discovered other alternate hosts of blister rust, Indian paint brush and lousewort. This decrease in emphasis on white pine research is a stark contrast to the recent emphasis placed on whitebark pine management by the Forest Health Protection Units. We will be discussing this document at the business meeting on Friday.

It is now time to turn the podium over to our keynote speaker for this meeting, Bob James, who is the winner of the 2004 WIFDWC Outstanding Achievement Award.
Keynote Address

Investigations of *Fusarium* Diseases within Inland Pacific Northwest Forest Nurseries

Robert L. James and R. Kasten Dumroese

Abstract – *Fusarium* spp. cause important diseases that limit production of seedlings in forest nurseries worldwide. Several aspects of these diseases have been investigated for many years within Inland Pacific Northwest nurseries to better understand disease etiology, pathogen inoculum sources, and epidemiology. Investigations have also involved improving disease control efforts to limit impacts. Major diseases caused by *Fusarium* spp. include pre- and post-emergence damping-off, root disease, stem cankers, and top blight. The major *Fusarium* pathogen of bareroot nurseries is *F. oxysporum*. It is a common soilborne species with pathogenic and nonpathogenic strains readily isolated from conifer seeds, diseased and healthy seedlings, and nursery soil. Pathogenic strains appear to be genetically distinct from common saprophytic strains. The major *Fusarium* pathogen of container nurseries is *F. proliferatum*. This species is especially adapted for rapid spread throughout greenhouses. Most tested isolates of *F. proliferatum* are highly virulent. Other *Fusarium* species commonly associated with seedling diseases include *F. solani*, *F. acuminatum*, *F. sporotrichioides*, *F. sambucinum*, and *F. avenaceum*. Several other *Fusarium* spp. are encountered infrequently. Most other *Fusarium* spp. are less virulent on conifer seedlings than *F. oxysporum* and *F. proliferatum*. Improved disease control has been obtained by reducing pathogen inoculum on seeds, reused containers, greenhouse environments, and within nursery soil. Biological control has not yet proven as effective as fungicides in reducing disease severity. Pathogens colonizing roots of seedlings are usually replaced by other mycoflora following outplanting on forest sites. Efforts to reduce dependence on pre-plant soil fumigation have been successful in some, but not all bareroot nurseries. Keeping fallow fields free of plants with periodic cultivation for at least one year may often be as effective as fumigation. Conversely, growing cover crops, incorporating organic amendments into soil, and rotating seedling production with *Brassica* green manure crops have usually proven unsuccessful because of stimulation of *Fusarium* populations following addition of organic matter to soil. Future research efforts should involve using molecular probes to quantify pathogenic *Fusarium* populations to allow better prediction of requirements for implementation of disease controls.

Introduction

Diseases caused by *Fusarium* spp. within forest nurseries have been investigated in North America for nearly a century. However, they still severely impact production of high-quality seedlings at many nurseries. Most previous investigations focused on diseases in bareroot nurseries and very little was known about disease epidemiology or procedures to reduce disease impacts in container nurseries. Therefore, we began investigating diseases associated with *Fusarium* spp. more than 20 years ago. Our goal was to understand disease etiology, sources of pathogen inoculum, and various aspects of disease epidemiology specific to greenhouse environments. This information would lead to developing improved disease control.

Production of container seedlings within greenhouses has greatly increased in western North America during the past two decades. Seedlings can be grown in shorter time periods to provide reforestation stock resulting from changing demands from land managers. Seedling sizes, especially root systems, can be tailored to particular specification requirements. As such, seedling stock may be more adaptable to forest site differences, thereby improving the performance of outplanted seedlings.

Types of Diseases

*Fusarium* spp. commonly infest conifer seeds, particularly contaminating outer seedcoats (Axelrood and others 1995; James 1985b, 1986a, 1987c, 1999). This inoculum allows rapid colonization of young, succulent seedling tissues upon seed germination which may result in both pre- and post-emergence damping-off (James 1986a, 2004; James and Genz 1981; James and others 1991). Although seedlings are usually susceptible to damping-off for only short time periods, losses can be extensive, particularly if seedlots are extensively contaminated with...
populations of *Fusarium* (Hoefnagels and Linderman 1999; James 1986a; James and others 1991). *Fusarium oxysporum* is the most common conifer seed-contaminating *Fusarium* species; other common seed colonizers include *F. solani*, *F. acuminatum*, and *F. sambucinum* (James 1987c; James and others 1991).

Root diseases caused by *Fusarium* spp. can occur throughout the life of the crop. Within bare-root nurseries, most mortality occurs during the first growing season (James 1987b, 2001b, 2002d), with little or no mortality associated with root disease occurring during the second or subsequent years. With container seedlings, however, the highest root disease mortality often occurs at the end of the growing cycle when seedlings are stressed to stop growing and initiate bud formation (James 1986a; James and Gilligan 1984; James and Perez 1998; James and others 1987). Stresses induced by reduced watering and restriction of nutrients can cause seedlings to begin to show disease symptoms. Many of these seedlings were infected with potential pathogenic *Fusarium* early in their growth cycle (James and Gilligan 1988a; James and others 1987) but disease did not appear because seedlings were provided with plenty of water and optimum fertilization. As seedlings become stressed, colonizing *Fusarium*, which previously acted like non-pathogenic root endophytes, begin to induce tissue necrosis and disease symptoms subsequently appear.

*Fusarium* spp. can potentially cause stem cankers, particularly in dense bare-root seedbeds within coastal nurseries where high humidity prevails (Hansen and Hamm 1988; James 1986c, 2003b). These cankers are either produced low on the stem, just above the ground line, or higher in the seedling near foliage. Incidence and severity of stem cankers vary from year to year and may be related to weather influences as well as level of soilborne inoculum (Hansen and Hamm 1988; Hansen and others 1990; James 2003b).

Top blight of container seedlings associated with attack by *Fusarium* is usually rare. Ponderosa pine (*Pinus ponderosa*) is the most susceptible conifer species to this type of damage (James 1992a, 2003a), probably because seedcoats may persistently remain attached to cotyledons. Seedborne inoculum may result in cotyledon infection with subsequent colonization of stem and root tissues resulting in seedling mortality.

### Major *Fusarium* Pathogens

Fungi in the *Fusarium oxysporum* species complex are the most important *Fusarium* pathogens in bare-root forest nurseries (James 2004; James and others 1991, 1996b, 2000). These organisms also occur on container seedlings, particularly as a result of seed contamination (Dumroese and others 1988; James 1987c; James and Genz 1981). Organisms classified as *F. oxysporum* may actually comprise several genetically distinct species (Gordon and Martyn 1997; Kistler 1997). Pathogenic and nonpathogenic strains are common within forest nurseries and usually appear very similar morphologically. Pathogenicity tests have indicated a wide range of potential virulence on conifer seedlings under controlled conditions (James and others 2000). Genetic markers may be useful in separating pathogenic from nonpathogenic isolates (Donaldson and others 1995; Gordon and Martyn 1997; Stewart and others 2004).

Isolates of *F. oxysporum* commonly reside in nursery soil and can remain viable for prolonged periods because of production of long-lived chlamydospores and sclerotia (James and others 1991; Nelson and others 1983). These fungi are excellent colonizers of soil organic matter, regardless of its source (Hansen and others 1990; James 1988b, 2002a; James and others 1996b, 2004a, 2004b, 2004c). These organisms also occur on container seedlings, particularly as a result of seed contamination (Dumroese and others 1988; James and others 1991, 1996b, 2000). When susceptible seedling crops are sown in infested soil, seedlings can become readily infected following stimulation of chlamydospores by host root exudates (James and others 1991; Schippers and others 1981). Once infected, seedlings can exhibit root disease symptoms, probably due to infection by virulent isolates. However, some infected seedlings may remain infected but not become diseased (James and Gilligan 1988b). In this case, *F. oxysporum* acts more like a nonpathogenic root endophyte than a true pathogen. Pathogenic isolates usually induce cortical cell necrosis resulting in root decay (James 2004; James and others 1991), and may readily colonize most root and stem tissues. Seedling mortality occurs when decayed root systems no longer function.

In container seedlings, the most important *Fusarium* pathogen is usually *F. proliferatum* (James 1997a; James and others 1995a, 1997). This *Fusarium* species is taxonomically placed in the section *Liseola*, which includes other important pathogens such as *F. circinatum*, cause of pitch canker of pines (Gordon and others 2001). *Fusarium proliferatum* is not commonly found on conifer seeds, nor is it a very common nursery soil inhabitant (James 1987c, 1997a, 1998a, 1998b, 2002b). This species does not
produce chlamydospores that would allow long-term survival (James 1997a; Nelson and others 1983). However, it is an excellent colonizer of organic matter and may colonize a wide range of plants without inducing disease symptoms (James 1997a; James and Gilligan 1988a; Nelson and others 1983). This species spreads rapidly throughout greenhouses, primarily from microconidia that are readily produced in chains (James 1997a; Nelson and others 1983). Roots of container seedlings may become infected by *F. proliferatum* when seedlings are relatively young (James 1997a; James and others 1987). However, they may or may not exhibit disease symptoms. Production of disease symptoms is primarily related to level of seedling stress (James and others 1990a). Most tested isolates of *F. proliferatum* exhibited high virulence (James and others 1986, 1989, 1997) under controlled pathogenicity tests that utilize a perlite-cornmeal-potato dextrose agar inoculum (James 1996; Miles and Wilcoxin 1984). Therefore, these fungi are capable of causing serious losses to conifer seedling crops grown in greenhouses. In some cases, entire crops have been lost to this pathogen, resulting in extensive economic consequences. However, healthy appearing seedlings can have roots that are extensively colonized by *F. proliferatum* by the end of the growth cycle (James and Gilligan 1988a). When seedlings with *Fusarium* root colonization are outplanted on forest sites, *Fusarium* spp. gradually declines on roots systems and are usually replaced of other mycoflora (Dumroese and others 1993a; Smith 1967).

Several other *Fusarium* spp. are encountered in forest nurseries. Those most commonly isolated include *F. solani*, *F. acuminatum*, *F. sporotrichioides*, *F. sambucinum*, and *F. avenaceum*. Several of these are routinely isolated from roots of diseased or healthy appearing seedlings (James and Gilligan 1988a, 1988b; James and others 1991), nursery soil (James 1984, 2002b, 2002c), peat-based container growing media (James 1985a, 2005), conifer seeds (Dumroese and others 1988; James 1987b), and the inner walls of reused styrofoam or plastic containers (Dumroese and others 1995; James 1989b, 1992b, 2001a). Tests to evaluate potential virulence of some of these species indicated that they are mostly saprophytes, although selected isolates may be capable of eliciting disease on conifer seedlings (James 2000b; James and Perez 1999, 2000). It is possible that some of these other *Fusaria* contribute to disease when associated with more aggressive pathogenic species such as *F. oxysporum* and *F. proliferatum*.

**Fusarium Disease Management**

Successful management of *Fusarium* diseases in forest nurseries requires emphasis on prevention (James and others 1990a, 1995b). Most approaches are designed to reduce inoculum of potential pathogens within and near seedling growing areas. In addition, it is important to maintain the vigor of nursery seedlings by proper manipulation of water and fertilizer (James 1997b; James and others 1990a). If pathogen inoculum is reduced to a minimum and seedlings are kept vigorous, *Fusarium* disease losses are usually small.

Seedborne inoculum can be greatly reduced by treating seeds with running water rinses or surface sterilization chemicals (James 1985a, 1986a, 1987a; James and others 1996a; Lock and others 1975). This type of treatment may be preferable prior to stratification because *Fusarium* spp. are known to spread throughout seedlots during prolonged stratification (Axelrood and others 1995; James 1987a, 1987c; Kliejunas 1985). Some chemical seed treatments are routinely used for particular conifer species (Sutherland and van Eeerden 1980; Wenny and Dumroese 1987). However, care must be taken to ensure that chemicals do not adversely affect seed germination or emergence of young germinants (Dumroese and others 1988; James 1986a; James and others 1995b; Pawuk 1979). The most effective and least potentially harmful treatment is exposing seeds to running water rinses for at least 48 hours during which seeds are periodically agitated (James 1985b, 1987a; James and others 1996a). This water treatment greatly reduces fungi, including pathogens, which commonly contaminate seedcoats.

Container seedlings are usually produced within either plastic or styrofoam containers. Because these containers are costly, several seedling crops must be grown within individual containers. As a result, potential pathogenic fungi, especially *Fusarium* spp., often contaminate the inner walls of reused containers (James 1989a, 2001a; James and others 1988). This inoculum presents an important potential hazard to subsequent seedling crops. Procedures for cleaning reused containers have changed over the years. Initially, containers were either washed superficially to remove most residual organic matter or steam treated. These treatments, however, were ineffective in significantly reducing populations of potential pathogens. Chemical treatments such as sodium hypochlorite (James and Sears 1990), sodium metabisulfite (Dumroese and others 1998), or coating containers with copper solutions (Dumroese and others 1995) were more effective, but...
were hazardous to use or resulted in chemical disposal problems. Immersion in hot water (at least 68°C for 30 seconds) became the most widely used, effective container treatment (Dumroese and others 2002; James 1982b; James and Eggleston 1997; James and Woollen 1989). However, high costs of energy required to maintain sufficient water temperatures, stimulated investigations into effective alternatives. Tests of radio frequency waves (James and Trent 2001), dry heat (James and Trent 2002), and large capacity steam rooms (Trent and others 2005) indicated that alternatives to hot water immersion are not only effective, but may be less expensive.

Cleaning greenhouse interiors (walls, floors, benches) with surface sterilants between seedling crops is necessary to reduce inoculum of potential pathogens, including *Fusarium* spp., which tend to accumulate within greenhouses during typical crop cycles (James 2004; James and others 1990a, 1995b). In addition, if greenhouse floors are not made of concrete, weeds growing within houses may harbor important pathogens (James and Gilligan 1984; James and others 1990a) and should be periodically removed. Sanitation procedures, such as periodically removing diseased plants, also helps reduce disease impacts (James 2004).

Soil is a major source of potential pathogen inoculum in forest nurseries. Several *Fusarium* spp. are particularly well adapted to maintain high populations in nursery soil. The most effective and widely used technique for reducing soil pathogens is pre-plant soil fumigation with non-specific biocide chemicals (Hansen and others 1990; Hildebrand and others 2004; James 1989a; James and Beall 1999; James and Ziedler 2004; James and others 1999b, 1996b). Such treatments are very expensive. Although potential pathogens are killed by soil fumigation, all other microorganisms, including beneficial and potential biocontrol fungi and bacteria, are killed as well. Therefore, once a nursery begins soil fumigation, growers usually have to repeat treatments prior to sowing each subsequent seedling crop (James 1989a; James and Beall 2000).

Biological control has potential for reducing *Fusarium* disease losses in forest nurseries. Several commercial biocontrol products are available with proven efficacy against *Fusarium* (James and others 1993). Unfortunately, most tests of fungal (Dumroese and others 1996; James 200a; Mousseaux and others 1998) and bacterial (Dumroese and others 1998) preparations in controlling diseases have so far been disappointing. This may be because tested formulations were developed for specific agricultural crops rather than for use in forest nurseries. Perhaps biocontrol formulations of effective pathogen competitors that routinely occur in forest nurseries, such as nonpathogenic isolates of *F. oxysporum* (James 2000a), may be more effective.

Most growers still rely heavily on applications of chemical fungicides to control many nursery diseases, including those caused by *Fusarium* spp. For some parts of the seedling growing cycle, such as the damping-off stage, chemical treatments are usually quite effective. However, to control root diseases of older seedlings, drenching soil or container media with fungicides has generally been ineffective (James 1984, 1988, 1998a; Shrimpton and Williams 1989). Incorporating chemicals into growing media prior to sowing container seedlings has also been ineffective (Dumroese and others 1990). Although *Fusarium* spp. are generally sensitive to particular fungicides, such as thiofanate methyl, treatments may not result in sufficient chemical concentrations reaching infected roots. In addition, most fungicides are good at preventing diseases rather than acting therapeutically (James 1988) so that treating seedlings already extensively colonized by potential pathogens has little value.

**Alternatives to Pre-Plant Soil Fumigation**

As indicated above, pre-plant soil fumigation has become widely used in many bareroot nurseries in the United States. Traditionally, the chemical of choice for fumigation is a mixture of methyl bromide (67%) and chloropicrin (33%). However, methyl bromide was recently identified as an important contributor to the destruction of stratospheric ozone (Hildebrand and others 2004; James and others 1994). As a result, it was programmed for reduction and eventual elimination as a soil fumigant and other applications worldwide. Although the deadline for use of methyl bromide in the United States was January 2005, exemptions have been allowed for industries for which no other viable alternatives exist. Some forest nurseries have applied for these exemptions.

When we first learned of the phaseout and eventual elimination of methyl bromide as a soil fumigant, we began a series of trials at different bareroot nurseries to evaluate potential alternatives. Our goal was primarily to find effective alternatives to chemical soil fumigation altogether rather than finding a replacement chemical fumigant. We found that fallowing fields for a least one year (preferably 2-3
years) with periodic cultivation to keep soil aerated, remove weeds, and expose propagules of potential pathogens can be as effective as chemical soil fumigation (Hildebrand and others 2004; James and Ziedler 2004; James and others 1996b). Fallowing is especially effective at nurseries with well-drained loam soils containing limited amounts of clay (James and Beall 2000). The beneficial effects of fallowing, however, can be eliminated by incorporating organic matter into soil, either as a direct amendment or incorporating a green manure crop (James 1998b; James and others 1996b). Fusarium populations greatly increase within soils to which high amounts of organic matter have been added (Hansen and others 1990; Hildebrand and others 2004). This effect occurs even if the green manure crop is a Brassica spp. (James and others 1996b, 2004a, 2004bg, 2004c), which is grown and incorporated into soil because residues produce methyl isothiocyanate, the active ingredient in some chemical soil fumigants such as dazomet (Smolinska and others 2003). Apparently, the amount of added organic matter overrides the potential toxicity to resident pathogens afforded by Brassica tissues.

Our investigations indicated that some nurseries can effectively replace methyl bromide by prolonged fallowing of fields or substituting with another chemical fumigant, such as dazomet (James and others 1996b). However, other nurseries have yet to have effective alternatives to methyl bromide and will probably use this chemical as long as the law allows. One additional alternative is conversion of most seedling production to containers, which has been used effectively throughout much of Canada.

**Future Directions**

Over the century we have learned a great deal about Fusarium diseases in forest nurseries. Losses to these fungi are now usually much less in both bareroot and container nurseries than previously. However, these diseases are usually still present at some level in most seedling crops and disease losses may become unacceptable on occasion. When new inexperienced growers are employed at nurseries or when new container facilities are installed, initial disease losses may be quite high. As growers become more experienced, they learn how to prevent diseases more effectively and losses from Fusarium usually decline over time.

Plant pathology entered the era of molecular biology several years ago. As a result, many plant diseases are often routinely diagnosed using molecular probes that can be developed for specific strains of particular pathogens (Gordon and Martyn 1997; Kistler 1997; Summerell and others 2003). Preliminary work with F. oxysporum in forest nurseries (Donaldson and others 1995; Stewart and others 2004) indicates that these tools have great potential application. If the pathogenic strains of this important fungus can be easily identified and quantified in nurseries, growers can more accurately predict expected disease levels and make appropriate adjustments to implementing control measures. Identifying saprophytic strains that are genetically different from pathogens opens the possibility of using these strains as more reliable biological control agents because they behave similarly, i.e., occupy the same niches, to pathogens.

More emphasis on cultural manipulations rather than chemical pesticides will be needed in the future. Many standard chemical fungicides used at high levels in the past are either currently or will shortly be unavailable in forest nurseries. Environmental concerns, such as worker exposure and groundwater contamination, are important considerations for the future of nursery disease management. More benign, but efficacious, treatments will be required in the future. Biological control has an important part to play in the future of nursery disease management. Evaluating new commercial products as they become available will be important.

As nursery growers reach the end of their careers, it is important that their replacements become knowledgeable about disease management. Successful disease management often evolves over time at particular nurseries and much of this important information can be lost to new growers. Continued support for nursery pathology is an important way to ensure that disease management information remains available to growers in federal, state, tribal, and private nurseries. This “extension” role is very important for agencies mandated to provide pathology assistance. Hopefully, nursery pathology assistance will continue to receive high priority.

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Photo by DeNitto
Early Warning System for Forest Health Threats

National Perspective
Borys M. Tkacz

Introduction

This paper describes the Early Warning System for forest health threats developed by the USDA Forest Service and partners. This national system is a comprehensive framework for early detection and rapid response to environmental threats to forest lands in the United States.

What is the Early Warning System?

Significant increases in human travel and trade have resulted in increased threats to forest ecosystems from the introduction of invasive species. Concurrently, natural forest ecosystems have become less resilient to disturbance events due to human-influenced changes. To ease this mounting pressure from environmental threats, we need enhanced protective measures provided by a coordinated national early warning system.

The authority for developing a coordinated national Early Warning System (EWS) is contained in Title VI of the Healthy Forests Restoration Act of 2003: which states that the Secretary of Agriculture “shall develop a comprehensive early warning system for potential catastrophic environmental threats to forests to increase the likelihood that forest managers will be able to:

1. Isolate and treat a threat before it gets out of control.

2. Prevent epidemics such as the American chestnut blight in the first half of the twentieth century, which could be environmentally and economically devastating to forests.”

The issues to be addressed by the EWS include: “insects, diseases, invasive species, fire, and weather-related risks and other episodic events”. In early 2004, a team from USDA Forest Service (FS) and partner federal and state agencies developed a roadmap to the development of the EWS and documented their recommendations in a web-based report: “The Early Warning System for Forest Health Threats in the United States.”

(http://www.fs.fed.us/foresthealth/ews)

What is the goal of EWS?

The goal of the EWS is “to prevent damage from new threats and to reduce to acceptable levels the impacts of existing threats.” The EWS was described as a conceptual framework to ensure that a comprehensive approach is taken when dealing with environmental threats.

Major objectives include:

- Developing logical, systematic framework for detection of environmental threats;
- Identifying gaps, weaknesses, and redundancies;
- Focusing research efforts to enhance detection and response;
- Highlighting opportunities for increased cooperation and collaboration;
- Facilitating timely, well coordinated response efforts.

What are the components of EWS?

The major components of the EWS are found in Figure 1.
Figure 1.

**EWS Steering Committee**

Implementation of the EWS is being coordinated by the EWS Steering Committee, an interagency, cross-Deputy Area committee that provides strategic vision and coordination among the various programs involved in the EWS. Its objectives are to:

- Develop strategic direction and priorities;
- Identify and remedy existing gaps in knowledge, processes, or linkages;
- Look for opportunities to streamline by enhancing collaboration and reducing redundancies.

The EWS Steering committee is working to identify critical next steps, develop specific EWS action plans, and identify short and long-term funding strategies and commitments. The members of the committee are given in the Appendix.

**Conclusions**

The conceptual framework of the EWS aids in ensuring that a comprehensive approach is taken when dealing with environmental threats and that all relevant resources are brought to bear upon the problem at hand. The EWS has many potential applications, including education, analysis, improvement, planning and decision-making. Through improved coordination among partners, the EWS will enable us to prevent damage to ecosystems from new threats and reduce the impacts of existing threats to acceptable levels.

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**Appendix**

**EWS Steering Committee**

Ralph Thier, USDA FS/ Forest Health Protection (Chair)
Borys Tkacz, USDA FS/ Forest Health Monitoring
Greg Reams, USDA FS/ Forest Inventory and Analysis
Frank Sapio, USDA FS/ Forest Health Technology Enterprise Team
Don Duerr, USDA FS/ Early Detection Rapid Response Program
Gregg DeNitto, USDA FS/ Wood Import Pest Risk Assessment Team
Mary Ellen Dix, USDA FS/ Vegetation Management and Protection Research
Susan Stewart, USDA FS/ Fire & Aviation Management
Gary Mann, USDA FS/ International Programs
Doug Powell, USDA FS/ Ecosystem Management Coordination
Coanne O’Hern, USDA Animal and Plant Health Inspection Service/Cooperative Agricultural Pest Survey
Osama El-Lissy, USDA APHIS/ Invasive Species and Pest Management
Eric Norland, USDA Cooperative State Research Education and Extension Service
Terry Cacek, US Department of the Interior /National Park Service
Rick Tholen, USDI/Bureau of Land Management
Bob Rabaglia, Formerly of Maryland Department of Agriculture
Jerry Beatty, USDA FS/ Western Wildland Environmental Threat Assessment Center
Danny Lee, USDA FS/ Eastern Forest Environmental Threat Assessment Center
Lucinda Riley, Department of Homeland Security/Agricultural Inspections
The Mission of the Western Wildland Environmental Threat Assessment Center

Jerry Beatty

The mission of the Western Forest Environmental Threat Assessment Center is to generate, integrate, and apply knowledge to predict, detect, and assess environmental threats to public and private forests and grasslands of the west, and to deliver this knowledge to managers in ways that are timely, useful, and user friendly. WWETAC is part of the larger USDA Forest Service Early Warning System (Figure 1).

Objectives

- Identify gaps in knowledge related to assessing cumulative risk of multiple interacting disturbances.
- Quantification and analysis of the cumulative risk from multiple interacting threats using probabilistic models to predict the likelihood of severe or uncharacteristic disturbances.
- Assessments of the range of potential ecologic and socioeconomic consequences of severe or uncharacteristic disturbances.
- Delivery of Center products through a variety of user-friendly transfer mechanisms useful to land managers including narrative analysis, summaries, graphics, and maps in sufficient lead time for agencies to take preventive actions.

The Center will provide the following for the Western United States at multiple spatial and temporal scales:

- Syntheses of existing knowledge and ongoing efforts to forecast the timing, potential extent, and severity of environmental threats.

Objectives

- Evaluate the effects and consequences of multiple interacting stresses on western forest health.
- Increase knowledge and understanding of the risks, uncertainties, and/or benefits of multiple environmental stresses on western ecological conditions and socioeconomic values.
- Provide science-based decision support tools for policy formulation and land management in the western United States.
- Provide land managers with credible predictions of potential severe disturbance in the West with sufficient warning to take preventative actions.

The Center will provide the following for the Western United States at multiple spatial and temporal scales:

- Syntheses of existing knowledge and ongoing efforts to forecast the timing, potential extent, and severity of environmental threats.

What makes the threat assessment center unique?

- Inclusive – examines a wide spectrum of environmental threats.
- Integrated – looks at how the composite is more than the sum of its parts.
- Predictive – focused on possible futures more than current conditions.


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 Sentinel – focused on raising awareness, not developing treatments or solutions.
Collaborative – represents an unusual partnership.

Where might we focus first?

- Synthesis and data mining.
- Early warning systems.
- Build a conceptual framework for integrated risk assessment.
- Forensic meta-analyses.
Caught in the Crossfire: Translating a Plant Pathogen into a Quarantine; *Phytophthora ramorum* in the USA

Susan J. Frankel, Kerry Britton, and Steven W. Oak

**Extended Abstract**—In September 2005, at the request of Congressman Pombo, the Government Accountability Office conducted a review of government’s response to three invasive species: *Phytophthora ramorum*, emerald ash borer and Asian long-horned borer. The review asked the USDA (United States Department of Agriculture) Forest Service to reply to several questions including: Are there lessons that can be applied to better manage ongoing or future invasions? What follows are our personal opinions, offered in a constructive spirit:

1. **Build communication networks in advance.** *P. ramorum*’s outbreak occurred on lands of mixed ownership. To coordinate a response swiftly it is essential that foresters, regulators, and local government officials get to know each other prior to an outbreak, understand each other’s concerns, responsibilities and authorities. Exercises in contingency planning for hypothetical outbreaks of pathogens or insects, on private, state or federal lands would be very helpful. To facilitate working together for invasive species response the USDA Forest Service, Forest Health Protection and the National Association of State Foresters produced a workbook in 2004 to bring people together to prepare. ([http://www.stateforesters.org/pubs.html#Reports](http://www.stateforesters.org/pubs.html#Reports)). Even where personal acquaintance with counterparts lubricates the cogs of interagency communication, such facility can be easily lost by changing personnel assignments. It would be very helpful to institutionalize the communication circuit for reporting and developing strategies to deal with new pests.

2. **Communicate the importance of forests.** Forest pathogens and insects are regulated on the state and federal level by agricultural departments. The client base, priorities, and abilities of the agricultural departments may be very different from those required to assess, suppress or eradicate a pathogen or insect on forest lands. The agricultural departments are pulled in many directions with west Nile virus, Newcastle disease, soybean rust, mad cow disease, and many other important and dificult problems. An outbreak of a forest problem in a wildland setting may be lower on the list of their priorities, but be very important to forest managers and landowners. Also, the mission of agriculture departments is to promote agriculture. This mission can conflict with the need to adopt and implement restrictive measures to control an insect or pathogen on forest lands.

3. **Resolve broad underlying issues.** Regulatory delays occurred due to conflicts between federal and state regulatory agencies on fundamentals of pathogen quarantine design, such as whether it is legal to regulate pathogen propagules. A multi-disciplinary forum proactively sponsored by the National Plant Board, American Phytopathological Society Public Policy Board, National Plant Diagnostic Network and others could clarify fundamental issues of pathogen regulation and develop general guidance.

4. **Broaden the use of experts.** The concept of a new pest advisory group should be expanded to include technical experts, regulatory experts, and ensure inter- and intra-agency cooperation and communication. It would be helpful to have a cadre of experts to provide advice and guidance for early detection and other monitoring, eradication and suppression, communication needs, diagnostic tool development, regulatory needs, ethics, and funding sources.


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5. **Foster collaboration.** Continue to foster communication and partnering within USDA agencies: Animal and Plant Health Inspection Service (APHIS), Agricultural Research Service (ARS), Forest Service and Cooperative State Research, Education and Extension Service (CSREES), and cultivate partnerships with other federal agencies, states, local governments, communities, non-profits and others.

6. **Staunch the flow.** The high volume of international plant material shipments, susceptibility of many popular horticultural plants, and evidence from Europe that the pathogen moved from infected nursery rhododendrons to the forest suggests that shipments of nursery stock or other plant material may have inadvertently introduced *P. ramorum* to the United States. Investigations into the possibility that *P. ramorum* originated on imported nursery stock are complicated due to minimal tracking of imported plants and movement of the plants once in the U.S. USDA APHIS records international imports, but not electronically. The data forwarded to State inspection agencies is too general to allow personnel to prioritize inspections upon arrival. Furthermore, movement within the U.S. is not recorded. Consequently, once plant material passes through a port city, its ultimate destination and propagation history are not known. Interstate shipments of nursery stock are not tracked. Nursery plants are often handled by brokers, with plants being moved efficiently from one location to another. Stock is often started in one state, grown in another, and shipped to yet another state or country at maturity for sale. The European Union has initiated a plant passport system to track stock from the original place of production to the final consumer. The United States should investigate the successes and pitfalls of this system, and try to develop something similar.

The threat of insect and pathogen introductions via imported nursery stock is increasing because nursery stock production is moving offshore. Before the 1970s, plants for planting were allowed in lots of less than 100, and were imported as new varieties or species for testing and development. Today plants are produced and marketed worldwide. In 2005, APHIS issued an Advance Notice of Proposed Rulemaking for Plants for Planting (7 CFR part 319 and 7 CFR part 360). However, issues surrounding free trade concerns will greatly inhibit the development of strict import regulations. The International Plant Protection Convention has been asked to develop an international standard; a process which will take several years. Pathologists should participate actively in the public input to these regulations.

7. **Reduce red tape.** On private lands, it is difficult for state and federal agencies to carry-out suppression projects without regulatory agency authority and support. Liability issues, slow contracting processes, requirements for environmental impact documentation, and diffuse or uncertain responsibility may become obstacles.

8. **Build capacity.** There has been a steep decline in the number of forest pathologists and entomologists nation-wide. When Sudden Oak Death was first noticed in 1995 there was not a single research forest pathologist employed in California, including Forest Service Research Stations and universities. Furthermore, technical experts aren’t well-trained or well-suited to many aspects of emergency response. Public affairs, legislative affairs, ethics, and emergency response training sessions would be helpful.

Photo by Angwin
Towards a Better Biological Understanding: The Search for the Origin of *Phytophthora ramorum* in Yunnan Province, People’s Republic of China

Ellen Michaels Goheen

Phytophthora ramorum, a previously unknown pathogen, was first seen in Europe on ornamental nursery stock in the early 1990’s (Werres et al. 2001). It has since been implicated in the death of thousands of oaks (*Quercus agrifolia* and *Q. kelloggii*) and tanoaks (*Lithocarpus densiflorus*) in the western United States (Rizzo et al. 2002). Populations of *P. ramorum* in Europe and the United States are largely distinct from one another, and the organism is considered invasive and introduced in both locations (Brasier et al. 2004). It is possible that *P. ramorum* may have originated somewhere in Asia and was unknowingly transported on commercial or privately collected ornamental plants. Asia is a center of diversity for many genera of plants that are *P. ramorum* hosts. More specifically, northern Yunnan Province in the People’s Republic of China has an abundance of oaks and tanoaks in a variety of forest ecosystems. Rhododendrons and azaleas, among others, have been widely collected in this region for at least 150 years by plant enthusiasts from across the globe. Furthermore, the climatic conditions in this part of the Province correlate well with those predicted to be favorable for *P. ramorum*.

The International Forestry, Forest Health Protection, and Vegetation Management and Protection Research staffs of the Forest Service, U.S. Department of Agriculture, have been working cooperatively with forestry officials in China over the past several years on a variety of issues related to forest insects and pathogens. In August 2004, Tom Kubisiak (Research Geneticist, USDA Forest Service, Southern Institute of Forest Genetics) and I traveled to Yunnan Province in southwestern China to begin collaboration with Zhao Wenxia, (Plant Pathologist, Chinese Academy of Forestry (CAF), and Deputy Director, Research Institute of Forest Ecology, Environment, and Protection) on projects investigating forest *Phytophthora* species in China with the more immediate goal of initiating a survey specifically for the pathogen *P. ramorum*.

We considered our 2004 effort to be a pilot or initial study where we could identify a strategy for a larger future survey. We visited four forest sites where a variety of species of *P. ramorum* host genera occurred. Three sites were in Diqing Prefecture in the vicinity of the city of Zhongdian (Shangri-la) in northwestern Yunnan Province. Plant associations on these sites included evergreen oak (*Quercus*), *Larix*, and *Picea* stands with *Rhododendron* understories, evergreen oak and pine (*Pinus*) stands, and pine forests with *Lithocarpus* species and other oak species in the understory. Sites ranged in elevation from approximately 2000 m to 3500 m. Genera of other *P. ramorum* hosts were also present including *Lonicera* and *Rosa*. We also visited a site outside of the city of Kunming in central Yunnan province. Forests there were composed of several *Quercus* species with *Lithocarpus* species and *Rhododendron* species in the understory.

We observed foliar and dieback symptoms similar to those associated with *P. ramorum* in the US and Europe on a variety of plants. Symptoms included inner bark necrosis, shepherd’s crooking of small diameter stems and new growth, leaf tip dieback, leaf margin necrosis, and leaf spotting. Symptomatic tissues were collected in the field, plated on CARP media in the evenings after collection, and then transported to the CAF pathology laboratory in Beijing for sub-culturing and DNA extraction. In Beijing, mycelium and plant tissues contained within a single plate were pooled together and DNA was extracted using a standard CTAB-based extraction protocol. We also extracted DNA from an additional 30 symptomatic plant tissue samples. The DNA assay performed while in Beijing was inconclusive and DNA samples were brought back to the U.S. All DNA samples were further purified and amplified using the ITS 6 and 7 rDNA primers developed specifically for use on Oomycetes. Out of 92 total samples, 29 samples amplified at least a single visible...
Phytophthora ramorum was not immediately identified from these samples.

In August 2005 I traveled back to Yunnan Province with Zhao Wenxia. Joining us were Everett Hansen, Professor of Forest Pathology, Oregon State University, and Nik Grunwald, Research Plant Pathologist, USDA Agricultural Research Service. Our 2005 sampling strategy was to spend as much time in the field as possible and to include water and soil sampling along with examinations for symptomatic vegetation. We concentrated our efforts in forests of NW Yunnan with a wide variety of Rhododendron species. We traveled with “field friendly” ELISA-based Phytophthora diagnostic kits, hundreds of plates of selective media, a small microscope, water filtration equipment, and apples for soil baiting. Wherever and whenever possible, Phytophthora field sampling methods were shared with Chinese scientists and forest protection staff.

Our itinerary included visiting forest ecosystems that included evergreen oak (Quercus), Larix, Tsuga, Abies, and Picea stands in various mixes with Rhododendron understories, evergreen oak and pine (Pinus) stands with Rhododendron understories, sites dominated by tree-sized Rhododendron species mixed with conifers, as well as open meadow low shrub Rhododendron species-dominated sites. Sites ranged in elevation from approximately 3000 m to 4700 m. Genera of other P. ramorum hosts were also present. We also visited portions of the Kunning Botanical Garden in Kunming (elev 2000 m) where dozens of species of Rhododendron and Camellia are grown for public display and propagation. By projects end, close to 300 water, soil, and plant samples were processed. Phytophthora species were readily identified from several water samples. Phytophthora ramorum was not identified from samples or cultures prior to our returning to the US. All sample cultures currently remain in China at Zhao Wenxia’s laboratory.

There are many challenges associated with a continued effort to find the origin of Phytophthora ramorum. Even with a commitment to more intensive field sampling in our 2005 effort one cannot say that the forests of NW Yunnan have been adequately surveyed for P. ramorum. Access is difficult; the wildlands are generally remote with few local laboratory facilities available for post-field culture work. Obviously building the skills for baiting, culturing, and identifying Phytophthora species in China is critical to our continuing to work there, and we do have plans to host a Chinese scientist in Oregon to continue technology transfer. Even so, the work to be done sometimes seems daunting. The forests we visited in Yunnan Province were remarkably similar in genera, yet profoundly different in species diversity and composition relative to forests on the west coast of the US where P. ramorum is currently found. Symptoms similar to those caused by P. ramorum on wildland and nursery stock target species (Quercus, Lithocarpus, Rhododendron) in the western United States, as well as minor P. ramorum hosts (such as Lonicera, Smilacina), were abundant. Is our focus on known host genera reasonable? Is it if we make an effort to pay close attention to other plant species and surrounding soil and water but the subtle effects of a native Phytophthora species may be easily overlooked. Other ecosystems exist within and outside of Yunnan that may offer similar opportunities. One may even question our approach. Should we concentrate our efforts on pathways rather than place(s) of origin and make a more conscious effort to investigate how and what plant materials are moving? If so, we have much to learn about the distribution, extent and practices associated with the nursery trade in Yunnan or other rural settings in China and beyond.

Without doubt, however; understanding the origin of a presumably introduced pathogen, such as P. ramorum, will help us better understand its biology, ecology, pathology, and genetics, and may lead to management actions that minimize its impact.

References


Photos by Angwin
**Nectria fuckeliana Infection of Pinus radiata in New Zealand: Research Approach and Interim Results**

Margaret Dick, Lindsay Bulman and Patricia Crane

Abstract—Stem malformation, typically developing after pruning, has become a problem in some Pinus radiata plantations in part of the South Island of New Zealand over the last 10 years. Infection through the pruned branch stub may result in extensive stain and decay within the stem, although tree crowns remain green and healthy. *Nectria fuckeliana*, a Northern Hemisphere fungus commonly recorded there as a saprophyte or weak pathogen of species of *Picea* and *Abies*, is the most commonly isolated fungus from affected trees. *Nectria fuckeliana* had not been recorded in New Zealand prior to 1996. Interim results from trials established to determine the effect of silviculture, stub treatment and environment on disease development indicate that pruning in winter results in more infection than summer pruning and that incidence is related to stub size. Aspects of the basic biology of the fungus such as spore production, dispersal and germination are being studied, with results so far indicating that *N. fuckeliana* is able to function over a broad range of conditions.

Introduction

*Pinus radiata* D. Don (Monterey pine) comprises 90% of the 1.8 million hectares of plantation forests (which cover 7% of the land area) in New Zealand. Selected as the primary plantation species for its fast growth, *P. radiata* typically has a rotation length of 25-30 years. Annual rings may reach over 25 mm in trees grown on high index sites and per tree recoverable volume averages approximately 2.4 m³. Pruning within the first 8 years is widely undertaken, and although an added cost to forestry operations, the practice enables the production of a clear-wood butt log within the short growing cycle. Any infection or malformation of the butt log can result in sufficient degrade to relegate the log from a high return end-use to a lower value product, and it may become uneconomic to harvest the log at all.

In New Zealand, *P. radiata* has a history of stem infection through branch stubs by *Diplodia pinea* (Desm.) J. Kickx f. (syn. *Sphaeropsis sapinea* (Fr.) Dyko & B. Sutton), causing the disease ‘Diplodia whorl canker’. Effects range from minor sapstain through extensive degrade to tree death following invasion of the stem at the whorl; the hyphae in the tissue effectively block water transport up the stem. The epidemiology of this disease was thoroughly studied during the 1980s (Chou 1987; Chou and MacKenzie 1988), resulting in effective recommendations for management based on the scheduling of pruning operations during cool, dry weather. In the mid 1990s an increasing incidence of stem malformation after pruning was reported in some plantations in the lower South Island in spite of following the Diplodia whorl canker management recommendations. External symptoms were identical to those caused by *D. pinea*, although tree death was rare and only associated with stem breakage at infected whorls. Wilt of the crown did not occur. A species of *Acremonium* was isolated from discolored sapwood and subsequently, with the formation of perithecia on the surface of branch stubs and on stem cankers, *Nectria fuckeliana* Booth was identified as the primary colonizer of cankered sapwood. This was the first record of *N. fuckeliana* in New Zealand.

*Nectria fuckeliana* is recorded in the Northern Hemisphere as a wound invader of conifers. In Europe and Scandinavia it is one of the most common inhabitants of wounded Norway spruce (*Picea abies* (L.) H.Karst.) stems, occurring in both stained and unstained wood (eg Huse 1981; Roll-Hansen 1962; Roll-Hansen & Roll-Hansen 1979, 1980, Vasiliauskas & Stenlid 1998). Other species of *Picea* and also of *Abies* are common hosts. In a 27-year-old stand of mixed *Picea* species in Scotland, substantial stem cankering associated with wood colonization by *N. fuckeliana* was recorded 3 years after a pruning and brashing operation (Laing 1947).
In North America, *N. fuckeliana* is usually found as a saprophyte on logging slash or fallen trees of both *Picea* and *Abies* species. It has also been occasionally isolated from dead or dying leaders of young *Picea glauca* (Moench) Voss, *P. mariana* (Mill.) Britton and *Abies balsamea* (L.) Mill. (Smerlis 1969). Its ability to act as a pathogen to a number of conifer species, including five species of *Pinus*, was demonstrated in inoculation tests (Smerlis 1969; Ouellette 1972). *Nectria fuckeliana* was associated with cankers of white fir (*Abies concolor* (Gordon) Lindl. ex Hildebr.) in southern Oregon and northern California (Schultz and Parmeter 1990), and pathogenicity of the fungus was established with inoculation tests. In overstocked stands some suppressed trees were killed within a year of inoculation, whereas vigorous trees often contained the infection, and callus tissue developed over the injured area. There are no published records of natural infection of *Pinus* spp. from the Northern Hemisphere.

To increase understanding of this little-known disease in New Zealand, a number of trials have been established and a number of surveys undertaken. The immediate imperative has been to find an operational management strategy that will minimize the establishment of infection. The ultimate goal of this research is to understand the disease epidemiology, to enable informed decisions about internal quarantine and movement of forest products, and to prevent spread of the disease to other parts of New Zealand.

**Distribution and host range of *Nectria fuckeliana* in New Zealand**

Perithecia of *N. fuckeliana* have been observed and collected only in the southern part of the South Island. However, fruit bodies of *N. fuckeliana* do not form on all trees and cankers nor does ‘fluting’ typical of *N. fuckeliana* occur throughout the country. Therefore a broad national survey was undertaken to determine whether *N. fuckeliana* is present in forests outside the known infected regions. Where stem fluting was present, 80-100 mm long increment cores were taken 10-20 mm above the affected branch stub. Cores were surface sterilized in 10% hydrogen peroxide for 3-5 min, cut into sections, and placed on Petri plates containing 2% malt extract agar for incubation at 20°C. This technique has proved a satisfactory method of evaluating sapwood infection without the necessity of felling trees. A total of 202 stands were inspected and fluting was recorded in 27 (13.4%). *Nectria fuckeliana* was not isolated from any of the samples taken.

An intensive survey within the known infected area was conducted where individual trees in over 600 plots were assessed for fluting. Of the 12,300 trees assessed, 25% had some form of fluting. Follow-up surveys and detailed analysis relating fluting intensity with environmental factors are in progress. A delimiting survey over the extension of the known infected area and beyond, to determine the distribution and extent of the fungus, is also underway.

*Nectria fuckeliana* has only been recorded from *P. radiata* in New Zealand. Although a number of species of *Picea* and *Abies*, the primary hosts overseas, have been tested in New Zealand, none have found favor for plantation forestry. Of the conifers other than *P. radiata* that are grown commercially, Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) comprises just over 6% of the plantation estate, with a mixture of species from several genera making up the remaining 1.8% of plantation softwoods. Six species of conifers other than *P. radiata* have been inoculated with *N. fuckeliana* in a pilot trial to determine susceptibility of sapwood to colonization and any tendency to canker formation. These are *Pinus contorta* Loudon, *Pinus ponderosa* Douglas ex C.Lawson, *Pseudotsuga menziesii* (Mirb.) Franco, *Cupressus macrocarpa* Hartw. ex Gordon, *Sequoia sempervirens* (D.Don) Endl. and *Larix decidua* Mill. Results of the inoculations should be available within two years.

**Ecology and epidemiology of *Nectria fuckeliana***

**Early stages of infection**

Although *N. fuckeliana* is the most commonly isolated fungus from affected trees, the relationship of the fungus to the disease is not completely understood. The nature of tree susceptibility to invasion requires further investigation. The *Acremonium* anamorph forms on the surface of cut infected wood and is produced in culture, but the primary inoculum in nature is thought to be the ascospores (Vasiliauskas and Stenlid 1997). An inoculation experiment to determine whether there is a difference in aggressiveness of these two spore states in causing disease is in progress. The early stages of infection will also be observed in inoculated trees after felling at regular intervals. Examination of
infected wood by confocal and scanning electron microscopy will show the location of the fungus within the tissues of the tree and the type of cellular damage it causes.

**Spore production and dispersal and relationship of disease to weather conditions**

Knowledge of the basic biology of *N. fuckeliana* will allow forest managers to prune trees when infection risk is low. In New Zealand, viable ascospores are present in the perithecia in all seasons of the year. They also remain viable within the perithecia for many months of storage at room temperature or 4°C. Therefore it is important to determine the conditions of their release into the environment. Preliminary laboratory and field observations show that the ascospores ooze out of perithecia under wet conditions. They germinate readily over a broad range of temperatures, with the optimum between 18 and 25°C. The ascospores can be successfully trapped using microscope slides coated with Vaseline (methods based on Ostry and Nicholls 1982). Weekly spore trapping is underway in two locations. Meteorological data will be correlated with the trapping results. It is hoped that these experiments will help to pinpoint the conditions under which infection is most likely to occur.

**Survival of *N. fuckeliana* in debris**

As it is common practice to thin the forests to waste rather than to extract thinned stems, large volumes of woody debris cover the forest floor in the years following thinning and pruning operations. With the invasion of decay fungi and the breakdown of wood by cerambycid larvae (primarily the native *Prionoplus reticularis* White) the material breaks down in less than 10 years. However the potential contribution that infected debris may make to inoculum buildup is unknown. It is also possible that material, such as firewood or poles taken from forests may carry living *Nectria* within the wood or as fruiting bodies on the surface. While the fungus remains confined to one geographical region we will attempt to prevent spread into new localities through restrictions on the movement of wood. An understanding of the behavior of *N. fuckeliana* in woody debris may indicate ways to influence infection levels through reducing inoculum buildup. The objectives of this project are to a) determine the potential for thinning and pruning debris to become infected and subsequently provide an inoculum source; b) determine the length of time *N. fuckeliana* survives saprophytically in logs from trees that were infected while standing; and c) the longevity of fruit bodies and their ability to produce ascospores once the trees have been felled.

**Managing the disease**

**Pruned stub trial**

Concurrently with the ecological and epidemiological studies of *Nectria* behavior, a trial to identify silvicultural options that will limit infection has been established. As for the other trials discussed, this work is still in progress and therefore only research concepts and interim results can be reported here.

Cankers can invariably be traced to a wound site and there is no evidence that *N. fuckeliana* can invade intact stems. Pruning wounds are the primary entry point. Altering the timing of pruning operations to a period when fungal inoculum is not available, or to when tree susceptibility to fungal invasion is low, could substantially limit the incidence of pruned stub infection. Colonization of the sapwood, with subsequent decay by other fungi, may be stimulated by a separate suite of contributing factors. In addition to the season of wounding or pruning, variables affecting colonization could include the surface area of injury, its depth and height above ground level, and the length of time since injury occurred.

A trial designed to examine some of these variables was established in 2003. A standard pruning operation was carried out in summer and again in winter. Wound inoculation was carried out for comparison with natural infection. The trial will test when, and for how long, stubs are susceptible to infection, the effect of treating stubs with a physical barrier/fungicide combination, and the effect of wound size on infection and canker formation.

**Plot layout:** Twenty-six plots were established within 2 treatment blocks, one plot of each of the 13 treatments within each block. Treatments (Table 1) were randomly allocated within blocks. This layout was replicated on 2 sites (ridge top and valley). Plots were 400 m² and contained enough trees to select 12 trees with similar height, DBH and branching characteristics from the approximately 40 trees in each plot. This gave a total of 48 trees per treatment, with 24 trees per site, 12 trees in each of the 2 plots.
Table 1 – Pruned stub trial treatments

<table>
<thead>
<tr>
<th>Summer treatment</th>
<th>No. of plots</th>
<th>No. of trees</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1 No summer fungicide</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>S2 Summer fungicide</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>S3 Summer fungicide delayed 3 months</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>S4 Summer inoculation immediate</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>S5 Summer inoculation delayed 3 months</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>S6 Summer inoculation delayed 6 months</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>U Unpruned</td>
<td>4</td>
<td>48</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Winter treatment</th>
<th>No. of plots</th>
<th>No. of trees</th>
</tr>
</thead>
<tbody>
<tr>
<td>W1 No winter fungicide</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>W2 Winter fungicide</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>W3 Winter fungicide delayed 3 months</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>W4 Winter inoculation immediate</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>W5 Winter inoculation delayed 3 months</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td>W6 Winter inoculation delayed 6 months</td>
<td>4</td>
<td>48</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>52</strong></td>
<td><strong>624</strong></td>
</tr>
</tbody>
</table>

Inoculation: Spore suspensions were prepared by placing flasks of 0.5% malt extract solution seeded with *Acremonium* spores onto a shaker for 5-7 days (ambient temperature). Solutions were then diluted to $1 \times 10^6$ spores/ml. Each stub in the inoculated treatments was sprayed with 1/2-2 ml spore suspension applied with a trigger sprayer, the volume depending on stub diameter.

After pruning, all branch stubs on 3 whorls per tree were measured and marked. The design allows some multiple replication among treatments; for instance, delayed inoculation after pruning acts as another control treatment until the inoculum is applied. The treatments were the same for summer and winter pruning; there are 288 trees in each treatment to compare pruning in summer and winter. A second-lift prune with the treatments repeated on two whorls was carried out on the same trees 2 years after the initiation of the trial.

Evaluations, undertaken on a stub-by-stub basis, where the assessor records stem fluting and fruit body formation are carried out every 3 months. Assessments of all study whorls will continue until December 2006, in order to confirm results and enable evaluation of treatments applied after the second-lift pruning. The low background level of

Figure 1: Percentage of pruned stubs with associated flutes

![Figure 1](image-url)

S = summer prune, W = winter prune, U = unpruned
1 = no fungicide, 2 = fungicide applied immediately, 3 = fungicide applied at 3 months, 4 = no fungicide, inoculated 0 months, 5 = no fungicide, inoculated 3 months, 6 = no fungicide, inoculated 6 months
stem flutes that are physiological in basis cannot be reliably separated from those associated with infection and all flutes are included in the assessment.

Results

Results 27 months after the establishment of the first stage of the trial are given here. For those treatments (S1, S3, W1, and W3) in which natural infection could be assessed, fluting was recorded, in at least one of the three whorls pruned during the first-lift, in 21 – 38% of trees. These figures are in the range of those that were recorded in the regional survey described above, in which fluting levels ranged from 0 - 80% in different stands, with an overall mean of 25%. The incidence of fluting was higher in trees that were pruned in winter. Winter pruning, without inoculation or fungicide, resulted in 38% infection compared with 21% for the summer pruned treatment. Percentage stubs infected is lower that percentage of trees

A total of 13,545 individual stubs are assessed every three months. Analysis of fluting on individual stubs is more sensitive than examining fluting incidence on individual trees and results are shown in Figure 1. Fluting was rarely associated with stubs smaller than 30 mm diameter (Figure 2). Only 0.4% of the 8,826 stubs in this category (excluding treatments where inoculum was applied immediately after pruning) were fluted. It seems likely that natural inoculum alone will not usually initiate infection of stubs this

Stubs inoculated immediately after winter pruning (treatment W4) had 18.6% infection, compared with 5.9% of those inoculated immediately after summer pruning (treatment S4). The rate of infection decreased significantly when inoculation was delayed by 3 or 6 months. Summer fungicide applied immediately after pruning resulted in 1.6% infection, identical to the corresponding winter fungicide treatment. Delayed fungicide application was slightly less effective, with 2.4% of stubs infected when fungicide was applied 3 months after summer pruning and 3.9% infection for application 3 months after winter pruning. Infection in the winter inoculation treatment was significantly different (P<0.0001) than the other treatments. Winter pruning and summer inoculation had the same level of infection, followed by the summer and winter fungicide treatments and summer pruning, although differences between the two groups were not statistically significant.

Figure 2: Percentage of stubs with associated flutes (excluding immediate summer and winter inoculation treatments).
size and artificial inoculation under optimal conditions is required. In the winter pruning treatment, 32% of the larger stubs (≥40 mm) had flutes compared with 16% for the summer pruning treatment, confirming the overall trend of increased fluting after pruning in winter. Fluting incidence was higher in the immediate winter inoculation treatment, when compared with the immediate summer inoculation, over all stub sizes.

Perithecial development is slow and rare. Perithecia were first seen 12 months after pruning and inoculation in summer, and 9 months after inoculation in winter. After 27 months, perithecia have been recorded on only 0.7% of trees.

The trial has now delivered some clear trends.

- Incidence of fluting is related to stub size.
- Winter pruning results in more infection than summer pruning.
- Inoculation immediately after pruning results in increased infection, in summer and in winter.
- Pruned stubs may be susceptible to infection for at least 3 months after pruning, as delayed fungicide application is ineffective.
- Fungicide application immediately after pruning reduces infection in both seasons.
- Fruiting bodies take at least 9 months to develop after treatment.

The key message is that pruning operations should not be undertaken in winter, and that fungicidal treatment of small stubs will not reduce overall disease incidence.

**Conclusions and future research**

This paper outlines research in progress on many aspects of the pine fluting disease associated with *N. fuckeliana* in New Zealand. In addition to the projects outlined, several other aspects are being considered. For example, it is not known whether appearance of the fungus in New Zealand resulted from a single or from multiple introductions. DNA analysis of the many isolates so far obtained will help to answer this question. Three genetics trials that were established some years ago to assess growth and wood characteristics of a number of breeding lines have been assessed for breeding values associated with resistance to *N. fuckeliana*. Other aspects of the disease process also warrant further investigation. One characteristic of the disease is the formation of zones of dry, non-living cells in the sapwood in association with many of the pruned branch stubs. These zones initially form radially from the surface wound into the pith, and then longitudinally and primarily up the stem. They are apparent in stems felled up to 2 years after the pruning operation. This feature is postulated to be due to cavitation in tracheids at the wound surface under the influence of desiccation, with air moving through to adjacent tracheids resulting in loss of xylem function. The importance of dead wood zones in the infection process by *N. fuckeliana*, and by other fungal colonizers, has not been established but will be a future research focus.

Identification of a new canker disease in New Zealand *P. radiata* plantations initially caused alarm in the New Zealand forest industry. It is hoped that the results of these studies, however, on many aspects of the basic biology of the fungus and its relationship to its host in this new environment will enable plantation owners to manage the disease effectively. These studies may also have implications for other areas of the world where *P. radiata* is an important forest crop.

**References**


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Left To right: Eric Smith, Jim Worrall, Diane Hildebrand, Russell Beam, Amanda Crump, Jessie Michaels-Glasser, Betsy Goodrich, Jim Walla, Jennifer Klutcht, Jim “We don’t need no stinkin’ sunscreen” Hoffman, Jim Blodgett

Photo by DeNitto
Observations of the Beech Bark Disease Complex on the National Forests of Pennsylvania and West Virginia

Martin MacKenzie

Introduction

Beech bark disease (BBD), a complex affecting American beech (Fagus grandifolia Ehrh.), includes both insect and fungal components. The classic concept of BBD, first articulated by Alex Shigo in 1972, remains the standard for today's forest pathologists. Shigo (1972) described three arbitrary, temporal disease phases: the initial scale front phase, the second killing front phase, and the final aftermath forest phase. During the initial phase, the exotic scale insect disperses through the forest, causing scale-induced alterations to patches of bark. The killing front phase begins 1 to 19 years after the arrival of the scale (MacKenzie and Iskra 2005). Throughout this phase, the scale-modified bark is killed and colonized by species of Neonectria, rendering the dead tissues vulnerable to additional decaying fungi. The resulting beech snap and mortality levels may reach 50 percent in 5 years. The final aftermath forest phase results in an ecological accommodation to the disease, resulting in either a change in species composition or the death of re-emergent beech. The genetically identical stump sprouts and root suckers, which appear following the initial BBD deaths and/or salvage, die in a second wave of BBD. A classic example of aftermath forest occurred in Maine, where beech thickets of root sprout origin developed following the harvest of dead or declining mature beech; these genetically identical, thus susceptible, stands are now poised for a new outbreak of the disease (Houston 1975).

The classical model of BBD is:

Exotic scale invasion \(\rightarrow\) (the scale front) \(\rightarrow\) scale-induced alterations in beech tree bark \(\rightarrow\) pathogenic Neonectria colonization of altered beech bark \(\rightarrow\) bark cankers being colonized by decay fungi \(\rightarrow\) beech death and snap (the killing front) \(\rightarrow\) a period of ecological adjustment of the forest ecosystem to the disease (the aftermath forest). This final phase may result in a second wave of the disease if the aftermath forest is derived largely from root suckers of susceptible trees.

Shigo's aftermath concept might well have been influenced by events occurring 30 years prior to his proposition, as it mirrors the effects of the exotic chestnut blight (Cryphonectria (Endothia) parasitica (Murrill) Barr.) on the American chestnut (Castanea dentata (Marsh) Borkh). Stump sprouts of trees killed by chestnut blight were in turn killed by a subsequent wave of this disease.

Insect Component of the BBD Complex

Cryptococcus fagisuga Lind. is the insect component of BBD. Accidentally introduced around 1890, this exotic scale traveled to North America on planting stock of European beech (Fagus sylvatica L.) (Houston and O'Brien 1983). The scale, first recorded in 1911 on American beech in Halifax, Nova Scotia, and recognized in Boston in 1929, spread south and west into Pennsylvania and New Jersey (Houston 1994a). These parthenogenic scales are female and only about a millimeter long, with a stylet of approximately 2 mm in length. Mature females deeply imbed their stylet in the outer layer of beech bark; in most instances, the stylet does not reach the beech cambium. The eggs of these hemimetabulous insects hatch as nymphs, known as the crawler stage, and can be windblown for short distances (Wainhouse 1979). Adult scales are wingless; transport over longer distances may involve...
human intervention (Houston 1994a). While crawlers are exposed and orange in color, the adult scales cover themselves in a white, woolly wax secreted from numerous glands, which in high populations creates the appearance that the beech stem is whitewashed. The presence of this protective wax is the signature characteristic of this insect. The scale feeding sites provide the lesions necessary for the colonization of the bark by *Neonectria*, the fungal component of this disease.

**Fungal Component of the BBD Complex**

**Tarry Spots as Indicators**

Red-brown exudate, a slime flux from small, local lesions, usually described as “tarry spots,” is often associated with the initial fungal colonization of scale-altered bark. Ascospores, or asexual conidia, are assumed to germinate on the bark surface and grow down the wounds produced by the scale stylets. Subsequently, *Neonectria* produces perithecia around these tarry spots (Houston and O’Brien 1983).

**The Fungi**

Two species of *Neonectria* comprise the fungal component of the BBD complex—one native to North America, the other considered exotic. John Ehrlich (1934), in his classic study of BBD in America, recognized the fungal component as being a *Nectria* species in the coccinea group. Lohman and Watson (1943) created the binomial *Nectria coccinea* var. *faginata* Lohman, Watson and Ayers var. *nov* accommodating the exotic fungus, while recognizing *N. galligena* Bres as the native component of the disease. Both BBD fungi produce a *Cylindrocarpon* asexual stage; based on the observation that a natural grouping of *Nectria* species existed with a *Cylindrocarpon* asexual stage, Rossman and others (1999) created a new genus, *Neonectria*, to accommodate them. Interestingly, in the proceedings of a recent symposium on BBD, almost all authors preferred using the older genus name, *Nectria* (Evans and others 2005).

**BBD on the Allegheny and Monongahela National Forests**

Extrapolating from scale distribution as depicted in Towers (1983), the BBD scale probably reached the Allegheny National Forest (ANF) in Pennsylvania around 1979; the first aerial detection report of BBD-induced mortality occurred in 1983 (Acciavatti and Dropp 1985). One of the earliest aerial sketch maps depicting BBD on the ANF (Bullard unpublished 1985) identified 1,284 polygon-acres of BBD within, or adjacent to, the northeastern boundary of the Bradford Ranger District, with the epicenter of this mortality occurring just outside the eastern boundary of the forest. Of the 1,284 polygon-acres of BBD mapped in 1985, only 583 acres fell inside the forest. Digital aerial sketch mapping in 2004, in contrast, recorded more than 14 times as much BBD within the ANF boundary, with a total of 21,180 combined polygon-acres being impacted by either BBD or the scale alone (MacKenzie unpublished 2004). The 2004 aerial mapping confirmed that the advancing scale front had swept over the entire forest, with about half of the forest now behind the killing front.

When BBD was first found in West Virginia on the Monongahela National Forest (MNF), the scale had infested beech on 70,000 acres (Mielke and others 1982). Only *N. galligena* was recovered from the MNF in 1982; by 1988 half of the stands surveyed yielded *N. coccinea* var. *faginata*, and by 1991 (although *N. galligena* was still present), all surveyed stands yielded *N. coccinea* var. *faginata* (Houston 1994b). Today, the only fungus the author has been able to identify from American beech on the MNF is the exotic variety, indicating that, once present, the presumably more evolutionary fit, exotic variety replaces the ubiquitous native species, *N. galligena*, at least on beech.

**BBD on the Tionesta Research Natural Area of the Allegheny National Forest**

The Tionesta Research Natural Area (RNA) and the adjacent Tionesta Scenic Area, with a combined area of about 4,000 acres, constitute one of the last uncut remnants of the beech-hemlock climax forest that once covered 6 million acres of the Allegheny Plateau in Pennsylvania and New York (Bjorkbom and Larson 1977). Aerial sketch mapping in 2000 indicated a large polygon of discolored beech, centered on the Tionesta areas, and ground truthing revealed many mature beech trees with lower boles almost completely whitewashed with the beech scale. Given the significance of these reserved areas, 12 randomly located, variable-radius monitoring plots were established within the Tionesta RNA, encompassing a total of 200 beech trees. Although aerial sketch mapping in 2000 placed these trees within a polygon of discoloration, by the spring of 2001 all trees apparently leafed out completely. Declining crowns were not detectable from the
ground until the summer of 2002. Each of these beech trees is assessed annually for mortality, crown condition, insect attack, and fruiting of fungi. Intensive searching for the perithecial stage of *Neonectria* began in 2001; several searches over a 5-year period, some involving 100 person-hours and many pathologists and technicians, failed to detect a single peritheium. Conversely, perithecia were easily found in the aftermath forest. During 2001, a very large number of tarry spots were observed on most beech trees; many of the tarry spots were black, not red-brown, and appeared to have a hole at their center. An intensive 2002 survey of tarry spots concurred with the establishment of an ethanol-baited Lindgren funnel insect trap. The trap ran monthly for a year beginning in February, with all Scolytids counted and identified (table 1). Survival of plot trees on the Tionesta RNA is depicted in figure 1.

**Table 1**—The results from a single ethanol-baited Lindgren funnel trap run monthly for 1 year on the Tionesta RNA. Exotic species, which collectively totaled 48 percent of the catch, are in **bold**.

<table>
<thead>
<tr>
<th>Insect</th>
<th>Trap Count</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Dryocoetes betulae</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Gnatotrichus materiarius</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Monarthrum fasciatum</em></td>
<td>7</td>
</tr>
<tr>
<td><em>M. mali</em></td>
<td>28</td>
</tr>
<tr>
<td><em>Trypodendron lineatum</em></td>
<td>38</td>
</tr>
<tr>
<td><strong>Xyleborinus alni</strong></td>
<td><strong>108</strong></td>
</tr>
<tr>
<td><em>X. saxesseni</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Xyleborus sayi</em></td>
<td>90</td>
</tr>
<tr>
<td><em>Xyloterinus germanus</em></td>
<td>51</td>
</tr>
<tr>
<td><em>Xyloterinus politus</em></td>
<td>7</td>
</tr>
</tbody>
</table>

*The only true bark beetle; all others are ambrosia beetles.*

Using crown class to separate the beech trees yielded four times as many beetle holes associated with trees exhibiting poor crowns as those with full crowns; 78 percent of the tarry spots included a hole at their center. A concurrent survey, conducted in a healthy beech stand several miles ahead of the killing front on the Tionesta RNA revealed an average of 27 beetle holes per tree, between 0.70 m and 1.7 m. There were as many holes associated with trees exhibiting heavy scale populations as on those with light scale densities. The predatory twice-stabbed ladybird beetle (*Chilocorus stigma* Say), in contrast, appeared 10 times more often on trees with heavy scale than on trees with light infestations.

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Two inches of snow fell on parts of the ANF May 18, 2002, followed by 4 days of frost; the inclement weather occurred just as most beech trees were leafing out or completing the process. The widespread, out-of-season snow and frosts occurred from Pennsylvania to western Maryland and as far south as the Monongahela National Forest in West Virginia. By late June, the crowns of many beech trees remained reddened with dead leaves. Some
trees displayed only partial refoliation by early July; others never did refoliate.

The wet spring of 2003 in northwestern Pennsylvania served to break an ongoing drought and led to an outbreak of foliar anthracnose that reddened many tree crowns, detectable from the air when sketch mapping and obvious from the ground as well. While the beech on the Tionesta RNA suffered from a series of biotic and abiotic stresses, *Neonectria* was not one of them in 2003. Ahead of the killing front, a plot of 200 beech trees suffered no mortality (other than blowdown), while well behind the killing front, in aftermath forest, a third set of 200 beech trees incurred only one to two percent mortality. Surprisingly, beech snap and mortality of 30 percent of the trees occurred at the killing front in 2 years, raising the question of whether or not this was caused by BBD, without evidence of either beech canker or *Neonectria*.

In late summer, *Tremex columba* L. females were encountered ovipositing into the lower bole of often visibly stressed beech trees. Frequently, the insects would die after ovipositing, with the final few segments of their abdomen found attached to trees. M. A. Stillwell (1964) first pointed out that this insect is often associated with BBD-declining trees. Like the ambrosia beetles, *T. columba* exhibits an intimate association with fungi. While the ambrosia beetles vector in external mycangia, the wood-decaying fungi used to feed their brood, the siricids internally vector the fungi, which decays (prepares) the wood on which siricid larvae will feed. Attempts to rear *T. columba* from snapped beech trees recovered only the buprestid *Dicrea divaricata* Say. The common name for this insect, “divergent beech beetle,” denotes its long association with beech trees.

**Can the BBD Scale Alone be a Primary Cause of Beech Mortality?**

By 2005, after 4 years of observation at the killing front on the Tionesta RNA, 48 percent of the trees died, 8 percent are declining, and only 44 percent remain healthy. Scale populations appear to have declined significantly since the first observations of 2001. Yet, despite intensive searching, *Neonectria* has not been observed fruiting on any of the dead or declining trees; additionally, no cankered trees were recorded. The best explanation for these observations comes from the work of Manion (1991), who defines a decline as “an interaction of interchangeable, specifically ordered abiotic and biotic factors to produce a gradual general deterioration, often ending in death of trees.” His concept revolves around a “decline disease spiral” comprised of three sets of interchangeable and interacting factors. The first set includes predisposing factors, the second set inciting factors, and the third set contributing factors; at least one factor from each of the three sets must be present for tree decline to occur. Frequently, more than one factor is present from each set, acting in the spiral. A predisposing factor is needed in order for the decline to be possible, an inciting factor is necessary to initiate the decline, and finally, contributing factors are required for the stressed trees to be killed. The three sets of factors of the Manion decline spiral, as applicable to the Tionesta RNA, are listed in Table 2.

Table 2 illustrates that factors from all three sets occur more than once; therefore, under Manion’s concept of a decline spiral, the BBD complex at the Tionesta RNA is correctly described as a decline, not as a simple disease.

Depiction as a simple disease requires the occurrence of the primary pathogenic fungus, in this case, *Neonectria*. All but one of the decline spiral stressors existed in most parts of the ANF from 2001 to 2005, and yet the highest levels of BBD mortality were localized in the zone of the killing front. The single factor that was localized on the Tionesta was the very high beech scale population observed in 2001 and 2002. Apparently, given all the other factors in a fully stocked, old-growth hemlock-beech stand, the scale stressor precipitated a decline spiral, causing 50 percent of the beech to die before either *Neonectria* fruited or the trees developed bark cankers. Although the fungus has not yet fruited at the killing front, this phase of mortality should be considered part of the BBD complex. The stress the scale placed on the beech trees may have facilitated

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### Table 2—The three sets of decline spiral factors. Those present on the Tionesta RNA are in bold.

<table>
<thead>
<tr>
<th>Predisposing Factors</th>
<th>Inciting Factors</th>
<th>Contributing Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Host genetically predisposed</td>
<td>Defoliating insects</td>
<td>Canker fungi</td>
</tr>
<tr>
<td>Poor fertility</td>
<td>Drought</td>
<td>Bark and boring insects</td>
</tr>
<tr>
<td>Climate change</td>
<td>Frost</td>
<td>Armillaria root rot</td>
</tr>
<tr>
<td>Old age</td>
<td>Air pollution</td>
<td>Saprobiic decay fungi</td>
</tr>
<tr>
<td>Urban development</td>
<td>Excavation</td>
<td></td>
</tr>
<tr>
<td>Outside natural range</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
attraction of larger-than-normal populations of ambrosia beetles, siricids, and buprestids to the Tionesta.

**Aftermath Forest on the Allegheny National Forest**

My unpublished report on the aftermath forest areas of the ANF (MacKenzie unpublished 2005a) details that two-thirds of the beech basal area died in unmanaged, second-growth stands during the 12-year period between 1991 and 2003. These stands ranged in age from about 70 to 100 years old and were first impacted by the scale 23 years earlier. The stands are fully stocked today, making it safe to assume that the crowns of the maples, birches, and black cherry (*Prunus serotina* Ehrh.) expanded to take advantage of the growing space vacated by the dying beech. Unlike the situation in New England, BBD on the ANF acted as a killing disease instead of a debilitating, disfiguring disease. While the current aftermath forest on the ANF contains many fewer beech trees, a higher percentage of the residual trees remains both scale and canker free, apparently the result of evolutionary selection of the fittest. The aftermath forest in the Bradford Ranger District of the ANF contains few highly defective stems and, unlike the forests of Maine, is not set up for a second wave of BBD.

**Aftermath Forest on the Monongahela National Forest**

A survey of residual beech, made approximately 20 years after the discovery of BBD on the MNF, revealed nine times as many root suckers associated with canker-free trees than associated with dead or cankered trees (MacKenzie 2005b). Paralleling the observations on the ANF, the aftermath forest on the MNF had not come back as a thicket of beech sprouts. Trees susceptible to BBD either died or were heavily cankered, their place in the canopy filled by the expanding crowns of cucumber magnolia (*Magnolia acuminata* L.), black cherry, or sugar maple (*Acer saccharum* Marsh.). Failing to produce significant numbers of seedlings or root suckers, their place in the understory is occupied by red spruce (*Picea rubens* Sarg.). The extent to which this change in the understory is driven by the disease or by deer populations is not now obvious.

**Potential Aftermath Forest on the Tionesta RNA of the Allegheny National Forest**

The current areas of aftermath forest on both the ANF and MNF are in second-growth forest areas that are fundamentally different from the Tionesta RNA, which is a remnant of old-growth beech-hemlock forest and dominated by these two shade-tolerant species. While the Tionesta does contain some black cherry, yellow birch (*Betula alleghaniensis* Britt), and other species, most of its area is dominated by beech or Eastern hemlock (*Tsuga canadensis* (L.) Carr.). The total number of snapped beech trees rose from 1.5 percent to 20 percent in 2005. Several of these trees snapped below the first branch, exposing a sea of beech sprouts. A reasonable assumption might be that this is a classic BBD aftermath forest-in-the-making. Should the invasive hemlock woolly adelgid (*Adelges tsugae* Annand), currently only one or two counties away, reach this reserve, the area might then evolve into a dense beech thicket, typical of the aftermath forests of Maine surveyed by Houston (1975).

**Summary of Observations**

- Nothing observed on either national forest contradicts the basic BBD model initiated by Ehrlich (1934) and subsequently codified by Shigo (1972).
- When several factors overlap at the killing front, very high rates of mortality are possible within a few years. Under these circumstances, the system is best described as a BBD complex because the trees may die before the *Neonectria* fungus fruits.
- Tarry spots are a nonspecific indicative reaction of damaged beech bark. Spots associated with *Neonectria* tend to be brown-red in color; those associated with ambrosial beetle attack tend to be black, often with a bleached halo.
- Ultimately, the type of aftermath forest that develops is influenced by the age and composition of the stand at the time it falls behind the killing front.
- When a stand is dominated by mature to over-mature beech and composed of relatively few species, “classical aftermath forest” initially composed of beech thickets is likely to develop. Such a stand, originating from beech root suckers, is vulnerable to a subsequent wave of BBD. Alternatively, when the stand contains poles
of shade-tolerant species or the canopy contains numerous nonbeech species, these species are likely to expand their crowns, taking advantage of the growing space vacated by the dying beech trees. Undoubtedly, there exists a spectrum of scenarios between these extremes. At one end, the BBD complex behaves as a killing, disfiguring disease, ultimately resulting in an increased number of smaller, heavily cankered beech stems. At the other end of the spectrum, the BBD complex leads to a replacement of beech by more fit species.

References


Ehrlich, J. 1934. The beech bark disease: A Nectria disease of Fagus, following Cryptococcus fagi (Baer.). Canadian Journal of Research. 10(Special Number): 593-692.


Photo by Angwin
Left to right: Mike Schomaker, Bob Edmonds, Amy Ramsey, Mary Lou “Taking Names” Fairweather, Dave Russell, Gregg Denitto, Ted Hogg, Jim Byler
Identification and Distribution of Nectria Species Causing Cankers in Red Alder Plantations

Craig N. Cootsona

Introduction

In 1998, surveyors noted serious new stem defects on Weyerhaeuser red alder (Alnus rubra Bong) plantations. Symptoms began with dark spots on the outer bark that gradually developed into stem cankers and eventually resulted in tree death. Symptoms occur commonly in inland plantations located in Southwest Washington, with a noticeably lower incidence and severity in stands near the coast. It is thought that the rapid and uniform crown closure seen in plantations may provide a large inoculum load that could explain why infection is more common on plantations than in mixed-age natural stands. The precise mode of spread is not known but airborne and water-splash spore dispersal seems to play a role (Metzler and others, 2002; Swinburne, 1975). In 2002, Weyerhaeuser conducted a stand survey to measure the incidence and severity of cankers in plantations (Dobkowski, 2002). The survey indicated significantly different levels of infection among the stands. The commercial value of red alder and the importance of its role as a nitrogen-fixer and buffer for riparian areas make investigation of this problem worthwhile.

Species identification and distribution

An unidentified Nectria species morphologically similar to Nectria ditissima and Nectria galligena (described in Funk, 1981) was found in association with cankers on living and recently killed red alder trees, as well as recent thinning slash on Weyerhaeuser plantations. A review of literature revealed that researchers in British Columbia tested what they identified as Nectria ditissima for biological control of red alder. It was known by the trademark name Alderkill™ and has been developed using an inoculum known as PFC-Mycocharge™. The system consisted of a dowel of alder inoculated with a particularly aggressive, native isolate of Nectria ditissima. (PFC-082/ATCC 74260) (Dorworth, 1995). A study using the PFC-mycocharge produced nearly 100% inoculation success resulting in notable alder tissue damage and general reduction in tree health (Dorworth and others, 1996). Reproductive structures were not found on any of the inoculated stems during the experiment or in red alder trees near the experimental plot. A follow-up study showed that N. ditissima grew from discs collected from each living, inoculated tree 18 months following inoculation but was only isolated from one cankered stem 36 months after inoculation. The study suggested that N. ditissima merely provided an entrance court or increased stress to allow insects and other fungi to further degrade the stem. The study claimed that PFC-082 had limited or no ability to spread because the PFC-Mycocharge system inoculated older stem tissues where it was not thought to reproduce. The entire Alderkill™ system using N. ditissima is no longer available from Canada as a bio-control agent. Canada is currently promoting Chondrostereum purpureum as an effective bio-control method for red alder (Shamoun and others, 2002). It is not known if the Alderkill™ system was ever introduced in the US but the system does identify the susceptibility of red alder to infection by Nectria. It remains important to identify the species distribution on Weyerhaeuser plantations so a management plan can be developed.

Objectives

There are three main objectives for this project:

1. Use molecular methods to identify Nectria species, and look for correlations between species and geographic location to explain differences in infection.


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2. Use inoculated host tissue to complete Koch’s postulates and determine relative pathogenicity.

3. Determine if weather data correlates with infection data at each stand. These findings will be used to construct a management plan and risk assessment model for red alder plantations.

Methods

Isolates were collected from surface-sterilized bark near cankers, and grown on potato dextrose agar. DNA was extracted using methods adapted from Punekar and others (2003). An aliquot of 10 ul (5ng/ul) of each DNA sample was made into PCR tubes. To each tube was added 3 ul MgCl2, 1 ul of ITS1 forward primer, 1 ul of ITS4 reverse primer, 0.1 ml Taq polymerase, and 34.9 ml of sterile DI water. PCR was run using a PTC-100 Programmable Controller (MJ Research, Incorporated). The thermocycler was set for a temperature cycle of 95, 55, 72 degrees C for 35, 55 and 45 seconds. For cycles 17-29, 72 degrees C was held for 2 min, and increased again to 3 min for the remaining 10 cycles. Following PCR, DNA to be sequenced was purified using the QIAquick PCR Purification Kit (Qiagen Cat No. 28104).

ITS Sequences from several different Nectria species were collected from Genbank.com and compared with sequences from local isolates using Clustal X v1.8. The comparison showed a close similarity between N. galligena, N. diíssima, and the Nectria species found on red alder. The species above were similar enough that they could not be distinguished using the ITS sequences alone.

Due to the conserved nature of the ITS region among these Nectria species, future sequence analysis will use the Elongation Factor–1a (EF-1a) gene, which shows greater variability among the Nectria species. Phylogenetic trees will be constructed using EF-1a sequences from locally collected isolates and from N. galligena and N. diíssima from American Type Culture Collection. This work is currently in progress. This information will help determine the relationship among species and help to identify any correlations that may exist between species and stand infection data.

Koch’s postulates and relative pathogenicity

A Nectria species closely related to Nectria diíssima and Nectria galligena was found in Weyerhaeuser red alder plantations in association with cankers on living and recently killed trees, as well as recent thinning slash. Sequencing studies mentioned above identified the isolates as Nectria spp. but the species are not known. To confirm the cause of cankerling, Koch’s postulates were attempted. In addition, growth rates of cankers or necrotic zones in host tissue were expected to provide information on relative pathogenicity of isolates (Perrin, 1984; Perrin and Van Gerwin, 1984).

Isolates were collected from surface-sterilized bark in the region between necrotic tissue and live tissue. Isolates were maintained on potato dextrose agar at 4°C until needed. A trial study was conducted using 9 branch cuttings from local red alder trees. They were surface sterilized in 30% bleach/water solution and cut into 4” sections. The cut ends were immediately sealed in hot wax to prevent moisture loss from the cutting. A sterilized scalpel was used to cut a small flap of bark to expose the cambium. On 6 cuttings, a 5x5 mm plug of potato dextrose agar inoculated with a Nectria sp. isolate was inserted under the flap and covered with parafilm to prevent desiccation. Three control inoculations used sterilized potato dextrose agar. A sterilized moisture chamber was created that contained a rack to suspend cuttings above water, sealed in a plastic container, and stored at 25°C. After 10 days, the parafilm was removed from the inoculation sites.

No signs of necrosis appeared at 15 or 30 days. Hyphae from contaminating fungi began to grow and eventually covered the cuttings and the rack.

No signs of necrosis or cankerling were seen in the cuttings, so it is thought that the inoculum did not survive or was overgrown by saprophytic fungi. In retrospect, autoclaving the branch cuttings prior to inoculation may have prevented contaminating fungi from growing. Future trials should use live red alder seedlings when they are available. It is expected that live tissue will produce more pronounced necrosis or cankerling. Based on dimensions of the affected areas, relative pathogenicity can be compared amongst the isolates and regressed against stand infection data.

Correlation between weather data and plantation infection data

While collecting isolates for this study, it was noted that cankers and fruiting bodies associated with Nectria became increasingly more difficult to find in stands near the coast with higher rainfall. Drought stress weakens trees by reducing photosynthesis and mobility of resources. Drought stress often leads to higher incidence and severity of fungal infection
(Chapela and Boddy, 1988; Moricca, 2002), reduced growth rates (Giordano and Hibbs, 1993), and reduced formation of callous tissue around infected areas (Bevercombe and Rayner, 1980). It is hypothesized that sites with the least precipitation and highest temperatures will show higher incidence and/or severity of infection by *Nectria*. Although soil moisture or bark moisture data may provide the most accurate model for this regression, data collection for these factors is beyond the scope of this project.

Precipitation and temperature data will be regressed with the 2005 Weyerhaeuser red alder stand survey data, pending the release approval of the survey data. If drought stress causes greater susceptibility to infection, an inverse relationship is expected between precipitation and infection incidence and/or severity. The same inverse relationship is expected if temperature affects incidence and/or severity.

**Conclusions**

If strong correlations are found between canker incidence and/or severity and any of the following: species distribution, pathogenicity, precipitation, or temperature, GIS software can be used to construct a risk assessment model. Such a model could prove valuable in efficiently managing high-risk sites.

The *Nectria* species seen in red alder plantations reproduces sexually, so genetic diversity is expected to be high. Therefore, clonal populations are not expected to dominate specific stands or correlate geographically with infection data. It is possible that if multiple species exist on red alder, some species may be more virulent than others, causing greater infection in sites where that species is most abundant. However, it is expected that other site-specific factors such as moisture stress better explain the differences in infection among red alder plantations.

**References**


Influence of Climate on Inoculum Availability and Severity of Disease Caused by *Fusicoccum arbuti* on Pacific Madrone

Marianne Elliott

**Introduction**

Pacific madrone (*Arbutus menziesii* Pursh) is native to the western US and is one of the largest members of the family Ericaceae. The trees have been in a noticeable decline since a period of drought in the mid 1970s (Figure 1). A canker fungus, *Fusicoccum arbuti* D. F. Farr and M. Elliott, is associated with declining madrones (Farr et al 2005). Symptoms of infection by *F. arbuti* are cankers, branch dieback, and shoot blight. Depending on site conditions, disease progress can be very rapid, causing permanent defoliation of 75% of the tree in 8 years (Figure 1). This slow starvation reduces starch reserves in the root burl that are used to regenerate dead parts. Madrones are adapted to sites with frequent fires and resprouting from the root burl gives them the competitive advantage over conifers, which regenerate from seed (McDonald and Tappeiner 1990). In the absence of fire, disease plays a more important role in mortality of aboveground parts.

*Fusicoccum arbuti* was initially identified as *Nattrassia mangiferae* (Davison 1972, Hunt et al. 1992), which it resembles morphologically. Based on DNA sequence analysis, *N. mangiferae* has been given the name *Fusicoccum dimidiatum* (Farr et al 2005). These fungi have a sexual stage in *Botryosphaeria*, a group of fungi that cause canker and dieback diseases on woody plant hosts, usually after a period of stress (Crist and Schoenweiss 1975, Ma et al. 2001).

Optimum conditions for infection by *Botryosphaeria* species closely related to *F. arbuti* are days when the average daily temperature is between 20 and 33 °C with periods of wetness from 48 to 72 hours (Arauz and Sutton 1989, Von Broembsen and Van der Merwe 1990, Michailides and Morgan 1992).

![Figure 1. Declining madrone tree with progressive defoliation caused by *F. arbuti* and other canker fungi. This tree at Magnolia Bluffs Park in Seattle, WA had a full crown in 1996 (A) and is mostly defoliated in 2004 (B).](image)

The number of days per year with those temperature and moisture conditions has increased since 1976, especially in El Niño years where there are warm, wet winters in California. An increase in global precipitation of 2 +/- 0.5% is expected with each 1°C increase in temperature. Higher latitudes in the Northern Hemisphere are expected to have above-average increases in temperature and precipitation (Coakley et al. 1999). Warm, wet winters occur in the Pacific Northwest during La Niña years (Redmond and Koch 1991). In 1976 the atmospheric circulation over the Pacific and North America changed, causing a shift in weather patterns (Trenberth and Hurrell 1994). This was named the Pacific Decadal Oscillation (PDO) (Mantua et al. 1997) and it has more of an impact on the climate in the Pacific Northwest.
than ENSO (El Nino Southern Oscillation), which has a stronger effect on California’s climate. El Niño and La Niña events have been stronger and more frequent during this phase, but it is not known whether this is related to global warming caused by humans or is part of a larger natural cycle. The PDO was expected to enter a cool phase around the year 2000, and there is some evidence that this is occurring (Gedalof and Smith 2001).

The objectives of this study were to determine the distribution and severity of the canker disease and to understand the reasons for increased symptoms over the past 30 years. Climate change and its effects on fungal biology such as inoculum availability, sporulation, and infection courts were examined in detail. This article is based on preliminary results and more analysis is needed.

**Methods**

**Presence of *Fusicoccum arbuti* in healthy and diseased tissues of Pacific madrone**

Samples of both symptomatic and asymptomatic Pacific madrone leaves, shoots, twigs, and reproductive structures were taken from five trees at each of three Seattle city parks. This was done at two month intervals during the period of April to December 2004. Plant material was cut into small pieces, surface sterilized in a dilute bleach solution, plated on 2% malt extract agar plates, and presence or absence of *F. arbuti* was noted for each sample. Monthly rainfall and temperature data were downloaded from the National Weather Service website (NWS 2004). Normals for mean, maximum, and minimum monthly temperature and total monthly precipitation were calculated as the average over the period of record, 1947-2004.

**Tree surveys**

Surveys of tree health were done during the years 1996 – 2003 on sites covering the range of Pacific madrone (Figure 2). Survey data included live crown ratio (%LCR) and percent dead crown (%DEAD). From these measurements percent live foliage (%FOL) was calculated according to Equation 1:

\[
\text{Equation 1:} \quad \%\text{FOL} = \%\text{LCR} - \left(\frac{\%\text{DEAD} \times \%\text{LCR}}{100}\right)
\]

Percent foliage is an indicator of tree health and is influenced by the severity of stem canker, root rot, and branch dieback, as well as site factors such as crown class (Elliott 1999). Climate data from National Climatic Data Center (NCDC 1994) and Western Regional Climate Center (WRCC 2005) were downloaded for each site and climate division where surveys were done. Climate data were explored by means of scatter plots relating %FOL to climate variables during the entire period of record (1947-2003), and during PDO years (cool phase 1947-1976, warm phase 1977-1998, and cool phase 1999-2003).

![Figure 2. Study sites in the range of Pacific madrone by climate division. Black dots represent sampling locations.](image)
An index (X) for years with warm, wet summers was developed from Equation 2 for each climate division where Pacific madrone was surveyed:

Equation 2: \[ X = \text{Average Palmer Drought Severity Index (PDSI)} + \text{Difference from mean monthly temperature of period of record (1895-2003)}. \]

If X is positive, the summer (June-August) is warmer and wetter than normal, and if X is negative, the summer is cooler and dryer than normal for a given year.

Another index (Equation 3) was calculated for warm, dry summers:

Equation 3: \[ Y = \text{Average Palmer Drought Severity Index (PDSI)} - \text{Difference from mean monthly temperature of period of record}. \]

Data were averaged for each climate division and linear regressions were performed on standardized variables with average PCTFOL the dependent variable and the independent variables were X and Y indices for each phase of the PDO and for the period of record.

Results

Presence of *Fusicoccum arbuti* in healthy and diseased tissues of Pacific madrone

*Fusicoccum arbuti* was present in both symptomatic and asymptomatic foliage, although it was isolated less often from healthy foliage. The fungus was recovered rarely in newly emerging foliage. Senescing leaves had higher levels, but the fungus was isolated less often from litter, probably due to competing fungi from the soil. Bud break occurred in June. The fungus was present in asymptomatic twigs. Branches with both branch dieback and canker symptoms contained *F. arbuti*. Diseased wood had the highest isolation frequency for April, June, and December. Healthy and diseased shoots showed a similar pattern to wood with more isolation from June-December. *F. arbuti* was not isolated from seeds and was most commonly isolated from diseased fruit stalks and fruit during the summer months. Diseased fruit stalks had the highest isolation frequencies of any tissue in August and October.

The mean monthly temperature during the sampling period April–December 2004 was not different from normal in Seattle, WA. There were differences from normal precipitation, with a dry period from June to July followed by a wetter than normal August through September.

Disease frequency and severity

Madrones growing in climate regions with warm, wet summers had lower percent foliage than those growing in climate regions with cool, dry summers. The northern part of the range tended to have more severe disease and increased warm, wet summer weather relative to sites in the southern range. These climate conditions increased during the period from 1976 to 1998, corresponding with the warm phase of the PDO. These results were not significant, but point to climate as one factor contributing to madrone decline.

Discussion

*Fusicoccum arbuti* is present in asymptomatic plant tissues, but at lower levels than in diseased tissues. Diseased wood, shoots, and fruit stalks had the highest isolation frequency for all months. These tissues are important sources of inoculum and should be removed to prevent inoculum buildup. The fungus was isolated from healthy and diseased leaves and less often from litter. The fungus was not isolated from seeds.

High levels of *F. arbuti* in Pacific madrone tissues in October and December may be explained by new infections that occurred from increased inoculum during the wetter than normal months of August and September.

Infected shoots, branches, and fruit stalks are the most likely locations for the sexual stage of *F. arbuti*. These tissues should be examined in July and August. Overwintering pycniosclerotia derived from pycnidial stromata may be found on these plant parts in December, which would develop into ascocarps the following summer. These would be sources of new genotypes of *F. arbuti*. Distribution of these spores in stemflow would account for multiple genotypes found in an individual canker.

Warm, wet summers have increased since the mid 1960’s in western Washington and that there is a more random pattern to warm, dry summers, although individual drought years can have severe impacts on tree health. The period
between 1900 and the mid 1960’s was dominated by cool, dry summers in western Washington. Under these conditions, there is low transpiration and less water stress on trees, and also less favorable conditions for fungal reproduction and infection. Madrones in the southern range (Climate divisions CA04, CA02, and CA01) had more live foliage compared to madrones in the northern range (WA03, WA02, OR02, and OR03), and there was a difference in climate in these regions, with the southern range being much drier than the northern range.

Trees are under water stress during prolonged summer drought periods and symptom expression of *Botryosphaeria* can occur. A correlation between drought stress and severity of *Botryosphaeria* blight caused by *B. dothidea* on pistachio was demonstrated by Ma et al. (2001). The increase in *Botryosphaeria* blight in California was attributed to increased summer drought in the mid 1980s and the disease was first reported on almond in California in 1966 (Michailides 1991).

In the case of Pacific madrone decline caused by *F. arbuti*, too much moisture is more of a factor in the increased incidence and severity of the disease rather than too little. However, periodic severe droughts create new material on which the fungus can sporulate and increase inoculum potential.

**Conclusions**

Madrone decline during the last 30 years is due to increased moisture during the summer months, although other factors may be involved. Percent live foliage was reduced on sites which had experienced an increase in warm, wet summer climate conditions. These sites were in the northern range of Pacific madrone. *Fusicoccum arbuti* was found in both asymptomatic and diseased tissues of Pacific madrone in Seattle, WA, and isolation frequency increased after a wetter than normal period in August and September 2004. Cankered and blighted wood, shoots, and fruit stalks had the highest isolation frequencies and these are important sources of inoculum.

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**References**


Epidemiology for Hazard Rating of White Pine Blister Rust

Eugene P. Van Arsdel, Brian W. Geils, and Paul J. Zambino

Abstract—The ability to assess the potential for a severe infestation of white pine blister rust is an important management tool. Successful hazard rating requires a proper understanding of blister rust epidemiology, including environmental and genetic factors. For the blister rust caused by Cronartium ribicola, climate and meteorology, and the ecology, distribution, and pathology of ribes and other telial hosts affect spread and intensification of rust on white pine hosts (several sections in subgenus Strobus of genus Pinus). The importance of ribes (genus Ribes) for supporting effective inoculum production varies according to differences in susceptibility, diversity, distribution, and abundance by taxon and population. Temperature and humidity regimes and air circulation patterns at micro to synoptic scales influence the development and dispersal of the rust. Spatial and temporal variations in the dispersal processes are expressed as differences in rust severity distributions. These differences can be mapped as hazard zones and used to choose among alternative management prescriptions. When C. ribicola was first introduced to North America, its epidemiological behavior displayed a limited range of hosts and environments. The diversity of related rusts, however, suggests that C. ribicola may have the capacity to adapt to previously unrecognized hosts and environments. Pine populations have also shown some ability to respond with lower susceptibility to this introduced pathogen, indicating that the North American pathosystems are dynamic and evolving. Efforts to manage such high-elevation species as whitebark pine would be aided by continued research in the epidemiology of this pathosystem in diverse hosts and environments.

Ribes and Meteorology Affect Spread

Success in protecting, sustaining, and restoring white pine ecosystems (Samman et al. 2003) requires the understanding that epidemics of Cronartium ribicola (white pine blister rust) differ by region, landscape, and site. These differences result, in part, from variation in the biology and pathology of ribes\(^1\), limitations imposed by weather and climate, and genetic interactions of the hosts and pathogen. Both ecological and evolutionary processes determine where, how often, and how much the rust spreads and multiplies.

Mielke (1943) illustrated that an epidemiological approach could explain the early spread of the rust in western North America. In the coastal pine region of British Columbia, rust spread was more rapid to the north than to the south because of more favorable winds, hosts, and climate. Winds also had carried aeciospores to the interior white pine belt; but limited distributions of highly susceptible ribes retarded buildup of the rust, even where moisture was adequate. Spread in the western white pine (Pinus monticola) stands of the Inland Empire (northern Idaho, eastern Washington, and far western Montana) was early and rapid—white pine and very susceptible ribes were abundant neighbors in a favorable climate. In spite of adequate winds, however, spread into higher mountains farther east or south was delayed and slow (because the temperatures were usually too low for ribes infection). At cool elevations, even on highly susceptible whitebark pine (Pinus albicaulis), canker growth was slow; there were few generations of rust; and growing seasons were short. In the southern Cascades, precipitation diminished, but adequate moisture still permitted infection; susceptible white and sugar pines (Pinus lambertiana) and very susceptible ribes were present. Winds to transport spores from north to south were more frequent in the Coast Range (early spread) than in the Cascade Range (later spread). Winds capable of transporting spores were even less frequent near the California state line where a sharp, wind deflection to the east limited spread to northern

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\(^1\) Species of currants and gooseberries are in the genus Ribes. These plants are also identified with the common noun, ‘ribes’ and in some, older literature, the proper noun, ‘Ribes’. In this paper, we refer to these plants by their common, general name unless a technical, taxonomic name is required.
California, a region with susceptible hosts and an otherwise favorable climate.

In this paper, we review selected historical literature and identify developing concepts and technologies for applying epidemiological information to mapping potential losses from white pine blister rust. Observations on the present distribution and damage from blister rust provide baseline data for monitoring and initial modeling of hazard. A defensible projection of future trends and expected impacts, however, requires a greater understanding of the ecological and genetic interactions of hosts and pathogen and the environmental effects of temperature, moisture, and air circulation. This understanding begins with knowledge about the principal telial hosts in the genus Ribes.

## Ribes Diversity, Susceptibility, and Importance

There are numerous species, subspecies, varieties, ecotypes, and cultivars of ribes that differ in their response to blister rust infection, to their proximity to white pine, and to their epidemiological importance. Susceptibility is the capacity of a ribes to become infected and support inoculum production. Susceptibility is a genetic trait that varies by plant and environment (reviewed in Zambino and McDonald 2004). It can differ with the growth and habitat of the ribes and with exposure to different strains or pathogenic races of the rust. Susceptibility may be quantified or ranked from field observations, or from inoculations in the field, greenhouse, or petri-dishes. Because of genetic interactions and host physiology, assessment of susceptibility can be affected by technique.


Environment and developmental stage of the ribes greatly affect infection rate efficiency. Young, succulent, greenhouse-grown plants or leaf cuttings inoculated in moist chambers can show very high levels of infection that are well above levels seen in hardened, open-grown, field plants. Leaves are more readily infected after full expansion, and then decrease in receptivity with age (Harvey 1972; Lachmund 1934; Pierson and Buchanan 1938; Spaulding 1922a; Zambino 2000). The period when aging leaves can be infected is extended if shoots become dormant (Harvey 1972). Previous work also suggests that infection is significantly 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 et al. 1937). VanArsdel attributes much of the difference in natural infection of open-grown versus shaded plants to duration of conditions adequate for infection (temperature and dew period) rather than other leaf attributes (VanArsdel et al. 1956; VanArsdel 1965b). The type of infecting spores can also have an effect. Urediospores can cause significant infection in ribes leaves that would be 1 to 2 weeks too mature for aeciospore-initiated infection (Pierson and Buchanan 1938).

We use the term ‘importance’ of different ribes to describe their relative roles in blister rust spread and intensification. Epidemiological importance combines susceptibility (pathology and genetics) with abundance and distribution of ribes and pines (ecology). A very susceptible ribes can be rated low in importance if it were rarely associated with the pine hosts. Conversely, the importance of a ribes would be elevated if its proximity to other ribes and pines resulted in a synergistic increase of infection. For example, Ribes cynosbati is very susceptible to infection, whereas R. americanum has low susceptibility. Where only R. cynosbati is abundant, infection of the pine is uncommon because infected leaves of this species are usually shed before telia mature. Where only R. americanum is abundant, infection of the pine is nearly absent because infection on the ribes is infrequent and late so very few telia develop despite a large, host-leaf surface area. Where both ribes occur, pine infection may occur because abundant uredinia on the R. cynosbati cause some infection of the R. americanum which then produce a few telia. Other susceptible and early-defoliating species are R. pinetorum in the Southwest and R. roezlii in California. Ribes montigenum is an alpine species that often grows next to or under whitebark pine or limber pine but is seldom observed to be heavily rusted. Ribes hudsonianum var. petiolare is a western, riparian plant that grows below the alpine zone; it is very susceptible and capable of

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1 Uredospores or urediospores of older literature, terminology here follows that of the current American Phytopathological Society glossary of terms at [http://www.apsnet.org/education/IllustratedGlossary/](http://www.apsnet.org/education/IllustratedGlossary/).
supporting a large inoculum potential. This ribes may occasionally act synergistically with *R. montigenum*. Occasionally, urediniospores from distant *R. hudsonianum* var. *petiolare* cause heavy infections of *R. montigenum* in close proximity to pine and may lead to heavy pine infection (see Newcomb 2003). Importance is thus a function of the susceptibility of individual ribes plants and inoculum potential of the alternate host community.

The susceptibility and infection potential of western ribes had been reported by numerous authors. Spaulding and Gravatt (1917) conducted an early susceptibility test but did not rank species. Taylor (1922) quantified basidiospore production per unit area for several species. Hahn (1928) found that most ribes of the Northwest were susceptible to both *Cronartium ribicola* and *C. occidentale* (Pinyon blister rust). Mielke (1937) and Mielke et al. (1937) noted differences in susceptibility for the ribes associated with western white pine. Kimmey (1935, 1938, 1944), Buchanan and Kimmey (1938), Kimmey and Mielke (1944), and Kimmey and Wagener (1961) made field observations and inoculations in other western regions. As mentioned, the expression of susceptibility may be influenced by leaf age, shading, or leaf temperature and wetness.

More recently, Hummer and Finn (1999) reported on three years of uredinia production on 55 accessions of ribes exposed to natural infection in an open-grown garden. They noted variation within taxonomic sections of the genus (see Table 1), between species, and within species. Van Arsdel and Geils (2004) interpreted and summarized results of these studies for the ribes of the interior, western states (Table 2).

### Table 1—Sections of genus *Ribes* (by subgenera) with representative species.

<table>
<thead>
<tr>
<th>Subgenus Ribes</th>
<th>Currant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calobotrya</td>
<td><em>R. cereum</em></td>
</tr>
<tr>
<td>Coreosma</td>
<td><em>R. hudsonianum</em></td>
</tr>
<tr>
<td>Heritiera</td>
<td><em>R. laxiflorum</em></td>
</tr>
<tr>
<td>Ribes</td>
<td><em>R. rubrum</em></td>
</tr>
<tr>
<td>Symphocalyx</td>
<td><em>R. aureum</em></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subgenus Grossularioides</th>
<th>Gooseberry Currant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grossularioides</td>
<td><em>R. lacustris</em></td>
</tr>
</tbody>
</table>

**Identification of Ribes**

Ribes are shrubs, to 3 m tall or with prostrate or sprawling growth, and distinguished from similar shrubs by several characteristics (see Van Arsdale and Geils 2004). Leaves are alternate, simple, palmately veined, palmately lobed, with doubly serrate edges. Flowers have five petals, five sepals, five stamens, and a one-chambered ovary with a double style. The withered flowers remain attached to the berry as it ripens. The alternate leaves differ from the opposite leaves of maple and viburnum. Leaves lack a bitter taste, in contrast to the bitter leaves of ninebark. Ribes have no stipules at the base of the petiole, unlike ninebark and thimbleberry. Leaves of *Ribes americanum* have trichomes; the otherwise similar leaves of New Mexico raspberry lack these glandular hairs.

The genus *Ribes* includes three subgenera found in the interior western states. The currants (subgenus *Ribes*) have no spines or bristles and a mature berry which detaches cleanly from its jointed petiole (bearing several fruits); the gooseberries (subgenus *Grossularia*) are usually armed with nodal spines and the petiole remains attached to the berry; spiny currants or gooseberry currants (subgenus *Grossularioides*) are armed with spines and internodal bristles and have jointed floral pedioles. The several sections of the genus represented in the interior West provide a natural classification below the subgenus level (Table 1).

It is necessary to identify ribes by species because they vary greatly in epidemiological importance. We recognize 19 species in the interior western states (Table 2, expanded from list in Van Arsdale and Geils 2004); several of these species are further classified into subspecies or varieties (not presented here). Usually only a few species occur in a given forest, although many more may occur in the region. Identification begins with a review of the ranges and ecological distributions of the local ribes flora (e.g., Holmgren 1997). Floral and fruit characteristics are diagnostic. Ribes flower early in the growing season; fruit develop and persist to mid-season. Familiarity with the local ribes in flower and fruit allows confident identification of specimens later in the season when leaves are infected.

### Ribes Distribution and Abundance

Although ribes occurs across North America, there are notable differences in the mixture of species in the local flora and in their distribution and abundance by region, landscape, and site. Ribes eradication, successional stage, natural disturbance, duration of exposure to rust, and chance events can each potentially affect ribes populations, their genetics, and their epidemiological importance. In the eastern states and provinces, the single most epidemiologically-important ribes had been the cultivated *Ribes nigrum*; wild ribes there are much
less susceptible and less important. Ribes eradication in northeastern states had been aggressive and effective. In these areas, continued low levels of ribes, along with other characteristics (such as the preponderance of pine in abandoned fields that previously lacked ribes) have resulted in a low level of lethal infection of young pine. Present levels of disease in these pines cannot be taken as an indication that future rust damage would not occur if *R. nigrum* were again widely cultivated in proximity to pine. In the southern Appalachians, the important host is *R. cynosbati*. This ribes grows at higher elevations than eastern white pine, co-occurs at mid-elevation, and is absent at lower elevations of the white pine distribution (Fig. 1). In the interior West, ribes are rare in certain portions of the range of white pines, such as the Davis Mountains, TX, Santa Rita, AZ, and Snake Range, NV. There are, however, diverse susceptible rust-infested communities of ribes in the Sacramento Mountains, NM and Jarbridge Mountains, NV (Vogler and Charlet 2003). Even small, isolated populations of ribes can support a rust outbreak. Examples include the Black Hills, SD; Gallinas Peak and San Francisco Mountains, NM; and the Sangre de Cristo, CO (Blodgett and Sullivan 2004). Reconnexion of areas such as these provides an initial assessment of rust hazard and the potential role of disease escape in the developing epidemic. A genetic inventory of *Cronartium ribicola* populations across its entire range would greatly improve our understanding of evolutionary potential in the pathosystem (McDonald et al. 2004, 2005, and in press; see also Zambino et al. submitted).

Disturbance and succession change the distributions and age structures of pine and ribes, alter ribes-to-pine proximity, affect ribes population structure, and influence local microclimate. Therefore, disturbance regime and successional patterns influence blister rust epidemiology, and consequently, hazard. For example, wild or prescribed fire creates regeneration opportunities for the white pine; but also benefits ribes regeneration. Ribes regenerate primarily from the soil seed-bank or from bases of surviving plants. As the overstory develops, the abundance of each ribes species declines according to its shade tolerance. With succession, species are lost from the ribes community and others assume dominance. In the moister forest habitats, canopy closure can virtually eliminate the shrubby component of the understory. Within the range of western white pine in the Inland Northwest, four well-represented species of ribes ranked by epidemiological importance are *Ribes hudsonianum* var. petiolare, *R. inermis*, *R. viscosissimum*, and *R. lacustre* (Mielke et al. 1937). Shade tolerance is lowest for *R. viscosissimum*, intermediate for *R. inermis* and *R. hudsonianum*, and greatest for *R. lacustre*. Although neither *R. viscosissimum* nor *R. lacustre* rank high for importance, where they co-occur on some sites, *R. viscosissimum* produce abundant urediniospores which infect *R. lacustre* which then produces teliospores (Zambino, pers. observation). Loss of *R.

<table>
<thead>
<tr>
<th>Species</th>
<th>Section</th>
<th>Class</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>R. nigrum</em></td>
<td>black</td>
<td>A</td>
<td>cultivated</td>
</tr>
<tr>
<td><em>R. hudsonianum</em></td>
<td>black</td>
<td>A</td>
<td>= <em>R. petiolare</em>; northern (to n. NV and UT, w WY); wet</td>
</tr>
<tr>
<td><em>R. americanum</em></td>
<td>black</td>
<td>C</td>
<td>western</td>
</tr>
<tr>
<td><em>R. wolfii</em></td>
<td>black</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td><em>R. laxiflorum</em></td>
<td>dwarf</td>
<td>B</td>
<td>= <em>R. coloradense</em>; northwestern to UT (one locality) and CO</td>
</tr>
<tr>
<td><em>R. rubrum</em></td>
<td>red</td>
<td>D</td>
<td>cultivated</td>
</tr>
<tr>
<td><em>R. viscosissimum</em></td>
<td>stinky</td>
<td>B</td>
<td>western; disturbed</td>
</tr>
<tr>
<td><em>R. cereum</em></td>
<td>stinky</td>
<td>D</td>
<td>includes several ssp.; western</td>
</tr>
<tr>
<td><em>R. mescalerium</em></td>
<td>stinky</td>
<td>D</td>
<td>NM endemic</td>
</tr>
<tr>
<td><em>R. aureum</em></td>
<td>golden</td>
<td>C</td>
<td>native; cultivated; western; important host for <em>C. occidentale</em></td>
</tr>
<tr>
<td><em>R. lacustre</em></td>
<td>spiny</td>
<td>B</td>
<td>western; forest</td>
</tr>
<tr>
<td><em>R. montigenum</em></td>
<td>spiny</td>
<td>B</td>
<td>western; alpine</td>
</tr>
<tr>
<td><em>R. pinetorum</em></td>
<td>gooseberry</td>
<td>A</td>
<td>NM and AZ</td>
</tr>
<tr>
<td><em>R. inermis</em></td>
<td>gooseberry</td>
<td>B</td>
<td>western; may hybridize with <em>R. o. setosum</em></td>
</tr>
<tr>
<td><em>R. hirtellum</em></td>
<td>gooseberry</td>
<td>B</td>
<td>eastern but listed for SD</td>
</tr>
<tr>
<td><em>R. niveum</em></td>
<td>gooseberry</td>
<td>B–C</td>
<td>northwestern and CO (one locality)</td>
</tr>
<tr>
<td><em>R. oxyacanthoides</em></td>
<td>gooseberry</td>
<td>D</td>
<td>with var. <em>hendersonii</em> (NV) and <em>setosum</em>; UT, ID, WY, SD</td>
</tr>
<tr>
<td><em>R. leptanthum</em></td>
<td>gooseberry</td>
<td>D</td>
<td>AZ, NM, UT</td>
</tr>
<tr>
<td><em>R. velutinum</em></td>
<td>gooseberry</td>
<td>B</td>
<td>AZ, NM, UT, UT; NV; important host for <em>C. occidentale</em></td>
</tr>
</tbody>
</table>

*a* southern ID, WY, CO, UT, NM, and adjacent portions of MT, SD, AZ, and NV.

*b* Ribes are classified on the basis of their susceptibility to infection and capacity to produce telia under field conditions.

“Class A” species have the highest potential for contributing to spread; “Class B” species have moderate or variable potential; “Class C” species have low potential; and “Class D” species are usually insignificant.
viscosissimum after canopy closure ends this synergism.

Ribes genetics

One might expect that a ribes species or community with similar densities and other characteristics would contribute to hazard in a simple, predictable manner at every site. Ribes at different sites, however, belong to different populations with different histories and different genetics and frequencies of resistance mechanisms. Site-to-site differences of ribes for resistance to blister rust have been largely unexplored. Variability in resistance among individuals within a species is well documented by early and repeated studies (Hahn 1928; Kimmey 1938; Mielke et al. 1937). Resistance in wild ribes may be due to both R-gene and multigenic inheritance, as demonstrated for cultivated, European species (reviewed in Zambino and McDonald 2004). A complex distribution of R-gene resistance within the genus Ribes is also further indicated by recently identified clones of North American species that are completely immune to C. ribicola but susceptible to C. occidentale, and clones of the European species R. nigrum that are highly susceptible to C. ribicola but immune to C. occidentale (Zambino pers. observation). The highest proportion of apparently immune clones of North American ribes reported by Mielke and others (1937) was about 15 percent in Ribes hudsonianum var. petiolaris—a taxon usually considered highly favorable for rust development.

Ribes populations exposed to severe and prolonged rust may have changed significantly in resistance from those with only recent or moderately low rust presence. Rust has been shown to cause premature defoliation in both European and North American species of ribes and to lower survival in winter dormancy. The genetic cost of supporting rust infections could also cause more susceptible ribes to be prematurely stressed and to drop out of the population more readily, even before low light levels usually limiting to survival have been reached. As mentioned, ribes can regenerate from banked seeds even several hundreds of years after canopy closure. There is the potential that ribes established after a severe disturbance may “reset the clock” that is, regain a high susceptibility similar to pre-rust levels, even at sites where resistance had increased over the century. These topics require further investigation.

Meteorology Influences
Development and Dispersal

Environmental factors besides moisture, especially temperature regime and air circulation, control the timing and pattern of rust spread (Van Arsdel 1965b). Temperature affects rate of pine-host tissue colonization, development of spore-bearing structures, spore release, spore germination, and infection. Temperature responses differ at the various stages of the life cycle. Wind is important for carrying spores for local spread and long distance dispersal. Desiccation and solar radiation each affect spore viability, with differences among spore stages. Because reproduction and dispersal of blister rust is strongly affected by meteorological phenomena, climatic information, including daily and seasonal patterns and storm frequencies, are useful for predicting hazard. The following, selective review summarizes the micrometeorological requirements for blister rust as described by Spaulding (1922b), Spaulding and Rathbun-Gravatt (1925, 1926a, 1926b), Hirt (1935, 1937, 1942), Mielke (1943), Riker et al. (1947), Van Arsdel (1952, 1954), Van Arsdal et al. (1956), Bega (1959, 1960), McDonald and Andrews (1980), and Zambino et al. (1997). These investigations of rust spores and infected plants represent a limited number of populations; various authors use different rust and host populations and methods; they report different responses. Further refinements can be expected as behaviors of newly established populations in different regions are studied over time.
Aecial Stage

Aeciospores produced from perennial cankers on white pines can disperse few to hundreds of kilometers, land, and infect telial hosts (mostly ribes). Aeciospores are usually produced in the spring, over a wide range of moderately warm temperatures from 16 to 28° C. They are discharged in day or night when the air is saturated and within a few hours of blister opening. Fresh aecia are uncommonly found at some high-elevation sites as late as the end of August. McDonald et al. (in press) report that such late-season spores from whitebark pine at one site in northern Idaho were still infective on locally-collected specimens of *Pedicularis racemosa* under artificial inoculation conditions. Although it is unknown whether local conditions would allow natural infection this late in the growing season, leaves and flowers were still being produced on the *Pedicularis*, and all the local hosts (*Ribes hudsonianum var. petiolare, R. lacustre, R. inerme, P. racemosa,* and *Castilleja miniata*) bore young telia. If recent infections from aeciospores had occurred there, the environment appeared to favor the teliospore stage on the alternate hosts required to infect pine, as predicted by local temperature regimes (see below). So, aeciospores produced even late in the season could still have epidemiological importance.

Aeciospores are stimulated to germinate by freezing or exposure to 32 to 36° C for 8 hrs, but germination is restricted if they are exposed for 36 hrs to 36° C (Fig. 2). Free water or saturated air is required, but the necessary duration of exposure depends on temperature, varying from 5 to 25 hrs over a temperature range from 8 to 24° C. The minimum duration is 5 hrs at an optimum temperature of 12 to 16° C; minimum temperature for germination is from 5 to 8° C; maximum is from 19 to 24° C. Under normal conditions, spores can remain viable for several weeks to an extreme of 6 months (longer under low-temperature storage; Zambino 1997). Strong light rapidly reduces viability of most spores. The minimum time for ribes infection by aeciospores is 7 to 8 days of moderately warm weather for production of aeciospores, followed by 12 hrs of saturated air for spore release, and another 5 hrs at 16 to 20° C at >97% RH and wet ribes leaves for germination and infection.

Uredinial Stage

Urediniospores are produced on ribes leaves and dispersed within the same bush, to nearby bushes or, less frequently, to distant bushes. The period of urediniospore production starts 7 to 24 days after initial infection, up until mid-August when conditions are usually unfavorable for urediniospore production. The optimum temperature range for urediniospore production is from 14 to 20° C, with a nocturnal lower limit of 2° C. Formation of urediniospores can be inhibited by several days exposure to temperatures above 35° C before resuming optimum conditions, and prevented by 10 days exposure to this temperature. Under most natural conditions, viability of mature urediniospores declines rapidly and is less than 2 weeks; germination is best from fresh spores but can be poor in cool weather. Germination begins 4 to 6 hrs after spores are released and exposed to warm, saturated air and free water on the ribes leaves. A minimum temperature for germination ranges from 8 to 16° C, the maximum is from 25 to 28° C; and the optimum is 14 to 20° C (Fig. 2). Provided that plants have not been exposed to very warm temperatures, the scenario for ribes to be infected by urediniospores is a daytime temperature of 16 to 28° C for 7 to 24 days to produce the spores, followed by 4 to 12 hrs at 20 to 24° C, saturated air, and wet target leaves for urediniospore release, germination, and fresh infection.

Telial Stage

Teliospores are produced on alternate hosts and germinate in place to produce probasidia and basidiospores. The season of teliospore production on ribes begins a minimum of 2 weeks after aecial or uredinial infection. Production is stimulated by cool temperatures but inhibited by 3 consecutive days above 28° C, nocturnal lows above 20° C, or daily highs above 35° C. The favorable range for teliospore formation is 1 to 20° C, with optimum at 16° C. Warm (>20° C) or cold (0° for 12 hrs) temperatures at formation affect germination, but spores can recover from freezing. Teliospores remain viable for...
several weeks and germinate best at an age of 4 to 9 days, after exposure for about 12 hours to free moisture (rain, fog, or dew), and at temperatures (Fig. 2) from 10 to 18 (optimum 16)° C (range 0–1 to 21–22° C).

**Basidial Stage**

Basidiospores\(^1\) are delicate, short-lived spores produced from telia on the non-pine host, and are dispersed up to several kilometers to infect white pine. The season for basidiospore production begins when viable teliospores are present and moisture is present as free water or relative humidity exceeds 97% (germination is best at 100% RH when teliospores are in contact with water). The temperature range for basidiospore germination is from 0–1 to 20–21° C, but is best at 10 to 18° C. Basidiospore viability and germination are inhibited by a number of factors: extremes of drying and wetting (but cycles of moderate drying and re-wetting at 4° C can enhance germination rate, Zambino, pers. observation), temperature exceeding 21° C (>35° C lethal), exposure to direct light, sustained low humidity at moderate temperatures (26 hrs at 58% RH). The minimum time required at favorable temperature and humidity for the rust to proceed from teliospores to infections is variously reported as about 11 to 19 hrs; the process is usually complete by 36 hrs. Basidiospores may either germinate to form a germination tube for pine infection, or may produce secondary spores for re-dispersal. The minimum time course for pine infection given that ribes have been infected is 2 weeks of cool temperatures to produce telia, then 4 to 9 days for teliospore maturation, then (provided there had been no exposure to a 5-day period of temperature >35° C that would reduce pine infection or prolonged temperature >21° C to reduce infection), a minimum period of 48 hrs with saturated air, free moisture on leaves, and a cool temperature (<20° C); the latter provides for teliospore germination, basidial development, basidiospore germination, and germination tube penetration.

**Temperature Effect**

Although moisture has long been recognized as a potential constraint on rust spread, temperature can also be the critical factor in some locations. Portions of the north-central states and southern California are too warm for pine infection. In other parts of southern California, pine infections may only have resulted from infection in spring (McDonald 1996).

The high western mountains—above 2700 m (9000 ft) in northern Rockies and Yellowstone plateau (Hendrickson 1970) or 3000 m (10,000 ft) in the Southwest—can be too cold for development and intensification on the ribs. Although each spore and rust infection is actually responding to its own microclimatic environment, meteorological summaries and models can be statistically useful for conditions at larger scales. Canopy and site topography may affect the temperature and control the outcome for individual rust spores or infections, but the sheer numbers in rust populations and large sizes of air masses allow properly adjusted climatic averages to be meaningful for predicting epidemics.

For modeling at the landscape scale, Kears (2005 and these proceedings) demonstrates that monthly, climatic statistics adjusted to specific sites correlate well with corresponding rust incidence. For modeling at the synoptic scale\(^2\), Frank et al. (in preparation) illustrates that upper-level weather data with a 6-hr resolution could identify periods when surface temperature and humidity might be sufficiently favorable to allow viable long-distance dispersal and successful establishment of a new rust outbreak area.

Graphs of the percent germination of basidiospores by time and temperature (Fig. 3; unpublished data from R. Krebill) illustrate that rust populations might be adapted to different temperature regimes. Graphs for rusts originating from both limber pine in a warmer environment and whitebark pine in a cooler environment demonstrate that regardless of source, teliospores produce fewer basidiospores and production is delayed at sub-optimal temperatures. However, when teliospores of rust from whitebark pine were exposed to cool temperatures at which rust from the limber pine rust produced no basidiospores, some teliospores did germinate and produce basidiospores. Also, the upper elevation rust had a longer delay prior to teliospore germination at all temperatures, suggesting an adaptation that reserves teliospores for germination during periods that will be sufficiently long to allow the complete pine infection process. This implies that at the cooler elevations where whitebark pines are found, there is a selected portion of the rust population adapted to completing its life cycle under locally prevalent conditions.

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1 Sporidia in older literature—terminology here follows Ziller (1974).

2 Pertaining to meteorological phenomena occurring at the scale of migratory high and low pressure systems of the lower troposphere, with length scale of 1000–2500 km and time scales exceeding 24 hrs (Whiteman, Mountain Meteorology).
Dispersal

There are two forms of dispersal—diffusion and transport. Diffusion occurs on a local scale as the density of spores for deposition and infection from a single source declines in magnitude with distance according to a power function (e.g., the square root function). Although diffusion-like models were developed in early rust studies, Van Arsdel (1967) demonstrated that spores are also commonly dispersed by transport within an air mass (Fig. 4). Transport occurs at multiple scales as a result of laminar flow of distinct air masses differing in density, which are eventually disrupted by turbulent mixing. Such transport provides a “pipeline” which directs spores from an inoculum source to a corresponding deposition sink; the resulting infection pattern at the sink can appear in spatially diffuse as well as in defined patches. If source–sink transport were infrequent or shifting, the resulting infection pattern would appear random or as indistinct patches. Transport may disperse rust spores locally, as with valley air circulation (this occurs frequently over a length scale of kilometers); across landscapes, as where land–water temperature differences drive air circulation (occasionally and over tens of kilometers); and even regionally, as with flow of large air masses and long distance dispersal (this occurs rarely and over many hundreds of kilometers).

As an example of transport at the local scale, Quick (1962) relates canyon physiography to pine infection and basidiospore dispersal from individual ribes bushes in California. Van Arsdel (1965a, 1967) describes intermediate, landscape scale dispersal in Wisconsin from areas of high density ribes by laminar transport with an air circulation pattern driven by land–water temperature differences. Frank et al. (in preparation) discuss the relative likelihood of transport with large air masses moving from the Sierra Nevada eastward to the Rocky Mountains and Southwest.

Distribution

The observed patterns of blister rust canker distribution are the result of dispersal, infection, establishment, and recognition. Not only must spores be transported to a site, they must germinate and infect a host, intensify to a detectible level, and persist over years until chance recognition. Patterns of infection vary the most where distributions of hosts are discrete and climates favoring infection are irregularly distributed or are only marginally favorable. At a regional scale, new outbreaks or
satellite infestations are seen as young cankers (3 to 25 years old) that are quite remote from previously reported infestations. Frank et al. (in preparation) suggest that long distance dispersal can transport spores, at least occasionally, to nearly all sites downwind of a large infestation, but that further spread from and intensification at these remote sites may be limited by conditions at the deposition site. Important site factors are the distribution and size of the local telial host population, its proximity to pine, and the extent or frequency of a suitable environment (temperature and humidity regime). On a local scale, we recognize four distinctive patterns of canker distribution. Where a discrete inoculum source is nearby and climate is favorable, the number of cankers decreases with distance from source. Where there are many recognized or unrecognized sources of rust at various distances from the observed pines, canker distribution appears to be random—incorrectly interpreted as a lack of relationship between ribes and canker abundance. Where climate, topography, and pine–ribes distributions develop strong source–sink relationship, the rust occurs in discrete patches. Where the source–sink relationship is less strong, patches are less distinct. Where a cold climate usually prevents infection of the ribes in very close proximity to the pine (e.g., Ribes montigenum and whitebark pine), rust abundance on the pine is usually low and random; at these sites infection may result from infrequent transport from ribes at lower, warmer sites. The newly identified, non-ribes alternate hosts (Pedicularis and Castilleja species; McDonald et al. in press; Zambino et al., in press) are abundant in some upper montane sites, but their epidemiology importance and interactions require more investigation.

**Hazard Management Zones**

An understanding of blister rust epidemiology allows one to better map hazard and therefore manage white pine ecosystems. Hazard zones are two or more levels of potential for rust damage as mapped at a regional scale. Assumptions are that the combined presence of rust and its hosts and climate are predictable and that pines are susceptible to disease. Site hazard rating makes similar assumptions but is scaled to a defined stand or locality rather than across a landscape. Zone boundaries are delimited by pine and ribes distribution with primary consideration of regional climate and their modification by elevation, soil type, landform, and landscape structure (land–water interactions). Zones are intended to reflect the potential abundance, distribution, and infection frequency of the rust on pine. Zones should identify where different sets of management options are most appropriate and effective.

The first mapping of rust hazard zones is described by Van Arsdel (1961) for the Lake States and is updated here using a recent review of the historic, early pattern of rust spread in southern Michigan (Fig. 5). Van Arsdel (1961)

![Figure 5—Hazard zones from white pine blister rust in the Lake States, updated 2005.](image)

describes five infection-level zones and their corresponding control needs. Zone 0 includes all areas with a mean July temperature above 23°C, which are too warm for rust development. No infestation or damage is expected.

Zone 1 includes areas where the regional climate is too warm and dry for the blister rust infection in all but those few sites where several microclimatic factors combine to moderate temperature and humidity. These sites are small canopy openings (gap diameter less than tree height) at the base of a slope where nocturnal, cold air collects. Rust infection is infrequent, and even without disease control, pine losses are expected at less than 5 percent. White pine could be cultivated without disease control.

Zone 2 is that area where the regional climate is too warm and dry for blister rust infection in most locations but a single, microclimatic factor (Fig. 6) is sufficient to moderate temperature and humidity, allowing rust infection. Infections may occur on such sites as the open or brushy ground northwest of the edge of a woodlot or row of trees, which would shade morning sun and prolong the dew period. Disease control is usually not required for cultivation of white pine.

Zone 3 is that area where the regional climate is cool and moist enough for pines to become infected every
Figure 6—Drainage of cold air at night and radiant heat loss make locally cool wet spots.

few years, even on sites without any features that modify microclimate. In open fields and brush, scattered trees may be infected in the lower 2 m of crown. Damage can become severe, and disease control is recommended for commercial white pine plantations.

Zone 4 is that area where the regional climate is so cool and wet that all sites are suitable for infestation, and infection may occur throughout the crown. The extent of infestation and distribution of infections indicate that rust dispersal can occur over distances of several kilometers, resulting in severe damage. Special disease control or avoidance measures are necessary to minimize disease impacts. Numerous hazard maps and general observations for blister rust have been published; some are reviewed here. Although control maps were routinely prepared (e.g., Ball 1949), Van Arsdale (1961, 1964 and Fig. 5) expanded the mapping concept to include a more explicit consideration of climatic influences and generate a regional map of potential damage for the Lake States. Other relevant factors to be considered are the lack of ribes locally due to eradication or site characteristics (e.g., sandy soil) and lack of inoculum due to early defoliation of the locally prevalent ribes species. McDonald et al. (2004, 2005) suggest that over time, natural selection would alter the population structures of hosts and pathogens in regards to resistance and aggressiveness. Less lethal rust infections on pine, whether due to host or pathogen, would tend to survive and propagate after epidemics had removed susceptible trees, moving the pathosystem to behaviour more similar to those of endemic rusts. The regional and local effects of global climate change could also alter environmental factors with sufficient rapidity and magnitude to affect host and pathogen abilities to reproduce. Considering these historic, ecological, genetic, and climatic factors along with changing management objectives, a hazard map should be recognized as a spatial model with numerous, explicit and implicit assumptions, data dependencies, and resolution. A hazard map should be judged not for accuracy in predicting current levels of disease incidence or loss but for leading to appropriate management within a given policy framework (such as “sustaining healthy ecosystems”).

Several regions have been mapped for potential rust damage. Brown et al. (1999) adapted available map data to produce a GIS hazard map for Minnesota. Ostrofsky et al. (1988) recognized that prevailing incidence in Maine was less than predicted by Charlton (1963), and suggested that the significant reduction in rust was due to effective ribes eradication. Later, Lombard and Bofinger (1999) concluded that rust distribution in New Hampshire was consistent with Charlton (1963). Regional hazard maps were prepared for Ontario (Gross 1985) and Quebec (Lavallée 1986). For the Sacramento Mountains of New Mexico, Geils et al. (1999) mapped expected rust potential early in the outbreak history. Smith and Hoffman (2001) modeled rust distribution for southern Idaho and western Wyoming using site and stand descriptors. Kearns (2005 and these proceedings) developed alternative models and maps for southeastern Wyoming and Colorado using site, vegetation and climatic data. In regions such as British Columbia at which zonal hazard is uniformly high and management is intensive, site and stand hazard rating have been more commonly used than hazard zone mapping (e.g., Hunt 1983 or McDonald 2000).

Rates and incidence of infection in current, naturally regenerating stands in many of the areas in North America first affected by rust are much lower than during the early outbreak, and cankers on many trees in these areas are slow-growing, with little or no sporulation. Such changes in blister rust pathosystems do not invalidate the concept of rust hazard, but reaffirm the idea that hazard is relative to cultural treatments employed, including resistance and ribes density, and that ecosystem recovery might also be modeled. In the interior West, populations of white pines, ribes, and rust are more insular than the expansive forests of the Pacific and northern regions. Hazard zones may be especially useful where there are large, distinct differences in rust potential from forest to forest. Finally, reassessment of management tools associated with hazard zones may be needed, as changes to pine and ribes populations in response to decades of exposure to blister rust become recognized.
Several taxa of rusts occur on either white pines or ribes (Goodding and Bethel 1926). These rusts include *Coleosporium ribicola* (pinyon leaf rust); *Puccinia* spp. (on ribes and sedges); *Cronartium ribicola* (white pine blister rust); *Cronartium occidentale* (pinyon blister rust, on ribes and pinyon), and *Melampsora ribesii-purpureae* (on ribes and willows). Identification of these rusts can be made from the appearance of macroscopic characteristics of the rust on ribes (see Van Arsdel and Geils 2004 for a key and illustrations). *Melampsora ribesii-purpureae* is a typical rust of the *Melampsora epitea*–complex, but appears to be quite rare (see Ziller 1974). The *Puccinia* rusts are uncommon, usually seen as a single or few cup-like pustules, visually quite distinct from *Cronartium* and *Coleosporium* rusts. *Coleosporium ribicola* is common in the Southwest, especially on *Ribes cereum* and *R. leptanthum*; it may be found in other regions, on other ribes (*R. inerme*), and many kilometers from the nearest pinyon (Hedgcock et al. 1918). The telia of *Coleosporium ribicola* appear as a waxy cushion and should not be mistaken for uredinia of a *Cronartium*. On ribes, the two blister rust fungi *Cronartium ribicola* and *C. occidentale* are indistinguishable by their uredinia (Kimmey 1946, Miller 1967). Differences between the telia of the two species are recognizable if many leaves are carefully examined, but they can still be easily mistaken (table 3, previous page). Ribes host and time of onset for telia formation are more useful characteristics than differences in form, color, extent, pattern and development of infections, which are not consistently reliable for most observers. Vogler (these proceedings) resolves this difficulty with a molecular determination technique.

Among species of *Cronartium* (*C. ribicola*, related Asian rusts, *C. occidentale*, and others) there is notable variation in host range, pathogenicity, environmental requirements, life history, population genetics, evolutionary history and tendencies (Vogler and Bruns 1998; McDonald et al. 2005; Zambino et al., submitted). McDonald et al. (2005) suggest given this diversity, the pine stem rusts have a potential for genetic or epigenetic adaptation (or exapation, see Gould 2002 page 43), allowing a rust to exploit new hosts and new environments. This could include the aforementioned changes in aggressiveness, exploitation of non-ribes alternate hosts as utilized at blister rust’s center of diversity in Asia, but only recently discovered in North America, and adaptations for sporulation and spore germination at different ranges of temperatures than have been observed (e.g., McDonald et al. 2005, McDonald et al. submitted, Zambino et al.)

### Diversity of Rust Species and its Relevance to White Pine Blister Rust Pathosystems

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### Table 3—Comparison of signs and symptoms of *Cronartium ribicola* and *C. occidentale* on ribes.

<table>
<thead>
<tr>
<th>Character</th>
<th><em>Cronartium ribicola</em></th>
<th><em>Cronartium occidentale</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of telial formation</td>
<td>later</td>
<td>earlier</td>
</tr>
<tr>
<td>Form of teliospore columns and telial mats</td>
<td>straight, sparse, never “fur-like” in mass</td>
<td>bent, very dense, “fur-like”,</td>
</tr>
<tr>
<td>Color of mature, un-germinated telia</td>
<td>straw brown, orange-tinge</td>
<td>dark brown</td>
</tr>
<tr>
<td>Color of older, germinated telia</td>
<td>retains orange color longer but may have lavender-purple tinge</td>
<td>soon develops a lavender-purple tinge</td>
</tr>
<tr>
<td>Extent of telial production on infected portions of the leaf</td>
<td>much of the infected portion of the leaf without telia</td>
<td>most of the infected portion of the leaf with telia</td>
</tr>
<tr>
<td>Pattern of infection on leaf</td>
<td>small, scattered spots</td>
<td>large, continuous areas</td>
</tr>
<tr>
<td>Hardman symptom characteristics&lt;sup&gt;b&lt;/sup&gt;, in order of increasing severity</td>
<td>slight necrosis by spotting (from rust or secondary fungi); general necrosis of infected area; “blister necrosis” as large raised or sunken dead areas of leaf; angular necrosis delimited by veins; necrotic areas bear uredinia but no telia.</td>
<td>telia present with no visible host reaction; chlorosis around telia visible on dorsal side of leaf; “green island effect”&lt;sup&gt;c&lt;/sup&gt;; purpling&lt;sup&gt;d&lt;/sup&gt; on dorsal leaf side</td>
</tr>
</tbody>
</table>

<sup>a</sup> Table based on descriptions by Kimmey (1946), Miller (1967), and our observations.  
<sup>b</sup> Symptoms by D. Hardman, unpublished work described by Miller (1967).  
<sup>c</sup> A condition commonly caused by leaf spot fungi where green, infected patches are surrounded by yellow or light green areas.  
<sup>d</sup> Marked by a purple-black or -brown discoloration different from the usual reddening of diseased or aging leaves.

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submitted). Future models of rust hazard as estimated potential for causing unacceptable management impacts may need to broaden from a primarily ecological perspective (host community and climate) to include a population genetic perspective with gene flow (long distance dispersal) and isolation (local adaptation).

Conclusions

A great deal has been learned about how blister rust spreads from ribes to pine and the environmental factors that constrain it. This knowledge was gained from observing the results of early Blister Rust Control programs, from research, and from various management projects. To effectively address new management objectives, spread to new regions and hosts, and other new developments, much more can be learned about rust epidemiology. Especially useful would be investigations into the development and dispersal of the rust in alpine microclimates, and changes in pine, ribes, and rust populations and responses that have occurred over time in North American pine ecosystems with long exposure to rust. This information would provide a basis to predict hazard and long-term outcomes for whitebark pine and other species and locations having only recent infection histories.

References


Left to right: Amy Eckert, Bill Woodruff, Angel Saavedra, Greg Fillip, Kathy Lewis, Kelly Burns, Brendan Ferguson, Eun-Sung Oh, Ellen “Peekaboo” Goheen, Katy Mallams, Mike Mc Williams, Kristen Fields, Aaron Smith, Everett Hansen, Blakey Lockman, Dave Shaw, Pete Angwin
White Pines and Blister Rust in Western North America: Spread, Impacts and Restoration

John W. Schwandt, John Kliejunas, Blakey Lockman and John Muir

Abstract—Since its introduction nearly 100 years ago, white pine blister rust has had tremendous impacts on most of our native 5-needle pines. The impacts of this disease coupled with changes in fire regimes, forest management, and increased bark beetle activity have effectively removed white pines from major portions of their range and resulted in major changes to ecosystems. Restoration efforts are focusing on improved genetic resistance and enhancing natural regeneration. Successful restoration programs will require substantial coordinated efforts of many disciplines from many different agencies plus broad public support.

Introduction_________________

We are part of a large group of authors preparing a general review of white pine, ribes and blister rust in North America for publication in a special edition of Forest Ecology and Management. (Geils and others 2006). Our presentation is a synopsis of this information. We discuss several aspects of recent spread and impacts of the disease, restoration activities for white pines in western North America, plus include some future needs and directions that need resolution.

Spread of Blister Rust________

Hunt (2003) discussed the probable origins of blister rust in Asia and its spread to Europe and North America in detail. In the late 1800s, forest pathologists observed blister rust damage in Europe and warned of potential consequences to white pines if blister rust were introduced in North America. By 1912-14, quarantine regulations were established in North America to prohibit white pine importation, but by 1910, blister rust already had been introduced to several locations in North America. Blister rust introduction in western North America originated from infected eastern white pine seedlings that were raised in France and shipped to Vancouver, Canada. Subsequent spread of blister rust was rapid, particularly on currants (*Ribes* species), the alternate host (Figure 1).

![Figure 1. White pine blister rust range](image)

The 1960s brought significant changes to the management of white pines and control of blister rust in the West. The emphasis changed from large-scale attempts to eradicate currants to management of stands with white pines and development of rust resistance programs. Strategies varied. In the Intermountain west, an assumption was made that western white pine could not be protected from the rust. This led to decisions to accelerate harvest of white pines, to favor other tree species, to discontinue planting western white pine, and to expand the program for developing genetic resistance. In California, surveys (Byler and Parmeter 1979) suggested that the relatively hostile environment for the pathogen in the Sierra Nevada would constrain blister rust on sugar pine to sporadic outbreaks. These could be mitigated by silvicultural prescriptions, relying on a combination of site hazard rating and removal or pruning lethally infected trees. Today however, with a few exceptions, most of the western
white pine species are infested over much of their geographic range (Table 1).

<table>
<thead>
<tr>
<th>White pine species</th>
<th>Range of blister rust</th>
</tr>
</thead>
<tbody>
<tr>
<td>Western white pine <em>Pinus monticola</em> D. Don</td>
<td>Whole range</td>
</tr>
<tr>
<td>Whitebark pine <em>P. albicaulis</em> Engel.</td>
<td>Whole range</td>
</tr>
<tr>
<td>Sugar pine <em>P. lambertiana</em> Dougl.</td>
<td>Southern Oregon to Breckenridge Mountain, Sequoia National Forest, California</td>
</tr>
<tr>
<td>Limber pine <em>P. flexilis</em> James</td>
<td>Canada to southern Colorado</td>
</tr>
<tr>
<td>Southwestern white pine <em>P. strobiformis</em> Engel.</td>
<td>Southern New Mexico</td>
</tr>
<tr>
<td>Rocky Mountain bristlecone pine <em>P. aristata</em> Engel.</td>
<td>Sangre de Cristo Mountains, Colorado</td>
</tr>
<tr>
<td>Foxtail pine <em>P. balfouriana</em> Grev. &amp; Balf.</td>
<td>Marble Mountain, Siskiyou County, California</td>
</tr>
<tr>
<td>Great Basin bristlecone pine <em>P. langeava</em> D.K. Bailey</td>
<td>currently uninfected</td>
</tr>
</tbody>
</table>

**Impacts of Blister Rust**

Blister rust impacts on ribes are generally minor, causing leaf-spotting and defoliation. However, on white pines it causes lethal cankers on branches and stems. On young trees, infected branches and stems are often girdled and killed within a few years. As trees age this process takes longer, but older trees, particularly on steep slopes or near *Ribes* bushes, are often repeatedly infected resulting in dieback of branches and stems.

Despite damage from blister rust, western white pine and sugar pine historically have been very valuable tree species. White pine lumber is valued for its even straight grain that finishes well, particularly for doors, moldings and paneling. Stumpage prices for white pine logs and for lumber typically have been at least two to three times higher than those for associated tree species, with the exception of western redcedar (*Thuja plicata* D. Don) and redwoods (*Sequoia sempervirens* (D.Don) Endl). Western white pine and sugar pine also grow considerably faster and at higher densities than most associated tree species, often by 20 to 30 percent or more, thus multiplying the value per acre. These pines exhibit superior growth and high value over a wide range of forest ecosystems, from maritime to subalpine environments. The ecological values of 5-needle pines are hard to measure, but are crucial to long term healthy ecosystems. In the past 50 years, the loss of western white pine and sugar pine as major stand components over several million acres has been dramatic. These stands are now dominated by more shade tolerant, less drought resistant, shorter lived trees which have a myriad of insect and disease problems. Forest succession has been radically changed across many watersheds (Hagle and others 2000). High-elevation white pine species generally have little commercial value but many have very high recreation value and are crucial to healthy ecosystems. These species often dominate stands around high value ski areas or other sites such as Crater Lake National Park. The viability of these critical high elevation ecosystems is now being threatened by blister rust and other agents discussed below.

In many areas such as the Inland Empire (eastern Washington to western Montana) where western white pine is a valuable commercial species, incidence of blister rust and tree mortality has drastically reduced white pine and encouraged
replacement by other less-valuable and less ecologically desirable trees. Until recently when breeding and selection programs identified genetically resistant stock, silviculture of white pine had been largely abandoned in many areas due to a pessimistic outlook for survival of white pine and sustainability as a commercial tree species.

Other Impacts and Concerns

Many white pine species have historical relationships with fire that have been greatly altered by aggressive fire suppression over the past 50 years. Although not fully understood, in ecosystems where white pines are a seral component, fire suppression has allowed more shade tolerant species to out-compete white pines. This is apparent in many forests with western white pine, sugar pine, and whitebark pine. Conversely, lack of fire has allowed limber pine to encroach into the drier shrub/grassland interface. On these sites, trees appear to be overly stressed from being offsite, which is compounded by the current drought in the west (Taylor and Schwandt 1998). Diseases, which are normally minor in their effect, have had a greater impact on these stressed limber pines. Efforts are now underway in some areas to increase natural and prescribed burning to encourage natural regeneration. However, caution must be used as Rochefort (pers. com.) found some fires may burn so hot they inhibit natural regeneration by reducing soil nutrients.

Mountain pine beetle (Dendroctonus ponderosae Hopkins) is currently of great concern to white pines in western North America. Mountain pine beetle, which is the most destructive bark beetle in western North America (Furniss and Carolin 1977), attacks and kills apparently healthy pine trees. Under the right conditions, populations can build into epidemic proportions very quickly.

Mountain pine beetle outbreaks have occurred a number of times over the last century. An outbreak in the early 1900s originated in lodgepole pine then apparently "spilled over" into other pine species, including whitebark and other white pines. This outbreak created some of the high elevation "ghost forests" of whitebark pine in Idaho and Montana discussed by Ciesla and Furniss (1975), Kipfmueller and others (2002), and Logan and Powell (2001). A later outbreak in the 1970's and 1980's also originated in lodgepole pine, and subsequently caused widespread mortality in whitebark pine (Kipfmueller and others 2002). Observations of the earlier mountain pine beetle outbreak in whitebark pine indicated it was independent of nearby infested lodgepole pine stands (Gibson, 1939). In other places, mountain pine beetle populations built up in lodgepole pine, and then moved into the whitebark pine stands (Bartos and Gibson 1990). Climatic conditions at higher elevations where whitebark pine is found are thought to be unfavorable to the buildup of mountain pine beetle populations (Logan and Powell 2001, Amman et al. 1973).

Mountain pine beetle populations are again at outbreak levels in lodgepole pine stands, but they are also in outbreak numbers in whitebark and limber pine stands not necessarily associated with lodgepole pine stands. An example of such a buildup can be found in Yellowstone National Park. Mortality in whitebark pines is quite high, reaching 95 trees per acre over 3 years (2001 to 2004) in some stands (Gibson 2004). This same buildup has been observed throughout the Inland Northwest. Mountain pine beetle is also quite destructive in other white pines, causing mortality in larger sugar pines and western white pines.

Whatever the cause of recent outbreaks of mountain pine beetle, mature, seed-bearing whitebark pine trees are being killed, which is cause for great concern. With each outbreak that occurs, there are fewer and fewer mature whitebark pines, due to the continual presence of white pine blister rust killing all size classes of white pines. What is most alarming, is mountain pine beetle is not just attacking trees heavily infected by blister rust, but has shown a preference for lightly to noninfected individuals, particularly during endemic population levels of the beetles (Kegley and others 2004). This preference for healthy, mature whitebark pine is of great concern to the recent resistance breeding program for whitebark pine. Seed-producing trees in high elevation whitebark pine stands have been compromised by blister rust since its introduction, and now mountain pine beetle is attacking the remaining mature seed-bearing trees. Mountain pine beetle and white pine blister rust together are greatly impacting the reproductive potential and survival of this high elevation white pine.

Restoration of White pines

Current efforts to restore white pines have shifted from eradication of Ribes to breeding programs to capture and concentrate naturally occurring resistance and silvicultural treatments such as pruning, thinning, burning, and planting to improve natural regeneration and survival of planted stock. Breeding programs for western white pine and sugar pine have been established for many years. However, breeding
programs for other species have barely begun or are non-existent.

Many forest organizations are actively identifying trees with phenotypic resistance to blister rust that could be included in potential breeding programs. Unfortunately candidate trees for high elevation species may be difficult to protect from bark beetle and wildlife depredation.

Wildlife depredation can be reduced by caging cones and studies are currently showing promise in using pheromones to prevent mountain pine beetle attacks in whitebark pine (Kegley and others 2004). However, caging cones and applying pheromones to isolated trees on an annual basis could be quite expensive.

As blister rust continues to spread, suppression and prevention efforts are focusing on surveying and monitoring white pine populations to determine spread and intensification over time, and to detect infections in new areas and hosts. Many areas are now installing permanent monitoring plots using a standardized format (Tomback and others 2005). Plots are being established across the range of all species, and especially the non-commercial species that have had little monitoring in the past. A west-wide data base to store survey results for limber and whitebark pine has just been developed (Lockman and others 2004).

**Future Needs and Direction**

We have learned much about blister rust since its introduction nearly 100 years ago. However, there is much we still don't know about the rust and its interaction with the various hosts. For example, we don't know how much the rust varies in virulence between east and west coasts and the potential for new races with differences in virulence, aggressiveness and adaptation to different climates. The nationwide shipping of stock is a real threat to spreading blister rust. Another concern is that the increasing cultivation of Ribes sp. in many areas could form a bridge for spread and hybridization of eastern and western populations of C. ribicola (Muir and Hunt 2000).

White pine blister rust will probably continue to spread and intensify across the range of our native 5-needle pines. Therefore we need to explore ways to monitor and slow this advance, and find ways to help trees adapt to it. The most effective strategy to reduce impacts from the disease is to develop and plant trees with natural resistance. However, if there is no active program that will provide planting opportunities, there is little reason to develop improved genetic material. Additionally, much of the distribution of high elevation species may be relatively inaccessible or in areas where regular silvicultural practices such as thinning are not options.

The occurrence and distribution of resistance mechanisms of many 5-needle pine species have not yet been determined. Seeding or planting trees with improved blister rust resistance in wilderness areas may present both physical and political challenges.

Effects of fire and other silvicultural practices on both the trees and alternate hosts are not well understood and little is known about the feasibility of planting seed rather than seedlings. We know that wildfires and other natural events will occur at some point, and need to be ready to take advantage of any planting opportunities these might create.

Many of these species occupy harsh sites where little other vegetation can survive, and if white pine blister rust prevents these sites from being regenerated, these fragile ecosystems may be irreparably altered. Species such as whitebark pine that have close relationships with wildlife present additional challenges. Clark’s nutcrackers that spread the seeds do not cache seeds very successfully in areas that have not been burned and may leave areas without suitable cone crops.

Substantial, concerted efforts must be undertaken to restore native white pine species in the face of an introduced pathogen. Otherwise, species may eventually be lost from major portions of their native ranges, and many will probably become threatened or endangered species. The loss of white pines would have a ripple effect across ecosystems. In lower elevations, successional pathways may be altered such that white pines are replaced by shorter lived species that are much more susceptible to insect and disease agents. In high elevations, snow pack, water quality, and wildlife as well as species diversity and successional trends may be negatively impacted if white pines are lost.

For restoration to be successful, a coordinated effort will be required by scientists of many different disciplines, and by foresters and managers of many different public and private agencies. Restoration strategies will need to address all 5-needle species and deal with the differences in blister rust infection that may occur across the range of some species. In all these efforts, public interest and support will be crucial in implementing restoration activities.
Acknowledgements

This presentation was greatly facilitated by the kind and enthusiastic willingness of our co-authors and colleagues to provide background information, references, and their insights. However, any errors, omissions or oversights in this presentation are our responsibility.

Literature Cited


White Pine Blister Rust on High Elevation White Pines in California

Patricia Maloney, Dan Duriscoe, Dick Smith, Deems Burton, Dean Davis, John Pickett, Randy Cousineau, and Joan Dunlap

California is home to western white (Pinus monticola), whitebark (P. albicaulis), limber (P. flexilis), foxtail (P. balfouriana), and Great Basin bristlecone (P. longaeva) pines, i.e., five white pine species growing at high-elevations in the State and native to the United States. These species are key components of their forest ecosystems, contributing to ecological functions such as species diversity, wildlife habitat, and hydrology. While the distribution and effects of white pine blister rust (Cronartium ribicola) have been well documented on sugar pine, less is known about the effects of this introduced pathogen on high-elevation white pines in the State.

In 2004 and 2005, a network of 114 long-term monitoring plots was established to follow the initial incidence, if any, of white pine blister rust on these white pines. Permanent plots were established throughout the range of each species; from northwestern California and the Klamath mountains to the southern Cascades, Sierra Nevada, Great Basin ranges, and southern California mountains. Permanent plots were 30 x 50 m, with the length adjustable to include 50 trees of which at least 30 were alive. Plot data included landscape features, e.g., GPS coordinates, elevation, slope percentage, aspect, and topographic position, and biological factors, e.g., plant association, Ribes ground cover, presence of rust on Ribes, rust phenology on pine, white pine regeneration, presence/absence of Pedicularis and/or Castilleja and presence/absence of the Clark’s Nutcracker (Nucifraga columbiana). Individual tree data included species name, diameter, live/dead status of tree, crown position and condition, presence/absence of cones, presence/absence of blister rust, number of branch and stem cankers and whether cankers were active or inactive, unknown flagging, and presence/absence of mountain pine beetle and other pests.

No blister rust was found on Great Basin bristlecone pine, limber pine, or the southern population of foxtail pine. Statewide averages for rust on western white pine were 15.3% (range: 0-92%), on whitebark pine, 11.7% (range: 0-71%), and on the northern foxtail pine, 14.95% (range: 2-33%). Moderate to high rust incidence occurred in 3 regions of CA: northwest California, north central Sierra Nevada, and the southern Sierra Nevada (west slope). Average rust incidence was relatively higher for plots located on upper slopes compared to other topographic positions (e.g., lower slopes, benches, and ridgetops). Topographic position of high-elevation white pines may be a factor in blister rust occurrence as they are subject to local mountain wind currents as well as regional circulation patterns that may disperse inoculum from lower elevation sources. There were no observations of infected Ribes in upper montane and subalpine forests in California during our study period but infections on Ribes have been observed in lower montane mixed-conifer forests. Lower montane forests may be a potential source of white pine blister rust inoculum for high elevation white pine forests.

Data analysis will continue and relate disease incidence to a number of environmental parameters, specifically climate data (generated from PRISM models for these high elevation plots as well as lower montane forests) to determine the relationship between rust incidence and climatic conditions in high-elevation white pine forests of California.
White Pine Blister Rust in High-Elevation White Pines: Screening for Simply-Inherited, Hypersensitive Resistance

Detlev R. Vogler, Annette Delfino-Mix, and Anna W. Schoettle

Introduction

Recent concern about survival and recovery of high-elevation white pine ecosystems has returned white pine blister rust (caused by *Cronartium ribicola*) to prominence as a significant threat to forest health in the western U.S. (Samman et al., 2003). This, in turn, has spurred new research into potential rust-resistance mechanisms in high-elevation white pines, including whitebark (*Pinus albicaulis*), foxtail (*P. balfouriana*), Rocky Mountain bristlecone (*P. aristata*), and Great Basin bristlecone (*P. longaeva*).

The impacts of *C. ribicola* on low- and mid-elevation western white (*P. monticola*) and sugar pine (*P. lambertiana*) are well documented. Although limber (*P. flexilis*) and whitebark pine have been infected for over 60 years in the northern United States, the consequences of the disease for these ecosystems are only just becoming recognized (see Tomback et al., 2001). The disease continues to spread into the southern species and populations, including southwestern white pine (*P. strobiformis*), foxtail, and Rocky Mountain bristlecone pine; Great Basin bristlecone pine is the only North American white pine not yet infected with the disease in the field. Blister rust is likely to impact the high elevation species’ distributions, their population dynamics, and the functioning of their ecosystems (Schoettle, 2004).

The role major genes can play in fortifying the high-elevation white pine species against the impacts of the fungus deserves greater attention. Compared to non-specific, complexly-inherited forms of resistance*, relatively simply-inherited, specific mechanisms that prevent the pathogen from growing out of infected needles and into branches, thereby preventing sporulation, remain potent tools for reducing the impact of blister rust. We report here early work in bringing knowledge gained from the lower-elevation white pine species that have been screened intensively for major gene interactions with the blister rust fungus, to greenhouse inoculation studies of resistance in high-elevation white pines.

Before proceeding, it is important to address three terms that are fundamental to our discussion, and to substantiate them with formal definitions. These terms are resistance, resistance phenotypes, and heritable resistance.

Resistance

Resistance is an active host response. In a strict sense, it is the genetically-determined ability of a plant to actively resist inoculation, infection, growth, and sporulation by a pathogen, ranging from complete (pathogen may infect specific tissues, but is walled off or dies before extensive establishment and sporulation) to partial (pathogen survives and perhaps sporulates, but established infection does not prevent host reproduction or survival). Hypersensitive resistance is a form of complete resistance.

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* Such non-specific mechanisms are passive defense responses against all pathotypes, are often controlled by multiple genes, lack definitive, readily-scoreable phenotypes, and merely limit physical damage to the host so that it can survive to reproductive age. By contrast, specific mechanisms are active defense responses by particular host genotypes that cause a hypersensitive reaction against matching pathotypes.
Resistance Phenotypes

In our research, we are looking for experimentally repeatable host/pathogen interaction phenotypes that represent potential mechanisms of resistance. These include a range of host reactions to pathogen inoculation and infection (including, but not limited to, needle spots, needle shed, twig blight, and bark lesions) that are robust, readily characterized, experimentally repeatable, and demonstrably heritable.

Heritable Resistance

Once resistance has been demonstrated, it must be amenable to practical deployment in hosts under field conditions. Whether resistance is simply-inherited (a single major gene) or complexly-inherited (multi-genic), for it to be useful in disease management it must be amenable to selection, breeding, and deployment so that future host populations comprise a measurable component of desired disease-preventing and disease-limiting traits.

Background

Since 1970, white pine blister rust resistance research has operated in the presence of a paradigm that was familiar in agriculture as the gene-for-gene hypothesis (Flor, 1956), but seldom if ever recognized in forestry. This paradigm addresses the powerful role of major gene (simply-inherited) immune resistance (MGR), which was first reported in forestry in a wild, uncultivated conifer in 1970 (Kinloch et al.). The conifer was sugar pine, the host resistance gene came to be known as Cr1, and the pathogen that was recognized by this gene, preventing infection beyond the needle, was Cronartium ribicola.

In sugar pine, the presence of MGR was inferred by whether the host, in response to inoculation and invasion by the pathogen through stomata, developed a discrete, necrotic needle spot, within which the fungus was prevented from developing further (an active hypersensitive response by the host, indicating the pine had at least one copy of the dominant Cr1 allele, designated either Cr1cr1 or Cr1Cr1 in the diploid state), or developed diffuse, yellow, chlorotic needle spots from which the fungus rapidly colonized the needle, twig, and stem (essentially a non-response by the host, indicating alleles at the Cr1 locus were in the homozygous recessive state, cr1cr1, and the susceptible tree was incapable of recognizing and reacting to fungal invasion).

Simple plant-resistance genetic systems such as this are usually complemented by a similarly simple genetic system in the pathogen. A dominant resistance gene in the host (in this case, Cr1) is reciprocally complemented by an active avirulence gene in the pathogen (Aver1). If the pathogen is inactive (vcr1) at the avirulence locus, however, it is then capable of infecting both resistant and susceptible pines (i.e., both resistant, Cr1cr1, and susceptible, cr1cr1, hosts), whereas the Aver1 pathogen is only capable of infecting susceptible cr1cr1 hosts (Table 1).

Heritable needle resistance has been documented also in western white pine (Cr2, *P. monticola*) (Kinloch et al., 1999) and in southwestern white pine (*Cr3, P. strobus*formis) (Kinloch and Dupper, 2002), and shown by experimental inoculation to segregate in a Mendelian manner (i.e. simply-inherited). The latter data were derived from control inoculations with *C. ribicola* at the Institute of Forest Genetics (IFG) greenhouse in Placerville, using seed collected from maternal parent trees (families) that were open-pollinated by local paternal sources. Blister rust inoculum in these tests was known by repeated experiments to be avirulent to Cr1 (i.e., lacking the vcr1 allele), and was designated

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1 Convention dictates that dominant alleles are denoted with a leading capital letter (i.e., Cr1 or Aver1) while recessive alleles are denoted by all lower case letters (i.e., cr1 or vcr1).

2 This discussion can be confusing, since resistance is dominant in the diploid plant host, while avirulence (in which the pathogen generates a signal that is recognized by the host, and is thereby prevented from further development in planta) is the active allele in the haploid pathogen. Alternatively, virulence (the ability of the pathogen to circumvent host defenses by avoiding recognition and thus causing disease) is inactive in this pathosystem. The term active refers to the genetic capability of the pathogen to generate a signal or product that is recognized by the Cr1 host and leads to a hypersensitive response.

3 The notation “Cr1_” indicates that, whether an individual sugar pine is heterozygous resistant (Cr1cr1) or homozygous resistant (Cr1Cr1), the phenotype of that tree against wild-type inoculum remains the same (i.e., a resistant, hypersensitive needle spot).

4 One assumption of this complementary genetic system is that the host is diploid and the pathogen, which infects pines via uninucleate basidiospores, is haploid. Thus, the pathogen is either Aver1 (avirulent against Cr1 hosts, but virulent against cr1cr1 hosts), or vcr1 (virulent against Cr1_ and cr1cr1 hosts).
El Dorado wild-type because it was and continues to be collected (and monitored for virulence) from selected sugar pine populations east of the Institute in El Dorado County.

If a maternal parent carries one copy of the Cr allele and is exposed to the El Dorado wild-type inoculum, then 50% of her seedling progeny will be resistant; if she carries two copies of the Cr gene, all her progeny will be resistant; if she has no copies of the Cr allele, then all progeny will usually prove susceptible. In some cases, low levels of resistance (1-15%) are found in families in which the maternal parent has no copies of the Cr allele; this resistance derives from local pollen donors that carry a copy of the Cr allele. Thus, one may infer from bulk-lot seedling experiments whether hypersensitive needle resistance is present within a population, which is indicative of a Cr phenotype, but cannot confirm the presence of the Cr allele without demonstrating that the phenotype is simply-inherited.

Hypersensitive needle resistance has been documented in limber pine (Pinus flexilis) (resistance gene tentatively denoted Cr4, pending confirmation), but inheritance has not been determined because all seed tested so far have been from bulk lots or from small, usually single-family collections that exhibited no resistance (Kinloch and Dupper, 2002). Lacking a family structure to seed collections, it is impossible to confirm inheritance without experimentally controlling the maternal contribution of half the alleles that are expressed in the progeny. Assuming that Cr resistance is rare in most white pine species, bulk lot inoculations can only provide an indication of the presence of the resistant phenotype and a preliminary estimate of the presence of resistance alleles across the landscape. (e.g., limber pine, Table 2). Greenhouse inoculations at IFG and at the Placerville Nursery (J. Gleason, personal communication) have been and are being carried out on family-level whitebark and foxtail pine seed collections, but no evidence of needle hypersensitivity resistance has yet been documented in these species. Tests for simply-inherited resistance mechanisms in Rocky Mountain and Great Basin bristlecone pine are underway, and will be described below.

Virulence conferred by vcr1 has been documented in the sugar pine/C. ribicola pathosystem at two sites separated by several hundred miles in California (Kinloch & Comstock, 1981; Kinloch, 1996): at Happy Camp, Siskiyou Co., a sugar pine testing site where all survivors of field inoculations there carried the Cr1 allele (otherwise, they would not have initially survived the heavy inoculum load at this location); and at Mountain Home Demonstration

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### Table 1. General scheme for gene-for-gene interactions between a diploid host and a haploid pathogen. Resistance is dominant in the host, while avirulence is active in the pathogen. For a resistant interaction to occur, the pathogen must produce an active signal that is recognized by the resistant host. In susceptible interactions, either the pathogen (designated vr) produces no signal, or else the host (rr) is incapable of recognizing signals produced by the pathogen, and thus cannot respond to invasion with a hypersensitive reaction. This table demonstrates what H.H. Flor asserted, that “… for each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite” (Flor 1956).

<table>
<thead>
<tr>
<th>Pathogen avirulence genes</th>
<th>Host resistance genes&lt;sup&gt;a&lt;/sup&gt;</th>
<th>RR&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Rr&lt;sup&gt;b&lt;/sup&gt;</th>
<th>rr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avr</td>
<td>–&lt;sup&gt;c&lt;/sup&gt;</td>
<td>–</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>vr</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Generic labels for pathogen and host genes: Avr, active avirulence; vr, inactive virulence; RR, homozygous resistant; Rr, heterozygous resistant; rr, homozygous recessive.

<sup>b</sup> Note that, since resistance (R) is dominant, the RR and Rr states in hosts yield identical interactions with the pathogen.

<sup>c</sup> Types of host/pathogen interactions: –, no disease (resistant interaction); +, disease (susceptible interaction).

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§§ This is a tenable assumption, since we know from several decades of resistance testing (both at IFG and at the nearby USDA, FS, Placerville Nursery, where the Region 5 Rust-Resistance Screening Program (RRSP) has been testing white pines for several decades) that gene frequency for Cr1 in sugar pine throughout California is approximately 0.02 overall, ranging from 0.001 in the southern Cascade Mtns., to 0.03-0.05 in the central Sierra Nevada, to 0.05-0.07 in the southern Sierra, and then dropping to 0.03-0.06 in Southern California and to 0.00 in northern Baja California (the southern-most distribution of P. lambertiana) (Kinloch 1992).
State Forest, Tulare Co., a mixed conifer-Giant Sequoia forest with large Ribes populations distributed throughout, and a moderately high rust resistance in sugar pine (Cr1 frequency ~0.08). Abundance of inoculum on Ribes, locally conducive climates, and, most prominently, moderate to high frequencies of Cr1 in sugar pine hosts have contributed to selection within the local C. ribicola population of rare fungal mutants that are vcr1.

Within a few miles of each of these sites, the frequency of vcr1 drops to zero, supporting the hypothesis that both locations, with frequencies of vcr1 approaching 1.00, are isolated occurrences that arose because of a particular set of rust-conducive circumstances that is exceedingly rare elsewhere (Kinloch et al. 2004). We continue to test this hypothesis by making annual telial Ribes leaf collections at and near both Happy Camp and Mountain Home to monitor changes in vcr1 frequency over time. Plans are also underway for regularly testing other populations of C. ribicola from selected locations in California where frequencies of Cr1 are known to be higher than in the landscape at large; these sites are associated with USDA, FS, Region 5 Genetic Resources Program monitor plantations, comprising characterized lots of both resistant and susceptible sugar pine. To date, however, no occurrences of vcr1 have been documented at any monitoring plantations that are not in the immediate vicinity of either Happy Camp or Mountain Home.

Table 2. Results of limited inoculation tests on mixed lots of limber pine (Pinus flexilis) seed collected from 4 western U.S. States. Seedlings were inoculated at the cotyledon stage with El Dorado County, CA wild-type Cronartium ribicola inoculum in dew chambers at the Institute of Forest Genetics, Placerville, CA (data from Kinloch and Dupper, 2002, and from unpublished records of recent inoculations archived at the Institute).

<table>
<thead>
<tr>
<th>Seed Source (State)</th>
<th>No. of parents</th>
<th>Seedlings inoculated</th>
<th>Inoculation results$^a$</th>
<th>Allele frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>S</td>
<td>R</td>
</tr>
<tr>
<td>Montana</td>
<td>1</td>
<td>43</td>
<td>43</td>
<td>0</td>
</tr>
<tr>
<td>Colorado</td>
<td>Bulk lots</td>
<td>185</td>
<td>152</td>
<td>18$^b$</td>
</tr>
<tr>
<td>Arizona</td>
<td>1</td>
<td>196</td>
<td>192</td>
<td>0</td>
</tr>
<tr>
<td>California</td>
<td>2</td>
<td>147</td>
<td>144</td>
<td>0</td>
</tr>
<tr>
<td>Total seedlings tested:</td>
<td></td>
<td>571</td>
<td>531</td>
<td>18</td>
</tr>
<tr>
<td>Percent of all seedlings:</td>
<td></td>
<td>93.0</td>
<td>3.2</td>
<td>3.3</td>
</tr>
</tbody>
</table>

$^a$ S, diffuse chlorotic spots on needles, with subsequent stem infection; R, discrete hypersensitive spots on needles, with no subsequent stem infection; ?, needle and stem reactions unclear; D, test seedling died before needle symptoms or stem infection could be assessed.

$^b$ Assuming that each resistant individual was heterozygous for the putative Cr4 allele, the total number of resistance alleles was assumed to be one per individual, out of a total pool of 2 x 185 seedlings, or 370 total alleles at this genetic locus.
Virulence to major gene resistance in western white pine (Cr2) has been detected at Happy Camp and at varying frequencies throughout central and southern Oregon (Kinloch et al. 2004), and has been designated vcr2. Reciprocal inoculations of vcr1 and vcr2 onto homozygous-resistant families of sugar pine (Cr1) and western white pine (Cr2) revealed that vcr1 is avirulent against Cr2 in western white pine, and vcr2 is avirulent against Cr1 in sugar pine. (Table 3). Inoculations of vcr1 and vcr2 onto resistant southwestern white pine (Cr3) also demonstrated that these pathotypes are avirulent against Cr3. As yet, no complementary pathotype (putatively denoted vcr3) is known to occur that is virulent against Cr3 (Kinloch and Dupper, 2002; Vogler, unpublished data) (Table 3). If there were such a pathotype, it would presumably be virulent only against major gene resistance in *P. strobiformis* and not against resistance alleles in sugar pine or western white pine (Cr1 and Cr2, respectively), but this hypothesis is yet to be tested.

The current state of knowledge about major resistance genes in western North American white pines is summarized in Table 4. So far, major gene resistance has been documented in four white pine species. In sugar and western white pine, operational screening protocols*** are well established and routine; for limber and southwestern white pine, protocols are being developed experimentally at IFG. For southwestern white pine, family-level inoculation tests have identified several heterozygous resistant parents; these yield 50% resistant progeny (Kinloch and Dupper, 2002; Vogler, unpublished data). For limber pine, family-level inoculation trials have not been conducted to any significant extent. For the latter two species, when possible, homozygous resistant host genotypes (progeny 100% resistant) must be identified and developed as seed sources for experimental determination of putative virulence alleles vcr3 and vcr4†††.

### Table 3. Interactions between virulence genes and resistance genes in different white pine hosts, exhibiting how virulence alleles interact specifically with a complementary host resistance allele, but not with non-complementary alleles. All non-resistant pines will, however, be susceptible to all 3 pathotypes.

<table>
<thead>
<tr>
<th>Host resistance genes</th>
<th>Pathogen virulence genes&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>vcr1</td>
</tr>
<tr>
<td>Sugar pine (Cr1&lt;sub&gt;−&lt;/sub&gt;)</td>
<td>+</td>
</tr>
<tr>
<td>Western white pine (Cr2&lt;sub&gt;−&lt;/sub&gt;)</td>
<td>−</td>
</tr>
<tr>
<td>Southwestern white pine (Cr3&lt;sub&gt;−&lt;/sub&gt;)</td>
<td>−</td>
</tr>
</tbody>
</table>

<sup>a</sup> +, susceptible interaction (disease); −, resistant interaction (no disease).

<sup>b</sup> Pathogen alleles and interactions in brackets are hypothetical, since they have not been fully documented experimentally.

Until such time as these homozygous-resistant seed sources become available, accurate detection and delineation of *C. ribicola* pathotypes that are virulent against Cr3 and Cr4 will be difficult, though not impossible‡‡‡.

**Inoculation tests of high-elevation white pines**

Thus far, all inoculation tests that have demonstrated major gene, hypersensitive resistance to white pine blister rust have been with white pines within Section *Strobus*, Subsection *Strobi* (Table 5). According to the phylogeny developed by Price et al. (1998), the high-elevation pines that have exhibited no evidence of major genes for resistance, or have yet to be tested, are either within Section *Strobus*, Subsection *Cembrae* (*P. albicaulis*) or in Section *Parrya*, ‡‡‡.

**<sup>***</sup> Beginning in the early 1970’s, these protocols were developed experimentally by B.B. Kinloch, Jr. and colleagues at IFG, and later transferred to the USDA, FS, Region 5 Rust Resistance Screening Program at the Placerville Nursery in Camino, CA, where they were developed further and refined for operational use by S. Samman, P. Zambino, J. Gleason, J. Dunlap, and others.

**†††** Heterozygous resistant seed sources are not ideal for this purpose, since 50% of their progeny become infected with Aver inoculum, and thus it is difficult to determine whether infection was initiated by wild-type or by virulent inoculum. With homozygous resistant seedlings, we count and assess the phenotype of each and every needle spot, allowing us to detect those initially rare susceptible-interaction phenotypes that indicate virulence in the pathogen.

**‡‡‡** With heterozygous resistant seed sources, individual seedlings with both resistant and susceptible needle spots indicate that test inoculum is a mixture of Aver and vcr basidiospores, from which one may infer that virulence has arisen within the local *C. ribicola* population.
Subsection \textit{Balfourianae} (\textit{P. balfouriana}, \textit{P. aristata}, and \textit{P. longaeva}). A more recent molecular phylogenetic analysis (Gernandt et al. 2005), though supporting placement of all of the above except whitebark in Subsections \textit{Strobi} and \textit{Balfourianae}, nevertheless groups whitebark pine closely with pines in Subsection \textit{Strobi}.

Inoculations performed to date with \textit{P. albicaulis} and \textit{P. balfouriana} have revealed no evidence of MGR, but they have been too few to be conclusive. The potential close affinity between whitebark pine and sugar and western white pine revealed by Gernandt et al. (2005) suggests that whitebark, if family-level seed collections were surveyed from throughout its extensive range, might be a promising candidate for MGR. To date, our whitebark seed collections have been limited, nevertheless we will continue to test \textit{P. albicaulis} for MGR as seed become available from cooperators.

For this discussion we will focus on the three high-elevation pine species in Subsection \textit{Balfourianae}. As illustrated in Table 4, limited inoculation tests have been done with foxtail pine, but hypersensitive needle spots indicative of MGR were not observed (Delfino-Mix, unpublished data, J. Gleason, personal communication), leading to speculation that simply-inherited resistance mechanisms may not be found in this species. Foxtail pine is confined to two disjunct locations in California: high-elevation wilderness stands in the Klamath Mountains in the north of the State, and remote portions of Sequoia and Kings Canyon National Parks in the southern Sierra Nevada. We have not yet collected an extensive sampling of foxtail pine seed for testing. We have, however, collected a sizeable family-level seed collection of Rocky Mountain bristlecone pine from throughout Colorado and a smaller collection of Great Basin bristlecone pine from the White Mountains of California. Our current inoculation efforts have therefore focused on the latter two species.

An early trial with \textit{P. longaeva} had shown that seedlings inoculated with blister rust within 3-6 months post-germination became infected and died rapidly, well before needle spots or stem symptoms could be scored with assurance. We therefore modified our standard protocols (which had been to inoculate seedlings in the cotyledon stage, when only primary needles had developed) so as to inoculate bristlecone in the second year after sowing. This necessitated over-wintering seedlings in the lath house, and then returning them to the greenhouse the following spring, which dramatically improved their hardiness prior to inoculation. Consequently, at inoculation, cotyledons were either moribund or shed, and seedlings comprised mostly primary needles with secondary needles just beginning to expand.

### Table 4. Summary of experimentally-determined major resistance genes in western U.S. white pines, and corresponding virulence genes in \textit{Cronartium ribicola}.

<table>
<thead>
<tr>
<th>White pine hosts</th>
<th>WPBR-related genes(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common name</td>
<td>Scientific name</td>
</tr>
<tr>
<td>Sugar pine</td>
<td>\textit{Pinus lambertiana}</td>
</tr>
<tr>
<td>Western white pine</td>
<td>\textit{P. monticola}</td>
</tr>
<tr>
<td>SW white pine</td>
<td>\textit{P. strobiformis}</td>
</tr>
<tr>
<td>Limber pine</td>
<td>\textit{P. flexilis}</td>
</tr>
<tr>
<td>Whitebark pine</td>
<td>\textit{P. albicaulis}</td>
</tr>
<tr>
<td>Foxtail pine</td>
<td>\textit{P. balfouriana}</td>
</tr>
<tr>
<td>Rocky Mtn. bristlecone</td>
<td>\textit{P. aristata}</td>
</tr>
<tr>
<td>Great Basin bristlecone</td>
<td>\textit{P. longaeva}</td>
</tr>
</tbody>
</table>

\(^a\) Y, major gene resistance in the host or virulence in the pathogen have been documented in this species; N, resistance or virulence have not been documented by greenhouse tests; (), tests for major gene resistance have been conducted, but results are still too limited for definitive conclusions; ?, no controlled inoculation tests have been done with these species; P, inoculation tests are underway, but results are still pending.

\(^{111}\) \textit{P. albicaulis} is intermediate between the latter two taxa in the authors’ strict consensus of 55,536 trees based on \textit{rbcL} and \textit{matK} sequence data (Fig. 2, Taxon 54: 33-34).
For inoculation of *Cronartium ribicola* onto *Pinus aristata* and *P. longaeva*, we used El Dorado wild-type inoculum amplified via urediniospores on leaves of multiple ramets from a single *Ribes nigrum* clone****; because of their large size and tolerance of rust infection, leaves of this clone are ideal for controlled inoculations. Formation of telia was induced by cultivating inoculated ramets in the greenhouse with temperatures not exceeding 23°C in daytime and 18°C at night, and relative humidity in the range of 50-70%.**** Just prior to pine inoculation, telial *R. nigrum* leaves were harvested and soaked in sterile distilled water for 1 hour, and then placed telia-down on 64 cm x 58 cm wire-mesh racks with 5 mm-square openings. *Ribes* leaves were covered with moistened cheesecloth and racks were placed in the dew chamber†††† so that exposed telia were 5-10 cm above the tops of the test seedlings. Two 98-well supercell racks of pine seedlings were placed on the shelf directly below the ripe telia. *Ribes* leaves and pine seedlings were inoculated in the dark at 15°C and 100% relative humidity for 72 hours. At the end of inoculation, chambers were switched off, chamber doors opened, racks of telial leaves removed, autoclaved, and discarded, and racks of seedlings left in place for 4-6 hours until equilibrated with greenhouse temperature and humidity. Seedling racks were then placed on greenhouse benches for *C. ribicola* incubation, needle spot development, and scoring of phenotypes. For long-term observations and analysis, seedlings are over-wintered in the lath house.

Results of inoculations are shown in Tables 6 and 7 for *Pinus aristata* and *P. longaeva*, respectively. The former were rated for needle spots, symptoms of infection, and signs of fungal development three times, the last at one year post-inoculation, and thus provide the more complete picture of potential resistance mechanisms for these two species. The latter (*P. longaeva*) has been rated once at 4 months post-inoculation; one-year evaluations will be conducted in June 2006. The 1355 *P. aristata* seedlings inoculated in May 2004 represent 108 open-pollinated families from throughout Colorado. Number of seedlings per family ranged widely, from 1 to 55, based on quality of seed and percent germination. Overall, two-thirds of the trees that survived one year after inoculation became infected. Percent trees infected per family ranged from 0 to 100%.

### Table 5. Phylogeny of western North American white pines within the genus *Pinus* (Price et al., 1998).

<table>
<thead>
<tr>
<th>Genus <em>Pinus</em></th>
<th>Subgenus <em>Pinus</em> (hard pines)</th>
<th>Subgenus <em>Strobus</em> (soft pines)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Section <em>Strobus</em></td>
<td>Subsection <em>Strobi</em></td>
<td>Subsection <em>Cembrae</em></td>
</tr>
<tr>
<td><em>Pinus monticola</em>, <em>P. lambertiana</em>, <em>P. flexilis</em>, <em>P. strobiiformis</em></td>
<td><em>P. albicaulis</em></td>
<td></td>
</tr>
<tr>
<td>Section <em>Parrya</em></td>
<td>Subsection <em>Balfourianae</em></td>
<td></td>
</tr>
<tr>
<td><em>P. aristata</em>, <em>P. longaeva</em>, <em>P. balfouriana</em></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 6. Infection results for Rocky Mountain bristlecone pine (*Pinus aristata*) 12 months post-inoculation. Seed were sown in spring 2002, inoculated with *Cronartium ribicola* (El Dorado wild-type) in May 2004, and scored for stem symptoms and signs in May 2005. Trees noted as dead died for reasons unrelated to rust infection.

<table>
<thead>
<tr>
<th>Stem symptoms &amp; signs</th>
<th>No. of trees</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>None(^b)</td>
<td>294</td>
<td>22%</td>
</tr>
<tr>
<td>Discolored or swollen(^c)</td>
<td>152</td>
<td>11%</td>
</tr>
<tr>
<td>Discolored &amp; swollen, spermatial, or aecial(^d)</td>
<td>883</td>
<td>65%</td>
</tr>
<tr>
<td>Dead (non-rust)</td>
<td>26</td>
<td>2%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>1355</td>
<td>100%</td>
</tr>
</tbody>
</table>

\(^a\) Symptoms are evidence of host response to infection (i.e., stem discoloration, swelling, or both); signs are evidence of the pathogen (i.e., spermatia (pycnia) or aeciospores).

\(^b\) Within this category, 132 seedlings exhibited no needle spots attributable to infection; the remainder of seedlings in this category (162) developed needle spots.

\(^c\) Symptoms suggestive of successful infection, but remaining unresolved.

\(^d\) Definitive symptoms and signs of infection.
100%, with some families appearing to segregate 50:50 for susceptibility and non-susceptibility. With the latter families, however, replicate inoculations will be required to determine whether these ratios are statistically robust and therefore indicative of Mendelian segregation.

Needle spot characteristics did not correlate with whether or not an individual seedling ultimately became infected. Spots on primary and secondary needles ranged in color from yellow to brown to orange to red, and ranged in morphology from discrete to diffuse and spreading. However, there was no evidence of a correlation between any particular spot color or morphology and subsequent success or failure of *C. ribicola* to colonize the stem from needles. Thus, there was no evidence in this species for classic needle hypersensitivity (Kinloch and Littlefield, 1977), as evidenced with MGR in sugar, western white, and southwestern white pine, and suspected in limber pine. Although evaluations of *P. longaeva* inoculations are still incomplete, preliminary observations of the 23 families tested confirm that needle spot phenotypes in this species likewise do not correlate with subsequent infection or non-infection by blister rust.

One may be tempted to conclude from these results that simply-inherited resistance does not occur in either *P. aristata* or *P. longaeva*, but that would be confusing the mechanism of resistance with the mode of inheritance. Apparent absence of needle hypersensitivity as a mechanism, or phenotype, of resistance in these species does not preclude the possibility that other simply-inherited resistance mechanisms nevertheless may yet be found in *P. aristata* or in *P. longaeva*; the mechanisms and timing of resistance are not yet obvious. As noted earlier for *P. aristata*, there is suggestive, though very preliminary, evidence for simple segregation within families for resistance and susceptibility. Early results from *P. longaeva* show a similar trend. At this early stage of research, it seems possible that both species will exhibit a range of inherited resistance to *C. ribicola*, but it is not yet possible to state with certainty either the mode of inheritance or the mechanism by which it operates.

One caveat in interpreting early inoculation results is that failure of infection to develop is not evidence that resistance is occurring. Often, the simplest explanation for apparent resistance is that the fungus failed to inoculate and infect the host (a phenomenon known as “escape”). Although we closely monitor the amount and distribution of inoculum during dew chamber inoculations, occasionally individuals or small groups of seedlings are not directly challenged by basidiospores. Thus, when assessing the status of seedlings that did not become infected, we record whether or not they first developed needle spots consistent with invasion and establishment by *C. ribicola*. As shown in Table 6 for *P. aristata*, some 45% (132/294) of the seedlings that did not become infected did not exhibit pathogen-associated needle spots prior to evaluation at one year post-inoculation. To confirm whether these seedlings are actually resistant and not merely escapes, we have re-inoculated this set, and are currently evaluating them for needle spots and subsequent symptom development.

As to the remainder of the seedlings that did not develop symptoms or signs but did exhibit needle spots (Table 6: 162 *P. aristata*; Table 7: 140 *P. longaeva*), we continue to monitor them closely for
any and all phenotypes that may represent a hypersensitive or walling-off response elsewhere in the plant than in the needles, which theoretically may prevent establishment and subsequent sporulation of the rust fungus. Among the 162 asymptomatic \textit{P. aristata}, some 20\% exhibited pitchy lesions on the stem at or near the base of infected needles; these were often associated either with necrotic, occasionally purple, bark patches, or with bumpy, constricted swellings. Such lesions may be evidence of a hypersensitive reaction at the needle base, similar to the bark reaction that has been reported in sugar pine (Kinloch and Davis, 1996). At this time, and until repeat inoculations have been conducted with \textit{P. aristata} and \textit{P. longaeva} families selected from these initial tests, the inheritance, durability, and robustness of these putative resistance mechanisms remain to be determined.

In summary, preliminary results support several conclusions: 1) some individual seedlings (and possibly some families) of \textit{Pinus aristata} and \textit{P. longaeva} exhibit reduced susceptibility to \textit{Cronartium ribicola}, possibly resistance; 2) classic, simply-inherited hypersensitive needle-spotting does not appear to be a mechanism of resistance in these white pine species; 3) more detailed observations and re-inoculations of seedlings that did not express needle spots are needed to substantiate the possibility that these individuals actively resisted fungal invasion and did not merely escape infection; 4) replicate inoculations of putatively resistant families are needed to substantiate hypothesized mechanisms of resistance and modes of inheritance; and 5) experience gained from the bristlecone pines may benefit resistance screening efforts with foxtail pine, and until repeat inoculations have been conducted in Colorado: Potential consequences of their loss. Pp 124-135 in \textit{Sniezko, R.A., Samman, S., Schlarbaum, S.E. and Kriebel, H.B.}, eds., Breeding and genetic resources of five-needle pines: growth, adaptability, and pest resistance, Proceedings of IUFRO Working Party 2.02.15, 23-27 July 2001, Medford, OR, USA. U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, Fort Collins, CO. Report No. RMRS-P-32.

**Acknowledgements**

We thank John Louth, USDA, FS, Inyo National Forest, Bishop, CA, for facilitating our collections of \textit{Pinus longaeva} cones in the White Mountains, CA; Roger Stutts and David Johnson, USDA, FS, PSW Research Station, Institute of Forest Genetics, Placerville, CA, for their training and assistance in making cone collections in the White Mountains; and Luke Nemeth for field assistance with our collections of \textit{Pinus aristata} in Colorado.

**Literature Cited**


Modeling the Potential Distribution of White Pine Blister Rust in the Central Rocky Mountains

Holly S. J. Kearns and William R. Jacobi

Introduction

*Cronartium ribicola* (J. C. Fischer ex Rabh.), the causal agent of white pine blister rust (WPBR), was introduced to western North America via infected nursery stock imported from France to Point Grey near Vancouver, British Columbia (Mielke 1943). Primary infection of white pines occurs on the needles where fungal spores land, enter through stomata, grow within the vascular tissue, and then enter branches and stems. The fungus grows in the intercellular spaces within the bark of white pines where the production of spores breaks apart the bark causing the girdling of branches and stems; the ultimate result of which is tree death (Tainter and Baker 1996). White pine blister rust through its disruption of vascular tissues and bark, affects white pines by reducing their growth and reproductive potential, eventually killing the pine host and, in turn, affecting community structure and composition by removing the host from the community (Kendall and Arno 1990).

White pine blister rust has been present in northwestern Wyoming since the 1940s and in central Wyoming since the 1970s (Brown 1978), but has only recently been observed in Colorado. The disease was first reported in Colorado in 1998 with the area of highest infection located within approximately 18 km of the Wyoming border (Johnson and Jacobi 2000). In 2003, isolated WPBR infestations were discovered in the Wet and Sangre de Cristo Mountains of southern Colorado, more than 300 miles away from other known infections. Limber pines (*Pinus flexilis*) are infected in these new areas, but the first natural infections on Rocky Mountain bristlecone pine (*P. aristata*) were also discovered (Blodgett and Sullivan 2004), heightening concern about rust spreading throughout populations of limber, southwestern white (*P. strobiformis*), and bristlecone pines in the southern and central Rocky Mountains.

Research Objectives

The objectives of this project were to examine the current status of WPBR and to develop models that predict the likelihood of disease occurrence in and damage to native white pine populations in the central Rocky Mountains. White pine blister rust epidemiology is strongly affected by genetics, profusion of inoculum, nearness and distribution of hosts, and microclimate (Geils et al. 1999). In order to develop these predictive models, we attempted to characterize interactions between white pine and Ribes hosts, the pathogen, and environmental conditions, and how these interactions change through time.

Methods

To achieve these objectives we performed a survey of limber pine throughout central and southeastern Wyoming and northern Colorado. At each of 504 established survey plots, a series of transect in which the condition of limber pine and the occurrence and density of Ribes by species were recorded. In addition, an extensive Ribes survey consisting of 758 plots was conducted to determine the distributions, densities, and associated site factors of Ribes by species growing in the vicinity of white pines. Live cankers were collected and analyzed using dendrochronological techniques to examine the relationship between total canker length and time (i.e. canker growth rate).
Results & Discussion

WPBR was present on 55% of the 504 survey plots. Incidence, the proportion of infected trees, ranged from 0 to 100% and averaged 15.5% over all plots and 28.0% on infested plots. Diameter class and crown class were significantly related to likelihood of infection by WPBR. Incidence varied significantly by elevation and slope position and did not vary by aspect, limber pine density, slope configuration, or degree of canopy closure. Incidence of WPBR is currently low along the southern boundary of its distribution in southeastern Wyoming and northern Colorado. The current level of infestation has been attained within the past two to four decades, and with time the pathogen may spread to currently uninfested white pine populations and intensify throughout its current distribution. Monitoring of permanent plots and large-scale resurveys will be necessary to evaluate spread of the pathogen to currently uninfested areas and to determine the impacts of the disease over time.

Species of Ribes were found growing in the vicinity of white pines in all study areas surveyed, but the species present and the density of each species varied between the study sites. The most commonly encountered species were *R. cereum*, *R. inerme*, *R. lacustre*, and *R. montigenum*. Densities and probabilities of occurrence were related to site variables and varied by Ribes species. *Ribes cereum* had higher densities in dry, open areas than in moist, densely forested areas. *Ribes inerme* had highest densities on lower elevation, riparian and wetlands areas with components of aspen and willow. *Ribes lacustre* was associated with riparian areas and closed canopy forests with components of Engelmann spruce, subalpine fir, and alder. *Ribes montigenum* had highest densities in high elevation, open stands of Engelmann spruce.

An analysis of canker growth rates was performed on 134 WPBR cankers harvested from limber pine. There was a strong relationship between total canker length and time. Total canker length was related to number of years dead at canker center and proximal branch/stem diameter. Longitudinal canker growth rate varied by branch diameter, branch height, and condition of the branch distal to the canker, but did not vary by study area.

The data collected in the field surveys was used to develop a series of regression and categorical and regression tree analysis models that predict risk and hazard of WPBR in Colorado’s native white pine populations. Risk models predicting the presence of WPBR employed meteorological, Ribes, and tree size data and resulted in good agreement between predicted and actual presence. Forest land managers can utilize the developed risk maps in concert with the models to prioritize monitoring, management, and, if necessary, control efforts. By monitoring areas with the greatest likelihood of pathogen establishment, control measures can be applied where impacts of WPBR create conditions outside the desired range. When the pathogen becomes established in an area, the infestations can be followed through time, which should allow for better calibration of disease pressure and hazard models to specific conditions found in Colorado.

This research was funded by the USDA Forest Service Special Technologies Development Program, USDA Forest Service Rocky Mountain Research Station, and the Colorado State Agricultural Experiment Station.

References


Aspen Panel: Moderator Mary Lou Fairweather

Browse History of Aspen on the Northern Ungulate Winter Range of Yellowstone National Park: A New Look at an Old Debate

Roy Renkin

Synopsis

It is well recognized that aspen has been declining over the northern ungulate winter range of Yellowstone National Park throughout the past century. Understory recruitment has not occurred, except in a few isolated circumstances, since the early 1920’s. Various authors have addressed this decline using interpretations derived from comparative historic photographs or the behavior of aspen in exclosures protected from ungulate browsing, primarily elk. These interpretations have also been used as proxies for elk densities, with some authors arguing that few to no elk were present just prior to Euroamerican settlement in the 1870’s because the photographic evidence suggests little to no elk use of aspen. In a recent synthesis of the available literature on the subject, the NAS (National Academy of Science) stated that ungulates are a major factor contributing to the current absence of aspen recruitment. What is not known, they concluded, is how elk numbers and patterns of habitat use before park establishment might have influenced aspen dynamics. Such information, they reasoned, would be extremely valuable in understanding the dynamic interaction between elk and aspen in relation to the current situation. Historic elk numbers will probably remain speculative and relative to current levels, given the lack of census data and the well-recognized era of market hunting coincident with the settlement period. Historic habitat use expressed as the utilization of woody vegetation, on the other hand, might be inferred using innovative techniques that overcome the limitations of photo interpretation.

We used a technique that relied on exposing the inner pith architecture of fallen aspen trees to interpret whether the terminal or primary stem of the tree had previously been browsed while in the seedling/sapling stage. We specifically looked for the presence of a stub remaining from the original primary stem, and new primary stem emergence from a lateral bud, as evidence of previous browsing. We differentiated browse from branch architecture based on the more acute pith angle typical of branching. Where appropriate, we also collected paired cross sections from the bole of the tree at 0.2m and 1.5m above ground for age estimation to determine whether browse frequency had a measurable influence on tree growth rate. With a subset of recently-fallen trees of a known death year, we could further section through an identified browse occurrence to estimate year of browse. To support our interpretations, we additionally sampled the architecture of: a) transplanted aspen saplings that were protected and subsequently manually clipped to induce an architecture similar to ungulate browsing; b) dead aspen trees that grew within ungulate exclosures and theoretically were protected from browsing; c) aspen saplings currently growing within ungulate exclosures; and d) fallen aspen trees sectioned from 2.0m to 3.5m above ground and therefore beyond the browse zone. Because of inherent limitations with our technique, we could not directly address qualitative levels of browse intensity. If overwinter elk populations and subsequent utilization of aspen were indeed low to non-existent prior to and during the settlement period of the 1870’s, we would expect to find little to no elk use of aspen and no influence on aspen growth during this era.

We obtained browse architecture from 251 dead and downed aspen trees from 113 randomly established aspen monitoring sites and from another 28 recently-fallen but foliated aspen trees that we encountered while traveling to established sites. From these 279


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trees, we tallied 492 browse occurrences, for an average of 1.8 per tree. Fourteen percent (n=40) of our sample trees showed no evidence of previous browsing, while the remaining 86% (n=239) showed from 1 to 7 browse occurrences on the primary stem. We recorded 3 or more browse occurrences in 26% (n=72) of our sample trees. One hundred fifty two of 279 trees were of a condition that allowed ring counts and age estimation from paired cross sections. Overall, it took an average of 6.8 +/- 3.6 years for our trees to grow from 0.2m to 1.5m (breast height) above ground, with a range from 1 to 17 years. The distribution is not normal, but rather bimodal, with one large peak from 4-7 years and another smaller peak from 9-12 years. Eighty-four percent (n=127) of the trees grew to 1.5m in 10 years or less, while the remaining 16% (n=25) took more than 10 years. There was a non-linear relationship between browse frequency and time (years) required to grow to breast height that was due to the inherent variability in growth rate among aspen clones. The population regression was not linear (F = 3.7, p>0.05) and browse frequency explained very little of the variability in growth rate, (r = 0.27), but the slope of the regression line was positive and not equal to zero (F=11.92, p=0.0007). Median and 50th percentile measures of central tendency for time to grow to breast height increase as browse frequency increases, suggesting that the more frequently our aspen trees were browsed as seedlings/saplings, the longer it generally took to grow to breast height. From our subsample of 28 recently-fallen trees with 56 browse occurrences, we were able to determine that the majority of our trees originated between 1860-1920 and were subsequently browsed between 1865-1925.

To lend support to our interpretations, we were able to identify architecture similar to browsing in 5 different protected aspen transplants whose terminal stem and lateral branches were clipped annually until the sucker grew out of reach. We were also able to demonstrate the lack of browse architecture in 22 of 26 dead aspen trees protected in ungulate exclosures for more than 70 years, and attribute mechanical damage during or browsing prior to exclosure construction for the remaining 4 trees. We further demonstrated that browse architecture is almost nonexistent beyond the browse zone from 2.0m to 3.5m above ground, as evidenced by 1 of 38 aspen trees with dieback/mechanical damage along this bole section that could be confused with browse architecture. In 203 protected saplings among 5 different aspen clones within 3 ungulate exclosures, we found that 22% (n=45, range of 10% to 30% per clone) of the aspen had pathogen-induced dieback or mechanical damage that might be confused with browse architecture under continued tree growth. Such “false positives” represent a potential bias in quantifying historic browse frequency and must be recognized in subsequent interpretations.

In conclusion, and considering the potential for inherent bias:

1) The current aspen overstory was achieved in spite of ungulate browsing, not because of a lack of browsing. This browsing occurred prior to and during the settlement period of 1870-1890.

2) Conservative estimates of historic browse frequency had a measurable negative effect on aspen growth rate that was mitigated by the clonal variation in such growth rates.

3) We were not able to directly address qualitative levels or changes in browse intensity, but repeated browse frequencies and the prolonged time to achieve breast height for some trees suggests the full spectrum of browse intensities were encountered.
Impact of the 2001-2003 Drought on Productivity and Health of Western Canadian Aspen Forests


Abstract—Forests dominated by trembling (quaking) aspen are a major component of the landscape in western Canada. A severe drought from 2001 to 2003 has led to a pronounced decrease in the growth of the region's aspen forests, and massive dieback of aspen and other trees has been noted in the most strongly drought-affected areas. Annual forest health assessments from a regional network of aspen plots (CIPHA study) show a major increase in wood-boring insects, along with modest increases in some fungal pathogens, during the period of drought. Despite the return to moist conditions in 2004, aspen mortality continued to increase during 2004-2005, suggesting that the full impact of the drought has not yet been fully realized. The severity of this drought and its impact raises concerns for the future productivity and health of aspen forests in the region, particularly under the drier conditions projected over the next few decades under climate change.

Background

Trembling aspen (Populus tremuloides Michx.), also known as quaking aspen, is the most abundant deciduous tree species in the North American boreal forest, and is also the main native tree in farm woodlots along the northern edge of the Canadian prairies. Aspen is important ecologically (e.g., for wildlife habitat, biodiversity and recreation), and over the past two decades, it has become an important commercial species for the forest industry (Peterson and Peterson 1992).

Since the early 1990s, aspen dieback has been noted across parts of the Canadian provinces of Alberta and Saskatchewan. This has raised concerns from both the forest industry and the general public, as to what caused the dieback, and whether climate change might be playing a role. Previous studies (Hogg and Schwarz 1999, Hogg and others 2002a, Brandt and others 2003) pointed to the following as major factors causing dieback and reduced growth of aspen in western Canada: insect defoliation, drought, thaw-freeze events, and fungal pathogens. These previous studies relied primarily on tree-ring analyses, in combination with comparisons of assessments of damage agents such as insects and diseases in healthy versus declining aspen stands. Unlike the situation of aspen dieback in the western U.S. (DeByle 1985, Hart and Hart 2001), ungulates such as elk are not a major damage agent of aspen in the Canadian boreal forest.

There is a common misconception that aspen is an unsuitable species for tree-ring analysis, but we have found that tree-rings are readily visible if cores or disks are carefully polished (Hogg and Schwarz 1999). Furthermore, aspen forms distinctive “white rings” during years of severe defoliation by forest tent caterpillar (Malacosoma disstria) and other agents (Hogg and others 2002b), thus providing a useful diagnostic tool for determining when growth reductions are associated with defoliation versus other factors such as drought.

One of the difficulties in determining the cause of aspen dieback is that it is often caused by several factors, acting sequentially or in combination. The model of Manion (1991) provides a useful framework on how these factors may operate over time to induce dieback (elevated mortality of branches within trees) and decline (partial or total loss of forest cover that often includes an impairment of tree regeneration). The Manion model was applied and extended in a recent review of landscape-level aspen dieback by Frey and others (2004). A simplified version of the
The conceptual model of Frey and others (2004) is shown in Figure 1. In this model, it is postulated that the main ultimate causes of aspen dieback and mortality are a) gradual exhaustion of stored carbohydrates that are necessary for spring leafing and other ecophysiological functions of the tree, and b) a catastrophic loss of the ability of the tree to conduct water from root to leaf, e.g., through xylem cavitation (Frey and others 2004). Drought is postulated to be especially important, because it can lead to either or both of these impacts (Figure 1).

![Figure 1. Diagram showing how drought, in combination with other factors, may cause dieback and decline of aspen (simplified from Frey and others 2004).]

Although drought has long been a concern for agriculture in the Prairie Provinces of western Canada, it has only recently emerged as a major issue for the forest sector in this region. In 1998, a record-warm year both regionally and globally, severe drought affected parts of the western Canadian boreal forest. This event, coupled with earlier concerns, motivated the establishment of a large-scale research and monitoring study in 1999-2000, entitled CIPHA (Climate Impacts on Productivity and Health of Aspen). The objectives of CIPHA are a) to conduct a regional-scale tree-ring analysis to determine the factors affecting aspen growth during 1951-2000; b) to provide early detection of large-scale changes in aspen productivity, health and mortality, especially those that may be caused by climate change; and c) to provide a data base suitable for the development and validation of climate-driven models of aspen growth and carbon sequestration.

Since CIPHA was established, the 1998 drought has persisted in some areas, and became exceptionally severe and widespread during the years 2001-2003. This paper provides an overview of the impacts on western Canadian aspen forests that have been recorded to date, from research and monitoring under the CIPHA study during 2000-2005.

**Methods**

The CIPHA study was initiated in 1999-2000 by Ted Hogg and James Brandt of the Canadian Forest Service (CFS) of Natural Resources Canada, and Bob Kochtubajda of Environment Canada. Since 2000, CIPHA has included annual monitoring in 144 permanent plots in 24 study areas that extend across western Canada from the southwestern Northwest Territories, across Alberta and Saskatchewan, to southwestern Manitoba (Figure 2). More recently, 6 additional CIPHA study areas have been established to extend the monitoring network to include the Alberta foothills, eastern Manitoba and Ontario, in collaboration with the CFS Great Lakes Forestry Centre and the Ontario Ministry of Natural Resources (results not included in this paper). All plots are located within 40- to 85-year old pure aspen stands that are relatively free from human-induced disturbances such as seismic lines, cattle grazing, emission sources and herbicide applications. Half of the study areas were located in the boreal forest, and the other half are in the climatically drier aspen parkland zone. One of the study areas was co-located with the BERMS Old Aspen flux tower site (location shown in Figure 2), where detailed monitoring of forest-atmosphere exchanges has been conducted continuously since 1996 (Barr and others 2004).

Within each plot, all trees were numbered and measured (including height and DBH, i.e., stem diameter at 1.3-m height) in 2000 and again in 2004. Each plot was 10 m wide and of sufficient length to include at least 25 living aspen of >7 cm DBH.
Figure 2. Location of CIPHA study areas in western Canada. The drought-affected area covered by aerial surveys of aspen dieback and mortality in August 2004 (Michaelian and others unpublished) is delineated by a dashed line.

Forest health assessments were conducted on each tree annually from 2000 to 2005, including crown dieback and vigor, mortality, and the incidence of agents causing stem damage, defoliation and mortality. Other measurements have included leaf litter fall, soil analyses and aspen regeneration within the plots.

In the fall of 2000 and again in fall 2004, three aspen stems were sampled for tree-ring analysis outside each plot (total of 432 trees). In the 2000 sampling, all trees were sampled by felling and disks were collected from the base, 1.3-m height and at 1/3 and 2/3 of total stem height. The 2004 sampling was mainly conducted using increment cores at 1.3-m height. Disks or cores were dried and polished, and tree-ring analyses were conducted according to the methods of Hogg and Schwarz (1999). The presence of white tree-rings, denoting years with severe insect defoliation (Hogg and others 2002b) was also noted. The tree-ring sampling design enabled the estimation of stand-level increments in basal area from ring width measurements (Hogg and others 2005). Daily climate histories for the period since 1930 (maximum and minimum temperature, precipitation) were developed from climate stations adjacent to each study area, and were used to calculate climatic indices of growing season warmth (growing degree days) and drought (climate moisture index, CMI, see Hogg and others 2005). In addition to the plot-based measurements, aerial surveys of aspen dieback and mortality were conducted by Mike Michaelian in August 2004, over a >100,000 km² area of western Saskatchewan and eastern Alberta (shown in Figure 2) that was most strongly affected by the severe drought of 2001-2003.

Summary of Results to Date and Discussion

Results from the CIPHA plot measurements and tree-ring sampling in 2000 have been presented by Hogg and others (2005). One of the striking results from the stand mensuration was that the aspen in the dry parkland zone were stunted, with a mean height of 14.8 m, compared to a mean height of 19.0 m for the aspen in the cooler and moister boreal forest. The aspen in the CIPHA plots of these two vegetation zones were comparable, having the same mean age (60 years, range of 41-81 years) and DBH (16 cm). Because of lower stand densities, stand basal areas were also smaller in the parkland, and average estimated aboveground biomass was 37% less than in the boreal forest (Hogg and others 2005). This suggested that the warm, dry conditions of the parkland have been unfavorable for aspen growth over the long term, relative to those in the boreal forest, although factors other than moisture (e.g., soil conditions and wind) might also explain this difference. The tree-ring analysis of samples collected in 2000 showed that there have been major, regional-scale oscillations in aspen growth since 1951. Regression modeling of detrended growth in stand basal area showed that drought and insect defoliation were the most important factors causing reduced aspen growth. The most striking event was from the late 1970s to the early 1980s, when a period of repeated defoliation and drought led to a 50% collapse in regional aspen growth (Hogg and others 2005). The most recent peak in aspen growth was in 1997, a cool, moist year across the region. Drought affected parts of the region in 1998, and the drought intensified during 2001-2003, when conditions were the driest in over 110 years across parts of northern Alberta, and in the parkland from central Alberta to central Saskatchewan. By June 2004, the drought had ended in most areas with the return of heavy rainfall, and the summer of 2005 was generally cool and wet.

Preliminary results from the reanalysis of tree-rings in 2004 have shown that regional aspen growth has decreased by 31% since the peak growth year of 1997. Although a formal analysis has not yet been conducted, there are indications that the greatest growth reductions have occurred within the most severely drought-affected areas. Preliminary analysis of the annual forest health assessments have also
shown a dramatic, regional-scale increase in the incidence of wood-boring insects such as *Saperda calcarata* from 5% in 2000 to 20% in 2004. In contrast, the incidence of fungal pathogens showed relatively modest increases during this period, e.g., *Peniophora polygonia* increased from 11% to 16% and *Phellinus tremulae* increased from 10% to 12%. Insect defoliation remained at minimal levels except for localized outbreaks in northern and western Alberta.

Within the CIPHA plots, regional aspen mortality was 2.3% per year during 2000-2002, and increased to 3.5% in 2003-2004. An unexpected result, however, is that our preliminary 2005 assessments have shown a continued increase in mortality to about 5% during 2004-2005, a moist period following the end of the 2001-2003 drought. This raises concerns that the drought through its impacts on stand vigor (Figure 1), may be leading to longer-term impacts on the growth, health and dieback of aspen in this region. Indeed, within the CIPHA plots in the most severely drought-affected areas, the cumulative, 5-year mortality rate was more than 20%, and massive aspen mortality approaching 100% was observed in some areas starting in the spring of 2003, especially in the smaller patches of aspen in the parkland, i.e., areas not included in the plot-based monitoring. Other trees in this area were also severely affected, including planted birch and ash in urban areas such as Edmonton, and widespread, drought-induced mortality of white spruce (*Picea glauca*) and other conifers was also noted (Ted Hogg, Pers. Obs.).

With plot-based networks it is difficult to capture the patchiness of aspen in the parkland, in combination with the patchy nature of mortality that was observed in many areas. Previous studies (Kneeshaw and Bergeron 1998, Hogg and Schwarz 1999) suggested that the clonal nature of aspen also contributes to high variation and patchiness of aspen dieback and mortality. Thus, there has been a clear need to supplement our plot-based monitoring of aspen with alternative methods such as remote sensing (e.g., Hall and others 2003) and aerial surveys. In August 2004, the spatial extent of aspen mortality was assessed through 60 hours of aerial surveys within a 100,000-km² area of parkland where the 2001-2003 drought impacts were most severe (area outlined in Figure 2). The surveys were conducted by M. Michaelian, using GIS on a laptop integrated with a global positioning system (GPS). Analysis of this work is currently in progress, but the surveys showed moderate to severe levels of mortality across a large proportion of the aspen stands in the surveyed area, and in many stands, it appeared that drought had caused aspen mortality resembling the effects of stand-replacing fire.

In the areas of boreal forest covered by the CIPHA study (Figure 2), aspen mortality has also showed a general increase, but most areas of continuous aspen forest appeared generally healthy in 2004-2005. However, the tree-ring analyses show that drought has led to a similar decrease in regional aspen growth in both the boreal and parkland stands. Furthermore, detailed measurements at the BERMS Old Aspen site in the boreal forest have shown that aspen leaf area index decreased from about 2.8 in 2000-2001 to less than 2.0 in 2003 (Barr and others 2004).

These results suggest that the impacts of the 2001-2003 drought have not yet been fully realized, and thus there is a need for continued monitoring, using both plot-based and remotely sensed observation systems. Based on the complexity of interacting factors (Figure 1), we postulate that the drought could have indirect, long-term impacts, as the drought-weakened aspen may be more susceptible to damage by other agents, notably the potential for continued increases in wood-boring insects and fungal pathogens. On the other hand, aspen is highly adapted to stressors such as drought, partly owing to its ability to regenerate vegetatively from its roots following mortality of the aboveground stems. (Lieffers and others 2001). Recent studies have shown the role of carbohydrate reserves in spring leaf flush and late-season root growth of aspen (Landhäusser and Lieffers, 2002), which are in turn important to the success of aspen regeneration following disturbance (Landhäusser and Lieffers, 2003). One of the major knowledge gaps, however, concerns the mechanisms by which drought, in combination with other stressors, leads to landscape-level dieback and mortality of aspen and other species. Specifically, there is a need for improved understanding of the impact of multiple stressors on the carbohydrate status and hydraulic conductance (i.e., water transport) of aspen clones, and how these may operate to cause rapid dieback and regeneration, versus slow decline and permanent losses of aspen forest cover through clone death (Figure 1).

The recent drought has been unusually persistent and extensive compared with previous droughts in the Canadian Prairie Provinces (Bonsal and Wheaton 2005). Indeed, the impacts of this drought have been felt across a large part of North America, including the western U.S., where extensive mortality of aspen (USDA Forest Service 2005, Mary Lou Fairweather, Pers. Comm.) and other forest types such as pinyon...
pine (Breshears and others 2005) has been recorded. There is increasing evidence that droughts such as this may become more frequent and severe in future, so that an emerging priority for the future is the testing and implementation of innovative forest management practices to cope with drought and other extreme climatic events (Hogg and Bernier 2005). Under a changing climate, successful adaptation will also depend on increased communication of knowledge across disciplines and across land jurisdictions, to develop the integrated monitoring systems that are necessary to detect large-scale changes in our forests, especially in the drought-prone areas in the western portions of this continent.

Acknowledgements

The CIPHA study was conducted through funding support from the Canadian Forest Service, the Government of Canada’s Climate Change Action Fund, the Program of Energy Research and Development, the Meteorological Service of Canada, and Mistik Management Ltd. We thank T. Hook for conducting the tree-ring measurements and T. Hook, M. Salomons, R. Brett, J. Hammond, P.A. Hurdle, B. Tomm, T. Varem-Sanders, J. Weber, A. Engel, A. Johnson, S. Martin, R. Raypold, D. Sherling, M. Schweitzer, J. Snedden, V. Van Egteren, E. Van Overloop, B. Vroom, C. Charmin, P. Christensen, A. Durand, A. Gossell, A. Hamelink and T. Young for field and laboratory assistance. We also thank the personnel from numerous land management agencies for providing assistance in site selection and maintenance, and for permission to conduct this work. This work has benefited from useful comments and discussions with V.J. Lieffers and S.M. Landhäusser.

References


Photo by Thies
Are the Changes in Aspen Forests in Western North America a Forest Decline?

John C. Guyon II

Introduction

Trembling aspen (Populus tremuloides Michx.) is the most widely distributed forest tree species in North America (Jones 1985). Aspen is unique among the major western tree species in that it reproduces almost exclusively by coppice sprouts or “root suckers” produced by a shared clonal root system (Barnes 1966, Shepperd and Smith 1993). The above ground portion of an aspen clone is made up of individual aspen stems or “ramets” (Jones and DeByle 1985).

Across its’ broad range, aspen plays a great diversity of ecological roles (Romme and others 2000). Some aspen forests are classified as stable, and self-regenerating, usually in the absence of competing late successional species. But aspen is known primarily as a shade intolerant species that relies on fire or other disturbance to break apical dominance of the overstory, stimulating a new crop of root suckers and removing later successional conifers. If disturbance does not occur, then aspen clones can yield to later successional species. Some aspen clones have existed for thousands of years in the presence of later successional species, maintained by repeated disturbance over time (Kemperman and Barnes 1976). Aspen has also long been known to be prone to a large number of damaging agents including diseases, insects, and animal herbivory (Baker 1925, Hinds 1985, Jones and others 1985, DeByle 1985).

Recently, both qualitative and quantitative assessments of aspen forest condition have noted dieback or “decline” of aspen at a number of scales across western North America (Kay 1997, 2003, Bartos and Campbell 1998, Hogg and Schwarz 1999, Frey and others 2001, Kay and Bartos 2000, Ripple and others 2001, Hogg and others 2002, Rodgers 2002). Some authors have questioned this reported trend towards a decrease in the health of aspen stands and aspen acreage, and have reported an increase in aspen forest cover (Manier and Laven, 2001) over the last 100 years, or persistence of aspen at a landscape scale (Kaye and others 2001). Where decline has been noted the reasons given for this decline are many, including fire exclusion (Gallant and others 2002) succession to conifers and fire suppression (Rodgers 2002), persistent animal damage (Kay 1997), and weather events along with herbivory leading to death of clones (Fairweather 2005).

Regional Review

Several trends in aspen forest condition are evident from different areas where aspen research has been conducted across North America. In northwestern Wyoming, the area near the 2005 WIFDC, mortality rates for aspen ramets have been reported by several authors. (Krebill 1972, Guyon 94, Hart and Hart 2001). Annual mortality rates range between 0.1% and 5.0% and vary with size class and amount of animal damage. Hart and Hart (2001) noted that mortality rates nearly double in all size classes when heavy elk use was present. Generally, mortality rates are highest in small trees and decrease with age (Hart and Hart 2001, Mueggler 1994). Reports by Romme and others (2001) and Larsen and Ripple (2001) have both reported that there is a lack of overstory recruitment in the Yellowstone area due to heavy elk use was present. Generally, mortality rates are highest in small trees and decrease with age (Hart and Hart 2001, Mueggler 1994). Reports by Romme and others (2001) and Larsen and Ripple (2001) have both reported that there is a lack of overstory recruitment in the Yellowstone area due to heavy use by elk. Additionally, several authors have reported damage to aspen clones, often to the point where few suckers reach over 2 meters in height, with and without major disturbance, in several locations in northwestern Wyoming (Kay 2001a, 2001b, Dieni and others 2001, Hart and Hart 2001). Conversely, Barnett and Stolgren (2001) reported that 44% of aspen clones sampled in Gros Ventre drainage with heavy elk use had at least some suckers reaching over 2 meters in height. Wolves have been recently reintroduced into the Jackson Hole area, and perhaps wolves will impact elk browsing patterns via
trophic cascades similar to what has been reported in the Yellowstone area (Ripple and others 2001).

The situation in Canada appears to be more complex, and it is worth noting that wolves were never eliminated from these ecosystems like they were in the continental U.S.A. The presence of wolves, the dominant non-human predator in most trophic level models of aspen/elk/predator interactions, greatly complicates elk behavior and herbivory patterns. Overall, herbivory of aspen clones is not a critical factor in most areas in Canada that have been studied so far (T. Hogg, personal communication). Several Canadian authors have attempted to model and explain “aspen dieback” across over 100,000 km² of aspen forest (Frey and others 2004, Hogg and others 2002).

In Colorado, aspen stands are extensive and occupy a wide range of sites and overall ungulate use is relatively light (Romme and others 2000). Some studies have documented an increase in the amount of aspen forest (Manier and Laven 2000, Kulakowski and others 2004) in Colorado when compared to forest conditions in the 19th century. Other studies have concluded that aspen forests are declining due to intensive elk usage (Baker 1997, Olmstead 1979), but a recent study in the same area concluded that aspen is successfully regenerating except in the areas with heaviest elk usage (Kaye and others 2000). Conversely, Bartos (2000) reported a 49% decrease in aspen acreage in Colorado. Some reports of clone death have been reported in Colorado due to a complex of disease activity, site related and environmental factors (Jacobi 2000). Extensive outbreaks of defoliating insects have been noted in some cases (Jones and others 1985), and several diseases, especially canker diseases are common (Juzwick and others 1978).

Rodgers (2002) used USDA Forest Service, Forest Health Monitoring (FHM) permanent plot data to conduct an assessment of aspen forest condition in the Southern Rockies Ecoregion containing the states of Colorado, Wyoming and Idaho. He concluded that 61% of all plots with aspen present were “in a transition away from long-term aspen sustainability.” The most important factors in Rodger’s multivariate principle component analysis were: other species present and regenerating, stand age, and presence of damaged stems. Aspen had the highest level of damage (including forest diseases) of any of the principle species found on the FHM plots.

In Utah, a 59% decrease in acreage of aspen forest has been noted by Bartos and Campbell (1998). The primary causes given were fire exclusion and ungulate browsing. Kay and Bartos (2000) reported that aspen subjected to a combination of deer and cattle grazing failed to regenerate or had substantially reduced numbers of sprouts. Aspen had the highest amount of insect and disease damage of any tree species in Forest-wide Forest Health Protection surveys of the Fishlake and Dixie National Forests in Utah (Guyon 1994, Wadleigh 1995) with 80% and 72% the stems over 5” DBH showing some form of damage respectively.

In Arizona, recent drought, severe frost events, and grazing have led to overstory mortality, and in some cases clone death in as little as one year. These clones are typically smaller than aspen clones in Colorado and Utah, and are consequently more vulnerable to animal herbivory particularly when animals from a large area concentrate in already stressed clones.

**Clones and Decline**

Before applying decline concepts to aspen forests, it’s important to consider the clonal nature of aspen, and the relative importance of survival of individual ramets versus the survival of the clone. It can be argued that mortality of ramets is relatively trivial unless that mortality gives us some reason to believe that the clone is showing a decrease in health or the clone dies. Aspen ramets age and die like any other tree, and periodic death of ramets as cohorts age should be expected. In this context, death of overstory ramets, or young ramets for that matter, can be equated with branch dieback in non-clonal species. It’s also important to keep in mind that in some ways, a sudden event that kills overstory ramets and stimulates a new crop of root suckers could be beneficial to the long-term health of the clone if additional factors are not acting on the new root suckers and the clone has time to recover the carbon reserves lost from ramet mortality.

Two pieces of information point to the idea that the clone is basic unit on which a potential forest decline of aspen acts. First, ramets within a given clone are genetically identical, and should respond similarly to stress. Of course, older stems have different physiological requirements than young stems, but if they share the same root system and carbohydrate reserves, both young and old stems should experience similar levels of stress. Secondly, several authors have reported that resistance to pathogens varies by clone (Harniss and Nelson 1984, Hinds 1985, Guyon 1994, Wall 1971). Therefore, this discussion will center on applying the decline concept to aspen...
clones to see if the decline concept sheds any light on current trends in aspen forests.

A number of definitions of a forest decline exist (Manion and LaChance (1992), and in order to determine if the forest decline concept is useful in describing what is currently happening with aspen forests, it’s important to pick a functional definition. Manion’s definition states that a forest decline is: “an interaction of interchangeable, specifically ordered abiotic and biotic factors to produce a gradual general deterioration, often ending in the death of trees” (Manion 1982). Manion’s definition includes the terms predisposing, inciting and contributing factors, and defines them as follows:

*Predisposing factors* place a long-term stress on the host making it susceptible to other factors.

*Inciting factors* cause a short-term drastic injury to the host.

*Contributing factors* appear after the other two factors have stressed the host and are indicators of a weakened host.

Inherent in Manion’s definition is the idea that these factors are “specifically ordered,” and all categories are involved in a decline. Frey and others (2004) defined the predisposing, inciting and contributing factors in Canada, and the literature in this review are similarly summarized in Table 1. Since it is already being applied, Manion’s definition appears to be a good choice to evaluate use of the decline concept on aspen forests.

**Table 1.** Predisposing, inciting and contributing factors involved in aspen forests (Following Frey and others, 2004)

<table>
<thead>
<tr>
<th>Factor</th>
<th>Dominant spatial scale</th>
<th>Dominant time scale</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Climate</td>
<td>Landscape/Region</td>
<td>Long-term</td>
<td>Hogg and Hurdle 1995</td>
</tr>
<tr>
<td>Sucessional Processes</td>
<td>Stand</td>
<td>Decades</td>
<td>Liefferts et al. 2001, Shepperd et al. 2001</td>
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<tr>
<td>Ecosite</td>
<td>Stand</td>
<td>Long-Term</td>
<td>Stoeckler 1960, Shields and Brockham 1981</td>
</tr>
<tr>
<td>Forest Structure</td>
<td>Stand</td>
<td>Decades</td>
<td>Mueller-Dombois 1992</td>
</tr>
<tr>
<td>Age</td>
<td>Stand/Landscape</td>
<td>Decades</td>
<td>Brandt et al. 2003</td>
</tr>
<tr>
<td>Local Problems</td>
<td>Stand/Landscape</td>
<td>Variable</td>
<td>Karnosky et al. 1999,</td>
</tr>
<tr>
<td>Clonal Aspects</td>
<td>Tree/Stand</td>
<td>Long-Term</td>
<td>Schier and Campbell 1980, Kneseshaw and Bergerson 1998</td>
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<tr>
<td>Inciting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drought</td>
<td>Landscape/Region/Local</td>
<td>&lt;5 Years</td>
<td>Hogg et al. 2002, Guyon 1996 et al</td>
</tr>
<tr>
<td>Thaw-Freeze Events</td>
<td>Landscape/Region</td>
<td>&lt;5 years</td>
<td>Fairweather et al 2005</td>
</tr>
<tr>
<td>Wildlife stem damage</td>
<td>Stand/Landscape</td>
<td>&lt;Month</td>
<td>Hart and Hart 2001</td>
</tr>
<tr>
<td>Browsing Damage</td>
<td>Stand/Landscape</td>
<td>&lt;3 years</td>
<td>Kay and Bartos 2000</td>
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<tr>
<td>Defoliation</td>
<td>Stand/Landscape</td>
<td>&lt;5 years</td>
<td>Jones et al, Hogg et al. 2002, Harniss and Nelson 1984</td>
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<tr>
<td>Contributing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insect Borers</td>
<td>Tree/Stand</td>
<td>&lt;5 Years</td>
<td>Jones et al 1985</td>
</tr>
<tr>
<td>Windthrow</td>
<td>Tree/Stand</td>
<td>&lt;1 day</td>
<td>Webb 1989</td>
</tr>
<tr>
<td>Pathogens</td>
<td>Tree/Stand</td>
<td>Variable</td>
<td>Hinds 1985</td>
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<td>Of Regeneration</td>
<td>Clone</td>
<td>Variable</td>
<td>Hinds and Shepperd 1987, Jacobi 2001</td>
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<tr>
<td>Of Mature stems</td>
<td>Tree</td>
<td>Variable</td>
<td>Juzwick et al. 78, Hart and Hart 2000,</td>
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</table>
The clonal nature of aspen presents a further complication in applying forest decline concepts to aspen. Declines are usually thought of as disease complexes effecting older populations of trees (Manion 1982, Mueller-Dombois 1992). While this concept is easy to apply to older aspen cohorts, it doesn’t work well for aspen clones which are capable of self-renewal by root suckers and can live for thousands of years. So called “stable” aspen clones show this point well. If not experiencing substantial grazing pressure or competition from conifers, stable aspen clones are capable of creating a nearly all-aged type canopy structure. The oldest ramets could all die in stable aspen clones, but the clone could remain healthy and functioning as long as new suckers are produced. If you apply the idea that declines happen to aging populations to clones across a whole landscape, the idea of an aging population makes sense when a large number of the clones are apparently “declining”.

Overall, it might be easier to think of aspen decline of as a carbon balance equation. Any factor that causes a reduction in the overall carbon reserves by killing ramets or stressing the clone should contribute to the decline of the clone. For example, if factors such as a severe drought causing xylem cavitation and overstory dieback were to be followed by grazing of the root suckers, a severe loss in carbon reserves could occur, and the clone could decline and die rapidly. In such a case, the Manion model of a forest decline doesn’t fit well because both severe drought and heavy grazing pressure would be considered inciting factors, and the clone could die without predisposing and contributing factors be involved at all. The carbon balance equation model could be used in the case described above; if the net effect of the drought and subsequent grazing pressure is sufficient to exhaust the carbon reserves of the clone the clone could die, and defining predisposing, inciting, and contributing factors is unnecessary.

When rates of mortality due pathogens and insect damage are too low to break apical dominance of the overstory, it should lead to a gradual decrease in the carbon reserves of clones, and therefore a gradual decrease in clonal vigor. When this occurs paired with grazing pressure sufficient to remove most of the sucker sprouts, the clones are often viewed as “declining” (Figure 1). Other factors that cause low level stress on clones, such as low level grazing pressure or competition from invading conifers (Figure 2), should cause a similar impact.

Conclusions

Overall, several distinct pictures of aspen conditions are evident. First, in Canada, where large areas of aspen forest have been stressed by a number of agents including defoliators and drought, but grazing pressure due to animals is not acute except for localized areas, the decline concept as applied by Frey et al can be useful in helping to summarize the forces acting on these aspen clones.

Figure 1. “Declining” aspen clone with cankers and insect damage in Wyoming. Photo courtesy of Charles Kay.

Figure 2. Aspen with competing conifers. Photo courtesy of Charles Kay.

In Colorado, in the heart of aspen range in the western U.S., is in some ways similar, but more controversial. Questions exist as to whether the current distribution of aspen is an expansion due to broad-scale disturbances from the latter half of the 19th century which led to more aspen forests than the area had carried before. If this is true, then a decrease in aspen acreage paired with an increase in conifer cover could be a restoration of normal landscape patterns. The impact of ungulate browsing
in Colorado is also controversial, with some authors reporting considerable damage, and others reporting consequential damage only in the areas with the highest levels of grazing pressure. The decline concept would only be useful in these areas if it is agreed that decline is happening at all.

In the much of the rest of the western U.S., where grazing pressure or succession to conifers are more likely to be the driving force behind a reduction in the health of clones, the decline concept may not be needed either. It’s simpler to attribute the decrease in aspen range and clonal health to grazing pressure or succession without worrying if it’s a decline or not. The role of insects, diseases, and environmental perturbations play in the west needs further research, and until this role can be better understood the simple explanations are hard to refute. It is important to note that in many of these cases at least some of the factors in Manion’s view of a decline are present, but rarely are all three different types of factors clearly defined. For example, in clones that appear to be declining due to succession to conifers, contributing factors like aspen canker diseases are common. Due to aspen’s wide ecological amplitude, it’s also quite likely that at least some predisposing type stress is also present in most of these aspen forests. What’s missing in many cases is an inciting factor that causes a large draw on carbon reserves and pushes these clones into a downward spiral.

In cases where an environmental factors play a dramatic role, and grazing pressure is also high, leading to the death of ramets and clones such as what is currently occurring in Arizona, the decline concept is somewhat questionable, partially due to the speed of the death of clones. The leading factors in this case seem to be drought, defoliation, frost damage and grazing. If contributing factors such as canker disease are also involved, the case for applying the decline concept is stronger. Aspen clones in southwestern U.S.A., as well as in other areas on the edges of aspen’s range, are of particular concern to forest managers because drastic reductions in aspen acreage have occurred (Bartos 2000) in these areas.

References


Panel: Past and Current Research on the Biology of Armillaria ostoyae in the Southern Interior of British Columbia

Duncan Morrison, Moderator

Introduction

Welcome to the panel on Armillaria ostoyae research in British Columbia. As is the case in the northwestern U.S.A., the incidence and amount of damage attributable to the Armillaria root disease caused by A. ostoyae is much greater east of the Coast (Cascade) Mountains than west of them. Hence, nearly all research on the disease in B.C. has been done in the southern interior. Initially, work focused on determining the distribution of damage, making a host susceptibility table, monitoring stumping trials and developing management guidelines to reduce losses. The development of methods by Korhonen and Anderson to distinguish among the species of Armillaria using single spore cultures was an important milestone, allowing us to determine species distributions and behavior. Over the past 15 years, incremental funding has permitted research on a wide range of questions. The panelists will describe the results of some of that work.

Program

Fifteen years of research on Armillaria ostoyae: A legacy of the western root disease model project and FRDA II.

Duncan Morrison, Canadian Forest Service, Victoria, BC.

Resistance in western redcedar against Armillaria ostoyae host response to infection and mortality incidence in juvenile stands in the southern interior of BC.

Michelle Cleary, University of BC, Vancouver, BC.

Determination of Armillaria ostoyae infection events in roots of juvenile Douglas-fir, subsequent fungal spread and effects on tree growth.

Mike Cruickshank, Canadian Forest Service, Victoria, BC.

Impact of the research

Communicating the results of the research to practitioners at workshops, at field tours and in user-friendly publications has raised awareness of Armillaria root disease in B.C. With increasing awareness, there has been a gradual change toward management practices that reduce or do not increase damage. Use of stump removal and species mixtures for regenerating diseased sites is increasing. The model developed by Bill Bloomberg for Phellinus weirii is being adapted for A. ostoyae using our results and crystal ball. The A. ostoyae model is attached to TASS, the B.C. Forest Service growth and yield model for second growth stands. The model was used to develop Armillaria-specific operational adjustment factors that reduce the anticipated yield from diseased stands.

Photo by Schwandt
Fifteen Years Of Research On Armillaria ostoyae: A Legacy Of The Western Root Disease Model Project And FRDA LI

Duncan Morrison

Introduction

In the mid-1980s the US Forest Service began a project to develop a model of Armillaria [Armillaria ostoyae (Romagn.) Herink] and Phellinus [Phellinus weirii (Murr.) Gilb.] root diseases for silvicultural planning. The model is now known as the western root disease model. When the modelers began asking the pathologists for data, it became clear that we had little hard data on important root disease variables, especially for Armillaria. In 1990, I wrote a problem analysis which included a research needs survey of practitioners in industry and the BC Forest Service. The model project, research needs survey and problem analysis identified priority topics for Armillaria root disease (DRA), listed below.

- DRA mortality curves for a rotation.
- Effects of precommercial thinning on incidence of infection and mortality.
- Effects of selective logging.
- Species resistance: host response to infection and susceptibility to killing.
- Productivity of diseased stands.
- Disease dynamics: detectable vs actual amount, effect of climatic region on incidence, variation in isolate virulence, etc.
- Other: occurrence in ecological units, genetic basis for resistance.

One or more studies have been completed, and results published, or are ongoing on each of these topics. Between 1990 and 1996, most of the funding for the work was provided by FRDA II, the Canada/BC Forest Resource Development Agreement and since 1996, by the BC Forest Science Program.

In BC, A. ostoyae occurs from 49º N to about 53º N, with a few scattered collections north of that line, notably on the Queen Charlotte Islands and near Prince Rupert. The highest incidence and greatest damage occurs in the moist ecozones of the southern interior, and that is where most of the research has been done in the last fifteen years.

Priority Topics

DRA mortality curves

The temporal and spatial distributions of mortality caused by A. ostoyae and other agents were recorded on 2 ha plots in Douglas-fir plantations established in 1967 and 1972 in the Interior Cedar Hemlock biogeoclimatic zone (ICH) (Morrison and Pellow 1994). At plot establishment in 1984 and 1986, respectively, and at each biennial assessment thereafter the location of each dead tree was mapped and its year of death determined.

Douglas-fir mortality began five years after planting. From the onset of mortality to the present in each plantation, the rate of mortality has been similar, 1.0% per year in one plantation and 0.36% in the other. We consider these rates to be high and low, respectively for plantations in the ICH. Mortality has occurred in patches, and coalescence of adjacent patches has created openings up to 0.045 ha in size. Gradually, openings have filled with broadleaved and climax tree species and brush. Other agents, notably breakage, windthrow and snow press, began to kill trees about age 25 and had killed 8% and 2% of trees in the two plantations by age 35.
To verify the Rotsim/Armillaria model we need mortality curves for an 80-year-old rotation. It would be difficult to develop curves from surveys in existing stands due to uncertainty in determining cause of death and age at death on long-dead trees.

**Effects of precommercial thinning**

Cruickshank and others (1997) determined the incidence of Armillaria species in precommercial thinning stumps and spread of *A. ostoyae* from colonized stumps to adjacent Douglas-fir trees at 11 sites in four ecological zones on a coast-interior transect. *A. ostoyae* and *A. sinapina* were found in stumps in all zones, with the incidence of *A. sinapina* being higher in coastal zones and of *A. ostoyae* in interior zones, especially in the moist zone. *A. ostoyae* transferred from colonized stump roots to crop tree roots and formed lesions. In most zones, lesion size was related to inoculum volume. The frequency of callusing at lesions on crop trees was related to bole volume, and was highest in the moist coastal zone.

Between 1991 and 1994, 8 installations were established in juvenile stands showing low to moderate incidence of *A. ostoyae* mortality. Each installation consisted of 10, 20 m by 20 m plots with a 5 m buffer; crop trees were tagged in all plots. Five plots were randomly selected for thinning to BC Forest Service specifications and five were not thinned. Ten years after thinning, results are similar to those reported by Filip and Ganio (2004); there was no difference in incidence of crop tree mortality between treatments. However, in some thinned plots in installations where inter-tree distance is large, killing of 2-3 trees has produced large unstocked openings. Specifications for precommercial thinning in stands showing Armillaria root disease should prescribe a lower inter-tree distance.

Our results suggest that *A. ostoyae* has enough inoculum potential in thinning stumps to cause lesions on the roots of crop trees, but apparently has insufficient energy over several years to prevent the host halting advance of the fungus and callusing the lesion.

**Effects of selective harvesting**

Individual tree, group selection and shelterwood harvesting systems have been used for decades in the southern interior, and public resistance to clearcutting has led to increased use in recent years. The literature from elsewhere (Hagle and Goheen 1988, Shaw and others 1976) and anecdotal evidence from BC suggest that extensive use of these systems could increase *A. ostoyae* caused mortality of residual overstory trees and regeneration. The objective of our retrospective study was to determine the effects of selective cutting on the incidence of Armillaria root disease on residual trees and post harvest regeneration, on the incidence of mortality and on merchantable volume (Morrison and others 2001).

Four sites were studied, two in the dry Interior Douglas-fir (IDF) zone, one in the moist ICH, and one in the wet Engelmann spruce subalpine fir (ESSF) zone. Time since selective harvesting ranged from 13 to 30 years at the four sites. Sites were selected that had adjacent or interspersed undisturbed areas of the same timber type which had been cutover. Five 0.04 ha plots were established in the cutover and undisturbed parts at each site. Trees were tagged and their DBH, species, condition and coordinates were recorded. Trees were examined for basal signs and symptoms of Armillaria root disease. Trees were felled and trees and stumps were pulled from the soil by an excavator. Age of trees was determined on a stump disc. All root systems were mapped and the location of each *A. ostoyae* lesion and the host response were recorded. Merchantable trees were assigned to one of five classes based on tree condition, location of lesions and host response. Merchantable volumes were determined using the BC Ministry of Forests cruise compilation program.

At the two IDF sites 50-60% of selective harvesting stumps were colonized by *A. ostoyae* compared to 100% and 55% at the ICH and ESSF sites, respectively. In undisturbed plots, incidence of trees with *A. ostoyae* lesions belowground was 3.5% and 10.6% at the IDF sites, 77.3% in the ICH and 34.5% in the ESSF; in cutover plots the values were 14.6%, 52.4%, 88.1% and 36.9%, respectively. The probability of a tree being infected increased with DBH. Nearly all post–harvest regeneration occurred on the IDF sites where 6% and 26% of regeneration was diseased or had been killed. The number of *A. ostoyae* lesions per tree was higher on trees in cutover plots than in undisturbed plots. At the IDF sites, 85% and 99% of lesions were progressive, i.e., not callused, compared to 59% in the ICH and 73% in the ESSF. The greatest effect of the disease on current and future merchantable volume was seen at the IDF sites, and especially at the IDF site that was cutover 30 years ago. Little damage is evident until at least ten years after a selective harvest.

**Species resistance**

Tables giving the relative susceptibility of tree species to infection or killing by *A. ostoyae* have
been based largely on observations in mixed species stands (Morrison and others 1991).

Recently, Robinson studied the process of lesion formation and host response to natural infection by *A. ostoyae* of 6-8-, 18-19- and 85-95-year-old western larch and Douglas-fir (Robinson and Morrison 2001, Robinson and others 2004a, 2004b). Infection in the roots of 6-8-year-old trees advanced freely, overcoming any host resistance, quickly girdling the root collar and killing the trees. In 18-19-year-old trees, 43% of infections on larch roots and 27% on Douglas-fir roots were confined to lesions bounded by necrophylactic periderms with multiple bands of phellem. Host response was similar in 85-95-year-old trees, but the percentage of confined lesions was higher than in younger trees. The results suggest that larch shows resistance to *A. ostoyae* at a younger age and with greater frequency than Douglas-fir.

Currently, Michelle Cleary is conducting a similar study comparing response of western redcedar, western hemlock and Douglas-fir (see paper in this proceedings).

The results of these studies will allow us to recommend species mixtures for regenerating infested sites with greater confidence of success than heretofore.

**Productivity of diseased stands**

Bloomberg and Morrison (1989) measured stem volume growth of 80-100-year-old Douglas-fir in 5 classes defined by percentage of basal circumference showing resinosis caused by *A. ostoyae* (putatively disease-free; infected, no resinosis; resinosis <50%; resinosis 50-<100% and 100%, i.e. dead). Growth, expressed as a percentage of stem volume at the start of each 5-year period, decreased significantly as resinosis increased due to mycelial colonization of the tree base. Trends during the past 30 years showed greater declines in growth in classes with >50% resinosis relative to the disease free class than in the <50% classes. Growth decline began earlier, up to 25 years ago, in the >50% classes than in other classes.

In a 55-year-old Douglas-fir stand in which 80% of trees were infected by *A. ostoyae*, growth repression in the stand was 11% (Cruickshank and Morrison, unpublished).

Currently, Mike Cruickshank is determining growth loss in juvenile Douglas-fir plantations in the ICH (see paper in this proceedings).

**Visible vs actual incidence**

In our studies where trees were removed from the soil (Morrison and others 2000, 2001), trees were examined, without soil excavation, for basal signs and symptoms of *A. ostoyae*. When belowground incidence of infection was determined we could calculate the percentage of diseased trees that could be detected by a survey.

In planted and natural juvenile coniferous stands, 50% of trees with belowground infection showed aboveground basal signs or symptoms at five sites in the dry climatic region, 26% at sites in the moist region and 23% in the wet region (Morrison and others 2000).

In undisturbed plots in mature stands, 27, 15 and 22% of infected trees in the dry, moist and wet climatic regions, respectively, showed aboveground symptoms. In cutover parts of the same stands, 63, 20 and 29% of infected trees were detected.

When interpreting the results of surveys for Armillaria root disease, it is important to recognize that only a fraction of the trees infected belowground will show aboveground symptoms.

**Effect of climatic region on incidence**

Incidence of trees infected by *A. ostoyae* was consistently higher in the moist and wet climatic regions than in the dry region (Morrison and others 2000, 2001). Larger numbers of stumps colonized by the fungus, more rhizomorphs and a higher frequency of transfer at tree-inoculum contacts (Cruickshank and others 1997) in the moist and wet regions are the probable reasons for the difference. The higher percentage of infected trees showing aboveground symptoms in the dry region may be attributed to the lower frequency of callusing at lesions, resulting in more infections reaching the root collar.

**Variation in isolate virulence**

The virulence of *A. ostoyae* isolates from coastal and interior British Columbia, elsewhere in North America and Europe was assessed on Douglas-fir seedlings in pots during a 3-year trial. Isolates from most geographical locations infected similar proportions of seedlings, had similar average damage scores and killed a similar percentage of diseased seedlings. Isolates from the coastal region had a significantly higher probability than interior isolates that a diseased seedling received a damage score >3 on a 1-5 scale, and coastal isolates killed a higher proportion of diseased seedlings than interior isolates. The results indicate that the higher incidence and longer duration of mortality in the southern interior
of British Columbia compared to the coast can not be attributed to greater virulence of interior isolates of *A. ostoyae* (Morrison and Pellow 2002).

**Time to death**

How long does *A. ostoyae* take to kill a planted Douglas-fir? One of the responses of Douglas-fir to an *A. ostoyae* root infection is to produce a row of traumatic resin canals in the current annual ring of the infected root and lower bole above the infected root. These traumatic resin canals can be used to determine the year of the first and subsequent infections. A disc was cut at the soil line from 493 Douglas-fir trees killed two years or less prior to sampling in 32, 7- to 33-year-old plantations. The time from first infection by *A. ostoyae* until death increased from an average of about 2 years at age 7, to about 11 years at age 25 and then to 20 years at age 32.

**Occurrence in ecological units**

The biogeoclimatic ecosystem classification used in BC has the subzone as its basic unit. Subzones are grouped into zones, and divided into variants. The site series classification groups ecosystems within a subzone or variant that have similar environmental properties and vegetation (Braumandl and Curran 1992). Practitioners would like to know in which units *A. ostoyae* will occur, preferably at the site series level, so that management prescriptions can account for Armillaria root disease.

Preliminary work indicates that within the geographical range of *A. ostoyae* in the southern interior of BC the fungus’ occurrence is limited by extremes of soil moisture and temperature. For example, in variants in the ICH the fungus will be found in all site series except hydric and in the ESSF, it will occur in mesic series in low elevation variants.

**Genetic basis for resistance**

A preliminary study by Mike Cruickshank and Barry Jaquish, a geneticist with the BC Forest Service, suggests that some Douglas-fir families show greater resistance than others.

**References**


Resistance in Western Redcedar Against Armillaria ostoyae: Host Response to Infection and Mortality Incidence in Juvenile Stands in the Southern Interior of British Columbia.

Michelle R. Cleary and Bart J. van der Kamp

Abstract — Necrophylactic periderm formation and compartmentalization of infected tissue was studied in tissue samples collected from the roots of 20-30 year-old western redcedar (Thuja plicata), western hemlock (Tsuga heterophylla) and Douglas-fir (Pseudotsuga menziesii) trees infected with Armillaria ostoyae. A higher frequency of resistance reactions were induced in western redcedar trees following invasion by the fungus compared to the other conifers. In a survey of juvenile mixed species plantations throughout the southern Interior of BC, mortality in Douglas-fir trees was at least ten times more frequent than in western redcedar trees. The frequency of western redcedar trees showing effective compartmentalization at the root collar was significantly higher than in Douglas-fir trees. Results indicate that western redcedar is more resistant to A. ostoyae than other conifers and the inclusion of cedar in higher proportions when planting infested sites may reduce the overall impact of Armillaria root disease.

Introduction

Armillaria ostoyae (Romagn.) Herink is a significant health concern in the southern Interior of British Columbia and is particularly damaging in forests located in the Interior Cedar-Hemlock (ICH) biogeoclimatic zone. Within this zone, aboveground symptoms of Armillaria can be detected in only one-quarter of the trees with belowground infection (Morrison et al. 2000). Cumulative mortality in Douglas-fir (Pseudotsuga menziesii (Mirb) Franco) stands can be as much as 20% by age 20-years resulting in undesirable stocking in juvenile stands (Morrison and Pellow 1994).

Planting species of low susceptibility to killing by A. ostoyae can reduce losses in new plantations. There are no woody hosts that show complete resistance to Armillaria, although some are more tolerant than others. It is generally accepted that all conifers less than 15 years of age are highly susceptible to killing by Armillaria root disease and some conifers will show greater resistance to the fungus with age (Morrison et al. 1991, Robinson and Morrison 2001).

When trees become infected by A. ostoyae, defense mechanisms on root systems will determine the outcome of a particular reaction as being either susceptible or resistant. Several studies have shown necrophylactic periderm (NP) to be involved in resistant reactions in woody plants in response to mechanical injury or pathogenic invasion (Blanchette and Biggs 1992; Wahlstrom and Johansson 1992; Robinson and Morrison 2001).

Knowledge of effective resistance among conifers that are typically found intermixed with planted Douglas-fir in the ICH zone, namely western redcedar (Thuja plicata Donn ex D. Don) and western hemlock (Tsuga heterophylla (Raf.) Sarg.), and the age at which resistance is expressed in either of these species would enable forest managers to explore new silvicultural options with respect to species mixtures and potentially reduce losses caused by Armillaria in new plantations.

Preliminary results under the current study showed that the frequency at which resistant reactions were induced following invasion by the fungus was significantly higher in western redcedar trees than in Douglas-fir and western hemlock trees (Cleary et al. 2004). Indirect evidence from previous studies also suggests that cedar may be more resistant to Armillaria (DeLong 1996, Morrison et al. 1988, Morrison et al. 2000). Together these findings imply lower disease incidence for western redcedar relative to other common conifers.
The aim of this research was two-fold. First, to identify and characterize natural resistance mechanisms operating in the roots of western redcedar, western hemlock and Douglas-fir trees, specifically targeting necrophylactic periderm formation in the bark and barrier zone formation associated with compartmentalization of infected woody tissue. Second, to demonstrate through a survey of juvenile mixed conifer stands that the presence of these natural resistance mechanisms leads to mortality rates in western redcedar that are significantly lower than in other common conifers.

Inoculation Trials

Methods

From 2002-2004, four field inoculation trials were implemented at three sites in the moist-warm subzone of the ICH biogeoeclimatic zone in the southern interior of BC. Inoculum blocks consisted of fully colonized segments of Garry oak (Quercus garryana Dougl.) branches inoculated with A. ostoyae (isolate 87-01, provided by D. Morrison, CFS). In the field, the root systems of 20-30 year-old western redcedar, western hemlock and Douglas-fir trees were carefully exposed so as to not injure the outer bark tissue and trigger a host response independent of Armillaria infection. A single root was inoculated with an inoculum block and when possible, all inoculations on a single tree (up to 4) occurred on individual roots. Sampling of the roots occurred at different intervals over the course of a year, each year. Root systems were excavated, examined for Armillaria-caused lesions at inoculum contact, and harvested from the ground. Tissue samples were dissected to reveal the radial face of the infection front which could be recognized by the presence of either a necrophylactic periderm separating healthy, uncolonized inner bark tissue from infected, colonized inner bark tissue, or browning of tissue in advance of a penetrating mycelium. Bark samples were coated in OCT® (Optimum Cutting Temperature compound; Thermo Shandon, Pittsburgh, PA, USA) and immediately stored in liquid nitrogen.

In the lab, tissue samples were dissected in a cryostat set at –20°C. Frozen sections mounted on slides were examined microscopically under tungsten illuminated bright field (BF) and using a mercury lamp with fluorescence filter combinations for blue light (BL) and ultra-violet (UV) excitation on a freezing stage set at 35°C. Microscopic observation of infected bark tissue in this context permits physiological interpretation of any anatomical changes of cells associated with infected tissue based on changes in fluorescence characteristics when compared with healthy tissue. Additional sections were thawed and stained for lignin (using phloroglucinol + HCl) and suberin (using Sudan III or visualization by autofluorescence after staining with phloroglucinol + HCl). Woody samples showing compartmentalization and callusing were preserved in FAA, embedded in paraffin and later sectioned on a rotary microtome to describe barrier zone anatomy associated with compartmentalized woody tissue. Photomicrographs were taken of both frozen and stained cryostat sections.

Results

Results showed noticeable differences between species in their ability to produce successful resistance reactions (either a NP in the bark or effective compartmentalization) to contain the fungus.

A low frequency of resistant reactions occurred in both Douglas-fir and western hemlock roots. Slight variation in the frequency of successful resistance responses was observed from year to year, and between sites. The variation in response to infection exhibited by all species, including the rate at which NP was formed, may be related to the relative susceptibility of the host, changes in inoculum potential as the fungus advances in inoculated roots, as well as site and environmental influences. However, the low percentages of resistance responses observed in Douglas-fir and hemlock appeared to be consistent across all field trials.

In the majority of Douglas-fir and western hemlock roots, A. ostoyae was able to overcome initial host reactions and prevented the development of a NP resulting in a progressive lesion in the root. The adjacent phloem often appeared brown and either lacked significant hypertrophy or would appear to be invariably hypertrophied. At times, sporadic lignification of adjacent phloem tissue was seen in advance of a penetrating mycelium which indicated the initiation of non-suberized impervious tissue (NIT), a prerequisite for NP formation. However, distinct zones of NIT were frequently lacking and/or the fungus penetrated beyond the developing NIT zone. Following cambial invasion, few samples showed successful compartmentalization.

In hemlock roots, a NP would sometimes be formed initially, but then it was breached by the fungus. Further examination of breached periderms revealed that sclereids were an area weakness in the
development of new periderms. Sclereids will become part of the NIT zone, but a newly restored phellogen would often appear discontinuous around a cluster of sclereids and breached periderms often showed browning of tissue internal to a cluster of sclereids. The abundance of this cell type in the phloem of hemlock results in discontinuities in periderm barriers allowing Armillaria to grow through the tissue before a NP is complete.

However, field observations of naturally infected hemlock revealed that some trees are more successful at containing the fungus when infections occurred close to the root collar or on larger diameter roots where the NPs are more likely to consist of bands of thick- and thin-walled phellem: a structural characteristic that helps to impart additional resistance to the spread of Armillaria in other conifers like western larch (Robinson and Morrison 2001).

Resistant responses induced in cedar trees following invasion by A. ostoyae were up to seven times more frequent than in Douglas-fir trees. Little variation in the frequency of host reactions was observed in cedar between field trials.

Cedar frequently responded to infection by Armillaria by forming a necrophylactic periderm deeper in the bark tissue. A unique phenomenon involving a type of hypersensitive response consisting of rhytidome formation was found also in the vicinity of a site of initial penetration by the fungus. This response occurs following phellogen renewal and NP formation and appears to be non-specific, occurring in both abiotically wounded and inoculated roots. Successive periderm formation on either end of an Armillaria-caused lesion would extend for some distance proximally and distally along the length of the root forming one continuous rhytidome layer that may eventually be sloughed from the surface of the root.

Lesions to the cambium on cedar were frequently compartmentalized. The barrier zone formed in the uninjured cambium was comprised of a higher than average number of axial parenchyma that accumulated dense phenolic deposits and appeared to be very effective at limiting the extent of cambial invasion and/or girdling in roots.

Surveying Mortality Incidence in Juvenile Mixed Conifer Stands

Methods

Candidate sites for surveying mixed conifer stands were selected based on the following criteria:

1. 15-25 years-old stands planted to Douglas-fir with a significant (~30%) component of western redcedar in the understory.
3. Moderately to heavily infected with A. ostoyae.
4. No juvenile spacing.

At each site 10-m radius plots were established and all trees ≥ 3 cm DBH were recorded by species, tree size and disease status. The root collar was examined on all trees for evidence of old or current basal lesions. Non-lethal infections at the root collar were classified as either progressive (e.g. the fungus was advancing in the inner bark and cambial tissue) or callused (the fungus was compartmentalized and the spread of the fungus had been stopped).

The proportion of trees killed or infected by Armillaria was determined by species for each plot and site. Frequency data for species mortality was analyzed by Chi-square tests and a logistic regression model was also fitted to the data.

Results

Significant differences were found in the percentage of trees killed by A. ostoyae between species across all sites surveyed. The frequency of mortality for a particular host species is a direct measure of its susceptibility to killing by A. ostoyae and the ability of that species to survive in the presence of inoculum. Mortality caused by Armillaria was at least ten times more frequent in Douglas-fir than in western red cedar.

Cedar also had a lower percentage of progressive lesions at the root collar compared Douglas-fir and western hemlock trees. Most lesions of this type are “susceptible” host reactions showing no visible host response at the infection front in the form of NP formation. The smaller percentage of progressive lesions at the root collar in cedar compared to the
other conifers suggests that fewer cedar trees are at risk to being killed by *A. ostoyae*.

Compartmentalization and callusing at the root collar also differed among species. Effective compartmentalization was noticeably higher in cedar than in Douglas-fir and western hemlock trees.

Results also showed that the probability of mortality among trees infected by *Armillaria* depends on both species and tree size. Incidence of mortality was significantly greater in the smaller diameter size class than in the larger size classes for both species and cedar mortality was consistently lower than Douglas-fir. Although the risk of mortality decreases with increasing tree size in both species, the rate of decrease was noticeably greater among cedar compared with Douglas-fir trees.

There was an increasing trend in the proportion of infected trees showing effective compartmentalization and callusing and tree size for both species, but the increase was markedly greater among western red cedar compared with Douglas-fir trees.

In the majority of conifer plantations, cedar comes in as natural regeneration, so that trees are younger and smaller than the planted species. It is more likely that mortality in the planted Douglas-fir has been happening for at least a few years longer than in cedar. Nonetheless, reduced mortality rates exhibited in even the intermediate size classes and the higher frequency of callusing observed in the smallest diameter size class indicates that western redcedar is more resistant to infection by *A. ostoyae* and this resistance occurs much earlier than most other conifers.

Management Implications

Natural resistance mechanisms in the bark (including necrophylactic periderm formation and successive rhytidome formation around a point of invasion) and in the wood (compartmentalization of infected tissue) of cedar trees are effective at halting further spread of *Armillaria* in host tissue. Cedar appears to form these “resistant” reactions more frequently than other common conifers. It is more likely that the presence of these resistance mechanisms results in most infections being confined to tissue immediately surrounding a point of invasion, thereby limiting the extent of cambial invasion which might otherwise result in higher mortality rates in cedar.

The practical implications of using western red cedar are significant. The inclusion of cedar in higher proportions when regenerating infested sites may reduce the overall impact of Armillaria root disease. In stands with moderate to high levels of infection, cedar will act as a barrier to disease spread between susceptible conifer species and possibly minimize the risk of increasing secondary inoculum in the stand.

Acknowledgements

Funding for this research was provided by Forest Investment and Innovation Ltd. (FII), Forest Investment Account (FIA), Natural Science and Engineering Research Council of Canada (NSERC), IMAJO Cedar Management Fund, and the Canadian Forest Service - Pacific Forestry Centre. We gratefully acknowledge Duncan Morrison and Gary Jensen for their help with various aspects of the work, as well as the BC Ministry of Forests, Tolko Industries, and Pope & Talbot Ltd. for their continued support.

References


The Use of Traumatic Resin Canals as Markers of Infection Events in Tree Roots

M. G. Cruickshank

Summary

Douglas-fir trees infected with Armillaria ostoyae in the Interior Cedar-Hemlock biogeoclimatic zone from plantations aged 24, 30 and 32 years were examined to determine the date of infection. About 1000 trees in 25-10 m radius plots were removed from the soil with roots attached by a link-belt excavator with a clamshell attachment. Root systems were examined for root infections, stem discs were taken for volume determination, and the infection age of all lesions was determined using traumatic resin canals (TRCs). The year of infection at each root lesion was determined by counting annual rings from present until the occurrence of the TRCs. The infection date was used to calculate fungal spread characteristics and stem volume growth loss. Stem growth losses were determined from the volume difference between healthy and infected trees, and stand losses were determined from the sum of individual tree losses in the plots. The fungal spread rate at root lesions was found to vary with the type of infection (patch or girdle), the length of time that the fungus spread in the lesion, and the diameter of the tree (DBH). The fungal spread rate at girdled root lesions was double that of patch root lesions (p<0.0001). The fungal time to spread at roots was related to the spread rate by an inverse function (p<0.0001) where the spread rate was reduced the longer the fungus spread. For trees larger than 18 cm (DBH), the fungal spread rate was more than double the trees smaller than 18 cm (p=0.0294). Fungal spread rate at the root collar was affected by the same variables as those affecting root spread rate except that the rate was about half. Fungal spread rate at root collar lesions arising from a spreading girdled root was greater than the spread rate at collar lesions arising from a spreading root patch lesion, or a collar lesion initiated directly at the collar. The length of time the fungus spread at root and root collar lesions before it was stopped (callused lesion) was affected by the percent of infected roots on the tree, spreading longer when more of the roots on that tree were infected. In addition, the fungus also spread longer at girdled root lesions compared to patch root lesions (p<0.0001). Most root collar and root lesions were contained (callused) after 3 years and therefore rarely caused stem girdling. Some lesions are not contained for up to 20 years, and although they are infrequent, these lesions are very important because they can often result in girdling the root collar. Fungal containment at lesions has an associated cost for the tree that affects growth. Growth losses accumulated as a function of the time the tree had been first infected. Individual tree losses ranged from 37-49% and stand level losses ranged from 7-16% (average 9%) for stands of this age. The incidence of infection in these stands ranged from 23-60%; however, the fungus continues to infect the root system over time increasing the number of infected trees and roots.


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Root Disease Research in Support of Forest Management: Some Recent Examples

Walter G. Thies and Douglas J. Westlind

Introduction

For this panel we were asked to present examples from our research where questions raised by field foresters were answered in a manner having field application. The objective of this paper is to present three examples of disease management questions posed to us by forest managers, our approach to investigating each question, and the answers we found. A fourth example involved the use of data collected originally to evaluate black stain root disease to evaluate fire-caused-mortality of ponderosa pine. In some cases additional conclusions from the studies are provided. The reader is encouraged to examine the four papers relating to the four questions.

Researchable Questions

1. Can either removal of stumps or fertilization with nitrogen or both be used to manage laminated root rot?

*Phellinus weirii* (Murr.) Gilb. causes laminated root rot (LRR), a major disease affecting growth and survival of *Pseudotsuga menziesii* (Mirb.) Franco (Douglas-fir) and other commercially important conifer species throughout the Pacific Northwest, and is responsible for large annual losses in stand productivity. The biology, distribution, and impact of the disease, relative susceptibility of host species, and options for management are summarized elsewhere (Thies and Sturrock, 1995).

In 1975 forest managers concerned about losses from LRR were actively seeking disease management strategies. Earlier research indicated that two areas showed some promise. In 1968, Larry Weir established a nonreplicated study (Skimikin plots) near Salmon Arm, B.C., to test the feasibility of control of LRR through mechanical removal (scarification) of the larger sources of inoculum in the soil, and the planting of mixed susceptible and resistant species (Weir and Johnson 1970, Morrison and others, 1988). Earl Nelson did pioneering work on nitrogen (N) fertilization with lab, greenhouse, and field tests to show that N fertilization reduced survival of *P. weirii* in buried wood (Nelson 1970).

Building on this earlier work we began a study to assess stump removal or fertilization, or both as strategies for reducing the occurrence of LRR in replacement stands on infested sites. To allow for a regional area of inference, the study was repeated using the same design in five predominantly Douglas-fir stands in Oregon and Washington. Each stand was a complete study with identical treatments but differing in the number of replicate plots, metrics of the preceding stand, amount of LRR inoculum, and the year of occurrence of various activities. Because the studies were identical in plan, they were presented together to show region-wide similarities and allow readers to draw conclusions on the apparent occurrence and impact of LRR in the replacement stands and the growth of Douglas-fir regeneration 23 to 27 years after treatment (Thies and Westlind, in press).

The stands were surveyed preharvest and postharvest (clearcut). Each LRR-infected tree (i.e. represented as standing, dead, or down trees and stumps) was marked, and the location of its center was mapped. In each clearcut, a 2 × 4 set of factorial treatments of stump removal in combination with N fertilization was applied to 0.04-ha circular plots and replicated five to seven times. Treatments, applied in the fall,
the fall, included stump removal with a bulldozer (either all stumps removed or the plot left undisturbed) and broadcast fertilization with ammonium nitrate (0, 336, 672, or 1345 kg N ha⁻¹). Douglas-fir seedlings were planted in the winter following treatment application. When the stand was considered established, each plot was thinned to about 2.4 m inter-tree spacing and the trees tagged. Diameter at breast height, total height, and mortality of trees were recorded every 2 to 5 years. A total of 7,827 tagged trees on 239 plots were observed for up to 27 growing seasons following treatment.

2. Can removal of stumps or fertilization with nitrogen be used to manage laminated root rot?

Answer: Yes and No. Stump removal resulted in a 74% reduction in the rate of LRR-caused mortality in the replacement stand while N fertilization had no effect on LRR-caused mortality. (Thies and Westlind, in press)

Management implications: Managers can consider stump removal as a strategy for the management of LRR. A decision to fertilize with N can be made independent of LRR-management considerations.

Additional conclusions beyond the initial question: (Thies and Westlind, in press):

1) N fertilization applied immediately after planting increased growth of Douglas-fir seedlings but only in some stands.

2) N fertilizer can have a long, positive residual effect on Douglas-fir growth.

3) Effects on soil of the heavy equipment used in stumping operations did not reduce subsequent Douglas-fir survival or growth.

4) On plots where stumps were not removed there was 24.8% less wood volume at the end of the study in areas where LRR was present compared to areas where it was not present.

5) Stump removal from plots with LRR increased volume on these plots by 25.4%.

6) A relative inoculum index (INOC) was calculated for each infected tree based on its stump and root biomass (Thies and Cunningham, 1996) and the tree’s condition. Neither the INOC nor a count of P. weirii-infected stumps in a parent stand, were of value in predicting LRR-caused mortality of Douglas-fir in a replacement stand.

3. Can fertilization with potassium be used to reduce losses due to laminated root rot?

Inoculum reduction through stump removal, as above, is one strategy for reducing the impact of LRR; however, that approach can be both disruptive and expensive. An alternative strategy would be to help seedlings, or trees, be more resistant to disease by manipulating their nutrition. In 1980 the Intermountain Forest Tree Nutrition Cooperative (IFTNC) was organized at the University of Idaho with initial efforts concentrated on studying the effect of N fertilization on growth and survival of Douglas-fir. The efforts expanded to include the role of potassium (K) and other specific elements in tree nutrition. It was noted that although N fertilization usually increased growth of Douglas-fir, sometimes there was an increase in tree mortality that corresponded to the treated areas (Mika and others, 1992) and it was found that foliar K status corresponded to the mortality patterns (Mika and Moore 1990). Some phenolics and lignin in root bark may be a natural defense mechanism of the trees and these compounds are associated with lower levels of Armillaria root disease in Douglas-fir inoculation trials (Entry and others, 1991a, 1991b, 1992). Mandzak and Moore (1994) provide a rational for the role of forest nutrition in the general health of western forests.

Given the promising results at reducing Armillaria root disease by increasing K, local foresters in western Washington began considering the use of potassium fertilization to alter the root chemistry of Douglas-fir to make them more resistant to LRR. We were asked for advice and agreed to test the hypothesis that the application of K as KCl to a planting site will change the incidence of Douglas-fir seedling mortality caused by LRR.

The study was established on one 52-ha clearcut near Morton Washington. Each plot was established around the stump of a LRR-infected Douglas-fir tree that was standing-dead prior to logging. Logging slash and brush was cleared from around the stump to create a 0.02-ha treatment plot. A total of 44 plots were created. Each stump within the plot was evaluated for the presence of LRR and a relative inoculum index (INOC) was calculated based on the calculated stump and root biomass (Thies and Cunningham, 1996) and the stain observed on the stump top (Thies and others, in preparation). The INOC of each stump on a plot was aggregated to determine a plot’s total INOC. Plots were stratified based on total INOC into 11 replicate blocks of four
plots each. Four fertilization treatments (no fertilizer applied; 224 kg K . ha-1; 448 kg K . ha-1; 224 kg K . ha-1 + 224 kg N . ha-1) were randomly assigned within each block of four plots. The fertilizer was uniformly broadcast using a cyclone-type seed spreader. Each plot consisted of a measurement zone extending 3.05 m from plot center, and a buffer zone (extending from 3.05 to 8.02 m from plot center) and received one treatment. Seedlings to be monitored were planted within the measurement zone. On each plot, a total of 50 Douglas-fir seedlings (2-1 bare-root) from a local seed source were planted on each measurement zone (intertree spacing about 0.8 m apart; 17,000 seedlings . ha-1) and additional seedlings were planted within the buffer zone (intertree spacing about 2.1 m; 2200 seedlings ha-1). Planted seedlings in the measurement plots were distinguished by a wire flag placed by each. Seedlings were planted in January 1999 under cool wet conditions, and treatments were applied in February 1999.

A total of 2200 seedlings were planted on the measurement plots and followed through 2005. Needle samples were collected and analyzed in fall 1999 and 2001, and root tissue samples were collected and analyzed in 1999. Some treatments caused a significant increase in N and K in the needles in 1999 but not in 2001. The K+N treated seedlings had a higher phenolic/sugar ratio than all other treatments. There was no significant difference in either mean seedling height or proportion of LRR-caused seedling mortality as a result of K or K+N fertilization.

Can fertilization with potassium be used to reduce losses due to laminated root rot? No, at least not on this study area. (Thies and others, in preparation).

Management implications: Fertilization with K will not be a tool for management of LRR.

4. Does the season of a prescribed burn influence the amount of black stain root disease-caused mortality in a ponderosa pine stand?

Since the mid-1980s, USDA Forest Service, Pacific Northwest Region (R6) pathologists, in particular Craig Schmitt and Don Goheen, have been calling attention to black-stain root disease (BSRD) of ponderosa pine (Pinus ponderosa Dougl. ex Laws) caused by the fungus Leptographium wageneri var ponderosum (Harrington and Cobb) in stands east of the crest of the Cascade Range in Oregon. Since initial identification of BSRD at Emigrant Creek Ranger District, Malheur National Forest, in 1989, district personnel have been mapping locations of BSRD. BSRD and other root diseases concern resource managers because of the broad effects of tree mortality: a) on wildlife, especially related to big game winter range and hiding cover; b) on high value stands managed for fiber production on a reduced land base; and c) on increased fuel loading and risk of wildfire. As more BSRD was located, managers questioned how their management practices may have affected the spread and impact of the disease. They were concerned that the increased use of prescribed burning in spring may create stressed trees and make the stands more attractive to potential insect vectors of the fungus which would likely be active in early to mid-summer. The managers in eastern Oregon have two relatively short windows of opportunity for prescribed burns: late spring and early fall. To pick the most viable option, managers needed to know how the seasonal timing of prescribed burning will affect the future development of BSRD in the stand.

A study of the seasonal effects of prescribed burn on tree mortality was established in mixed-age ponderosa pine at the south end of the Blue Mountains near Burns, Oregon. Each of six previously thinned stands was subdivided into three 12-ha experimental units and one of three treatments was randomly assigned to each: fall 1997 burn, spring 1998 burn, and no burning (control). Burns were representative of operational prescribed burns, given weather and fuel conditions. Trees within six 0.2-ha circular plots on each experimental unit were observed for four post-burn growing seasons to determine fire damage and to detect immediate and delayed mortality and occurrence of BSRD. There were 5321 tagged ponderosa pines alive at the time of the burns that were observed for four growing seasons. There is no evidence that the median proportion of trees that died with BSRD was affected by treatment.

Does the season of a prescribed burn influence the amount of black stain root disease-caused mortality in a ponderosa pine stand? No

Management implications: A decision to burn in a particular season can be made without concern for influencing BSRD.

Additional conclusions beyond the original question (Thies and others, 2005):

1) Distribution of BSRD is wide-spread but doing little damage in the six study stands with about 23%
of the trees infected but an annual mortality rate of only 0.5% of infected trees.

2) Needles clustered at the ends of branches with a lion’s tail appearance, a purported symptom of BSRD, was not a reliable predictor of BSRD incidence.

3) The percentage of ponderosa pine mortality was higher after fall prescribed burns than after those in spring.

4) Season of prescribed burn had an effect on survival of ponderosa pine as it influenced fire intensity and burn severity.

5. Bonus question: Can we predict the probability of mortality in fire-damaged ponderosa pine following prescribed fires in eastern Oregon?

While establishing the season of burn study described above we asked forest managers from eastern Oregon for guidance on predicting which trees would die as a result of the prescribed burns. We wished to avoid collecting data on trees that would die because of the fire rather than from BSRD. It became clear that managers needed information on characteristics of trees that would experience delayed mortality. We established a set of tree-burn damage variables that we believed might foretell future susceptibility of the trees to insect or disease attack. Because pest damage in these stands was minimal we were able to use the fire damage variables collected immediately after burning to develop predictive models for delayed fire-caused mortality. Managers would like to evaluate the immediate and predictable delayed mortality to quickly determine how successful the burning prescription was at achieving such management objectives as post-fire stocking level, improving future prescriptions, and better planning additional activities. Post-fire predictions need to be based on easily observable morphological and burn-damage characteristics.

Fire damage data was collected from 3415 trees in stands burned in either fall 1997 or spring 1998. These trees were observed for mortality through fall 2001 (four growing seasons). Tree mortality represents a binary categorical response variable. Trees alive before the treatment burns were applied were coded as alive (0) or dead (1) in 2001. Logistic regression analysis was used to investigate how this categorical response variable (mortality through 2001) was associated with a set of explanatory variables.

Two models have been developed for predicting the delayed mortality of ponderosa pine after a prescribed fire conducted in either fall or spring. Either the full model with five measured variables (live crown proportion, needle scorch proportion, bud kill proportion, basal char severity, and bole scorch proportion) or the reduced model with two measured variables (needle scorch proportion and bole scorch proportion) may prove useful for projecting delayed mortality in stands that have received a prescribed burn. These models will be helpful to managers planning post-burn operations (such as salvage or planting) and to ecologists interested in the role of prescribed fire in determining the structure of forest communities.

We found that ponderosa pine mortality from similar prescribed burns was higher after fall burns than after spring burns. We concluded that this was because dryer fuels and burn conditions caused the fall burns to be somewhat more intense even though the plan was to keep the fire intensity low. We concluded that the mortality is related to the damage inflicted on each tree rather than the season as it relates to the physiological state of the trees. Care should be exercised in extrapolating results and using these models beyond the geographical area of the sampled stands or to species other than ponderosa pine until additional data sets are available to validate the models for other areas or species.

Can we predict the probability of mortality of fire-damaged ponderosa pine following prescribed fires in eastern Oregon? Yes. (Thies and others, in press)

Management implications: Managers now have a tool to use soon after a burn to evaluate the likelihood that a given tree will die as a result of a burn. This information can be used during a ground survey to gather information to evaluate the stand level impact of a burn, or to mark trees for salvage if that is the management objective.

References


Forest Pest Surveys: Changing Forestry in Manitoba

F.A. Baker, K. Knowles, Y. Beaubien and I. Pines

Abstract--A series of pest damage surveys have been implemented in Manitoba’s forests. Results of these surveys demonstrated the need to consider insect and disease damage in determination of regeneration success and forest establishment. Damage surveys are now done by timber companies on all regenerated stands, and in stands that are planned for harvest. When appropriate, Forest Health specialists follow up with specific pest surveys, and develop management prescription to minimize pest damage. The end result is that all stands harvested since 1985 are – or will be – fully stocked with damage free trees. Addressing insect and disease problems prior to harvesting allows forest managers to avoid or greatly reduce the effort needed to minimize insect and disease damage in regenerating forests.

Introduction_________________

The forest economy in Manitoba accounts for more than $418 million and employs more than 9,000 people in this province of 1.1 million people. There are more than 14 million ha of forest land, 94% of which is owned by the province, 1% by the federal government, and 5% privately owned. These forests are dominated by jack pine (Pinus banksiana), white and black spruces (Picea glauca and P. mariana) and trembling aspen (Populus tremuloides), and are used primarily for newsprint, paper, paperboard, softwood lumber and waferboard. Only 25% of the annual increment available is harvested. Most of the unharvested timber is inaccessible. $2.7 million m3 of timber is lost to insects and disease annually. This timber would contribute $498 million to the gross provincial product. Restoring or preventing this loss in accessible areas will increase the timber available for harvest.


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mortality or growth loss were considered. Between 1975 and 1992, 118, 500 ha of forest were regenerated. The most common causes of damage are illustrated in Figure 1. Insect damage varied between years, as one might expect. Dwarf mistletoe and Armillaria root disease seemed relatively constant over time. The important thing to note, however, is the large proportion of stems that were rendered non-merchantable at relatively early ages. Some stands had more than 50% defective stems. If the trend for damage does not decrease significantly, many of these stands will be out of production. In 1996 budget problems could have eliminated the renewed forest pest survey, but, data collection was integrated with the regeneration survey. Survey crews focused on damage, not on the causal agent. While some detail on pest information was lost, this integration not only preserved the survey, but it enabled examining all stands, rather than the sample used in the renewed forest pest survey. Forest regeneration surveys were originally done by Manitoba Conservation, but now they are done by company crews trained by Manitoba Conservation.

Dwarf mistletoe and browse damage were the most commonly observed problems during 1997-2001 (Figure 2). During this time of budget constraints, silviculturists were developing a new survey called the “free-to-grow” survey. This survey examines a stand 14 yr after regeneration, to ensure that crop trees are free from brush competition and well spaced.

Figure 1. Damage caused by various agents in regenerated stands.

![Figure 1](image1)

Figure 2. Major damages observed in 146 Forest Regeneration Surveys during 1995-2003 (>5216ha).

Pest impact collection was included from the start. Again, the focus was on damage rather than the causal agent. Armillaria root disease and dwarf mistletoe were the most common damaging agents in 14 year old stands (Figure 3).
Figure 3. Major damages observed in 147 Free to Grow surveys during 1996-2003 on >4900 ha.

As GPS technology became more affordable, crews began to record transect and plot locations, as well as the location of any obvious damage which occurred outside of plots. These surveys generated massive amounts of data. It was obvious from the start that detailed data analysis was not likely to happen, or even needed, for most sites. Forest Health staff needed a way to quickly identify stands with potential problems. To this end, they developed a simple block summary sheet on which crews estimated severity of important damages on the surveyed block (Figure 4). If damages were recorded, this information, along with plot data sheets and GPS coordinates, was forwarded to Forest Health. Forest Health staff then examined these data and decided whether Forest Health follow-up was needed. Follow-up surveys are selected from regeneration, free-to-grow surveys for maximum efficiency and to return in the same field season when the same damage is evident. Data collected on follow-up surveys is returned to the company, along with a pest management prescription, if necessary. Remember, a stand must meet free-to-grow specifications before a company can return management responsibilities for that stand to the province. If dwarf mistletoe is identified in a Free-to-grow survey, the stand will not be certified until the disease is removed from the site.

Map & Record Dwarf Mistletoe Pockets

DM infections on residual Jack Pine *

*host very important

Severity Levels:
Nil = “-” No pest occurrence.
Low = “L” 1 to 5 stems affected; rarely or infrequently seen in stand.
Moderate = “M” 6 to 20 stems affected; occasionally seen; small openings occurring in stand.
Severe = “S” >21 stems affected; frequently seen; large openings throughout stand.

Figure 4. Block summary header with guidelines for estimating damage severity.

The regeneration and free-to-grow surveys created an awareness of the damage that insects and diseases cause in regenerating forests. The damage caused by many insects and diseases in regenerating stands could be reduced or eliminated if managers were
aware of insect and disease presence on the site prior to harvesting. With this in mind, in 1998, the timber companies requested that pest impact information be collected in pre-harvest ecological assessments. Stem cankers and Armillaria root disease were among the most damaging agents found in mature stands (Figure 5).

![Figure 5. Major damages observed in 577 Pre-Harvest Ecological Assessments during 1998-2003 on >57,600 ha.](image)

**The Successes**

So, what are the successes? There are many. First, all stands are now regenerated with sufficient undamaged trees. Originally, it was assumed that once stands met regeneration criteria based on the number of live trees, they would be fully stocked at rotation age. With some stands having more than 50% of their stems rendered non-merchantable due to damage at an early age, that assumption is clearly not valid. Identifying impacted stands and requiring the timber companies to mitigate the damage ensures that the stand has the opportunity to attain maximum productivity.

A second success is that Forest Health staff found a way to continue collecting information that ensured that stands were regenerated, even during a budget crisis. This was done by incorporating pest damage collection into existing silvicultural surveys. Detailed information on damage cause is not available because crews lack the necessary experience to collect it, but for the vast majority of stands, sending skilled people is unnecessary. Unnecessary detail was compromised to gain knowledge of damage. In addition, Forest Health staff could visit only a sample of regenerated stands, but silviculture survey crews visit ALL regenerated stands.

A third and related success is that timber companies are actually collecting data on pest damage. Not only does this reduce the cost to Forest Health, but the companies actually believe the data that they collect. They take ownership in those data, and if stands have a problem, they are much more in tune with committing to an appropriate management prescription to restore the stand productivity. Evidence for this is the fact that the timber companies requested that Forest Health find a way to incorporate collecting data on pest problems into pre-harvest ecological assessments, when potential pest problems may be more easily prevented. Pest management is now considered in all stand management.

**Building On This Success**

The data collected will continue to provide benefits. Stand survey data are managed in a GIS, and become a part of the record for that stand. Over time, Forest Health staff can analyze these data and gain a better understanding of insect and disease problems which “carry over” from one stand into the next. They can evaluate prescriptions. With sufficient time and data, Forest Health specialists can begin to query the data for answers to questions about which pest where? When? Why? Understanding insect and disease impacts will lead to improved timber supply forecasts and appropriate harvest levels. This last point is particularly important to timber companies, which are facing a timber supply which is decreasing and becoming available at greater distances from mills.

Despite these successes, considerable work remains. Currently, there is little information available about insect and disease damage in stands between the age of 14 (free-to-grow) and rotation age, a period of 60 or more years. And even when forest managers are aware of pest problems, many management prescriptions are based on intuition rather than experimentation. Better information is needed to identify and improve effective prescriptions.

This paper is based on a manuscript in preparation for Plant Disease.
Introduction

The review and reflection on past management decisions is the central theme of this panel session. My talk concerns the decision to develop and implement IPM prescriptions on the Doak Mountain, Klamath Falls eagle habitat during the severe drought period from 1987-1994. These prescriptions were developed by an interdisciplinary team of scientists and foresters to address the threat of fire, insect and drought on one of the most productive eagle habitats in Oregon State.

In keeping with the panel theme, a revisit was made in early June of 2005 to understand to longer term impacts of these management decisions. For the panel discussion purpose, the situation of the forest in 1992 and current conditions in 2005 will be used to judge the “success” or “failure” of this operation.

The Resource

The Doak Mt. eagle reserve is situated 10 km north of Klamath Falls, Oregon. It encompasses some 4,000 ha at 1000-2600 m elevation. The complex includes some 57 active nest sites and 50 additional nest locations; part of roughly 20% of the bald eagle population of Oregon.

The area was set aside by Weyerhaeuser Company in 1967 as eagle habitat (11 years before the bald eagle was listed as threatened under the Endangered Species Act).

Eagle productivity (occupancy and chicks fledged) of individual nest sites have been under continuous survey since 1971 by Weyerhaeuser and Oregon Department of Fish and Wildlife (Figure 1).

Figure 1. Results of long-term eagle monitoring at Doak Mt. Klamath Falls (cited from Doak Mt. Management Plan for Forest Health and Eagle Habitat September 1992, Weyerhaeuser Company Publication).

Indicators of Environmental Change

Drought conditions in Klamath County began to impact stand health beginning in 1987 with the widespread mortality of Abies concolor (white fir) caused by Scolytus ventralis (fir engraver). As the drought intensified in Klamath County, our investigations showed that A. concolor was uniformly susceptible to S. ventralis attack, and that tree vigor was of little benefit. Concurrently, we observed a rise in pests such as Ips pini (pine engraver) with an increased threat of post-PCT-thinning residual Pinus contorta (lodgepole pine) mortality. Mortality caused by root-rot fungi also increased around the region. In 1992, the situation became more critical with the observation of a large Dendroctonus ponderosae (mountain pine beetle) population entering the Doak Mt. forest complex. Multiple group tree kills of Pinus ponderosa (Ponderosa pine) and Pinus...
lambertiana (sugar pine) was occurring in many areas surrounding Klamath Lake.

The final level of threat became fire. Long controlled in this forest, with at least 60+ years since the last recorded ground fire. These previous natural fires characteristically burn slowly along the ground, and remove non-fire resistant trees and shrubs, mistletoe infected trees and otherwise thin and maintain forest health. Absence of fire, ongoing mortality and minimal harvest activities for several decades had created a situation where a fire ignition at Doak Mt. would in all likelihood lead to a stand replacement fire with catastrophic consequences to the eagle habitat.

Silvicultural Plan

A forest management plan was initiated in spring 1992 to address multiple pest and stand risk factors. The Management Plan Team consisted of the following disciplines:
- Forest Manager
- Wildlife biologist
- Cartography
- Communications
- Research Forester
- Silviculture/Biometrician
- Forest Pathology/Entomology

Management Plan Objectives:
- Promote a continuous supply of live trees suitable to nesting eagles.
- Maintain future forest health and eagle productivity through timely and specific silvicultural intervention.
- Incorporate other resource goals into the plan:
  - Snags for white-headed and pileated woodpeckers.
  - Deer habitat and cover.
  - Western pond turtle habitat.
  - Nest and rookery trees for Blue Heron.
  - Aesthetics of multistoried stands.
  - Protection of significant wetlands.
  - Protection of Class-1 waters.
  - Recreational opportunities.

Management prescriptions consisted of three primary forestry operations objectives:

- **Drought Susceptibility**- preferentially remove drought susceptible species, reduce stocking levels, address steep south and west facing slopes with higher inherent stress, and remove competing vegetation immediately around eagle nest trees (see Barrett et al 1983).

- **Fire-Fuels Hazard Reduction**- remove “fuel-ladders” created by mistletoe infections, remove mortality and slash debris from around nest trees (see Agee 1981).

- **Pine Beetle Hazard**- risk rate stand components on pine basal area and appropriately remove trees to reduce pine beetle hazard, select non-host residual nest trees to serve as replacement nest trees, and harvest and remove “green-tree” mortality and other potential sources of MPB brood in a timely manner (see Littke and Gara 1986 and Gara et al 1984).

Communication was also critical to build Klamath community support for action.

Harvest activities commenced in 1993 through 1995 during periods of eagle nest absence including:
- 3000 ha addressed in forestry operations.
- Eagle surveys continue through the interim period.
- 57 nest locations retained along with 5,040 recruitment trees.
- Drought eventually subsides after a 7-year period.
- Fire eventually visits the Doak Mt. complex.

Current Situation (2005)

Arnett et al (2001) published an updated review of impact of stand management at Doak Mt. on eagle habitat. They concluded that eagle territory occupancy and productivity was maintained by previous careful, planned forest management.

In June of 2005, a review of the management sites revealed the following:

(1) Eagle productivity is higher than that observed in the previous surveys beginning in 1971.
Areas of low drought stress and considered low management risk priority fared the drought with little loss of forest cover.

Re-growth in severe drought prone areas (Squaw Point) from pine regeneration planted in 1993 and 1994 is excellent.

“Retention future nest trees” marked during forestry operations showed good vigor; three such trees have new eagle nests with one or more eagle chicks.

Incense cedar in retention areas suffered greater mortality than was expected during the later part of the drought.

Lightning caused fire burned areas outside of the management zone in 1994. A partial cut and removal area adjacent to Wacus Marsh was back-burned and suffered minimal fire caused mortality. A non-managed area west of Highway 140 was consumed in a stand replacement type fire.

The drought began to subside in late 1994 and climatic conditions returned to some level of normalcy by 1997. Today, the management area shows excellent vigor in foliar retention and leader growth. Bark beetle activity is at low endemic levels, with very scattered single tree mortality. The potential for catastrophic fire is currently low, but building in some areas as standing dead and other fuel sources rebuild.

Questions for Discussion_____

When is the time to act?

Answer: No one can foresee the future with respect to climate variation. In particular how long and severe will the drought be? The assumption that immediate action was needed determined the urgency of the plan review and addressing stand sections according the risk threat potential. The urgency for action was underscored by MPB killing several large sugar pine eagle nest trees.

What team composition is best?

Answer: In a short response mode, representation of all science disciplines is best to pull together a comprehensive risk management program. Equally important is the communication representative who must sell the implementation plan to the public at large.

How do you proceed without all the facts?

Answer: Management of multiple resources goals under the constraint of worsening stand conditions had to proceed even though some of the methods had not been yet tested on an operational scale. The diversity of the team allowed for modeling of stand attributes with a view towards host and pest biology. Some things, such as treating > 14 inch diameter cut-stumps with Borax in the immediate vicinity of eagle nest trees were simply done (Kleijunas 1989). Similarly, immediate removal of green MPB-infested and other potential brood material makes sense from a bark beetle control point of view. Wholesale removal of mistletoe infected white fir posing a high risk for carrying a ground fire into the canopy was also carried out.

Who determines what constitutes a success story?

Answer: The most rewarding conformation of success is through independent collaboration such as the publication on post-management eagle productivity (Arnet et al 2001). Another is the realization that the other management goals also seem to be have been, and are being met.

Unfortunately, the intervention was only a temporary success, given the dynamic nature of forest systems. Growth and in-growth has started the ball rolling towards another site crisis at Doak Mt. in the next decades. At some future point in time, the decision will again be made to act.

References__________________


Photos by Shaw
Magnesium Chloride - Based Dust Suppression Products and the Health of Roadside Vegetation and Riparian Systems

Betsy A. Goodrich, William R. Jacobi, and Ronda D. Koski

Abstract—In addition to its use for snow and ice control, magnesium chloride (MgCl₂) is applied to gravel roads during summer months for dust suppression and road stabilization. Dust suppressants are used to control maintenance costs, erosion, and fugitive dust. The use of dust suppressants is increasing in order to control particulates in the interest of air quality. Research quantifying the impacts of MgCl₂ on vegetation is limited. This project investigates tree, woody shrub, and herbaceous ground cover health along gravel roads in Larimer and Grand Counties, Colorado. Objectives of this study are to determine if MgCl₂ is moving from treated gravel roads into roadside systems, and if it causes adverse effects to roadside vegetation, soils, or riparian systems.

In 2004 and 2005, roadside and drainage vegetation health plots were established on 19 gravel roads treated and untreated with MgCl₂ in Larimer and Grand Counties. Trees, woody shrubs, and herbaceous ground species were rated for health and assessed for abiotic and biotic damages. In all plots, soil, foliar, and twig samples were collected and analyzed for chemical content. Water samples were collected upstream and downstream from 17 stream sampling sites along 14 treated roads in both counties. Water was collected bi-weekly from May through October.

Preliminary analysis (averaging over county, plot, and road) showed the chloride component of MgCl₂ moving from roads approximately 10–20 feet. Aspen, lodgepole pine, and Engelmann spruce trees appeared to be taking up and accumulating higher levels of chloride downslope from the road up to 30 feet away. Chloride accumulation in leaves can cause osmotic stress, leading to dehydration injury (i.e. leaf burn) typical of drought. Preliminary analysis showed a weak but significant correlation between foliar chloride and leaf burn symptoms in our aspen plots. Stronger correlations existed between foliar chloride and lodgepole pine tip burn as well as foliar chloride and Engelmann spruce necrotic foliage. Preliminary analysis of water samples showed significant differences in chloride concentrations due to stream position and time of year. Chloride values were significantly higher downstream from a treated road than upstream from the road.

In continuing analysis, all county, plot and road data will be separated to determine impacts of site differences on the movement of MgCl₂. Data collected from drainage plots in 2005 will be incorporated into project results. A controlled greenhouse study is in progress this year to determine the impacts of MgCl₂ on common roadside vegetation without potential interactions with other environmental factors. In our completed analysis, we will determine if MgCl₂ moves from roads into roadside systems, if there are detrimental effects to soils, vegetation, or water, and if there are needs to implement better management practices in application or maintenance procedures.
Blister Rust Canker Growth and Morphology: Can Canker Category Predict Tree Mortality in Three North Idaho Rust-Resistant Western White Pine Plantations?

Amy I. Eckert

Abstract - This research will help answer the question: Can external canker characteristics be used to predict tree mortality in white pine blister rust resistant western white pine plantations in North Idaho? Forest health surveys in Northern Idaho recently found that rust resistant western white pines within the same 20 year old plantations expressed stem cankers with very different characteristics. White pine blister rust stem cankers usually girdle and kill infected natural western white pine quickly. If, however, some rust resistant western white pines exhibit slower canker growth rates, it is possible that trees will survive much longer than expected, possibly long enough to meet stand management objectives. This study was initiated to elucidate the relationships between visual canker characteristics and canker growth rates on rust resistant western white pine. This project will create a stem canker classification system that will place stem cankers on resistant western white pine into three distinct growth categories based on external, visual characteristics. In 2004 and 2005, over 150 blister rust stem cankers were collected from three plantations in Northern Idaho. The cankers were cross-sectioned at the point of infection and analyzed using spatial analysis software. Statistical analyses were performed to relate canker classification with circumferential canker expansion and time. Preliminary trends show a correlation between canker category and canker growth rate. Future work will include examining host-pathogen interactions using microtechnique and performing genetic analyses to confirm the presence of pathogen DNA.


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Abstract—The ecology and potential function of two Douglas-fir and ponderosa pine woody root fungal endophytes, *Byssochlamys nivea* and *Umbelopsis* spp., were investigated at three study sites in eastern Washington. *Byssochlamys nivea* was found in the soil rhizosphere, while *Umbelopsis* spp. was not detected. The optimum growth temperature for both *B. nivea* and *Umbelopsis* spp. in vitro was 25º C. *Byssochlamys nivea* grew at higher temperatures than *Umbelopsis* spp., while *Umbelopsis* spp. grew better at lower temperatures than *B. nivea*. To investigate the potential function of these endophytes, *Byssochlamys nivea* was grown in culture with *Armillaria ostoyae*. *Byssochlamys nivea* did not inhibit the growth of *A. ostoyae* and no changes in hyphal morphology were observed.

Introduction

Fungal endophytes are fungi inhabiting, but not inciting disease within the leaves, stems, and roots of healthy hosts. They have been found in nearly every plant tissue and species examined. Hoff et al. (2004) found 27 different fungal genera in woody roots of asymptomatic Douglas-fir and ponderosa pine trees in eastern Washington. Little is known about these fungal endophytes, however. Our study was initiated to examine the biology, ecology, and function of two common endophytic genera (*Byssochlamys* and *Umbelopsis*) found in the woody roots of Douglas-fir and ponderosa pine in the dry, fire prone, forests of eastern Washington by Hoff et al. (2004).

The objectives were to determine:

- The distribution of the fungal endophytes in the rhizosphere soils around woody roots of Douglas-fir and ponderosa pine.
- The *in vitro* minimum, maximum, and optimum growth temperatures of the fungal endophytes.
- Whether *Byssochlamys nivea* is antagonistic *in vitro* against the pathogenic species *Armillaria ostoyae*.

Materials and Methods

Study area

The three study sites were within the Mission Creek watershed, a tributary of the Wenatchee River, which is located on the east side of the Cascade Mountain range, in central Washington state, USA (47°25’N, 120°50’W) (Dolan 2002). The Wenatchee National Forest climate is strongly influenced by the Pacific Ocean and geography provided by the Cascade Mountains and prevailing westerly winds, with most of the precipitation occurring in late fall and winter (USDA 1990). Annual precipitation ranges from 25-100 cm, with the majority occurring between October and April as snow (USDA 1990, Lillybridge et al. 1995). Hot, dry summers are typical of the eastern Cascades, with average maximum temperatures of 29.5º C occurring between June and August (WRCC 2004).

The vegetation of the area is typical of eastern Cascade dry forests, dominated by Douglas-fir and ponderosa pine. Soils are sandy and well-drained, developing on non-glaciated Tyee sandstone with limited amounts of shale and conglomerate arising from the Swauk and Chumstick formations (Tabor et al. 1982, Soil Survey Staff 1995).

Field sampling

Trees that were positively identified as having *Byssochlamys* or *Umbelopsis* spp. present by Hoff et al. (2004) were mapped on each site (US Forest
Service, Wenatchee, WA). One positive tree from each site was used to establish the plot for this investigation. All selected trees (40 at each site – 120 total) were within a 75 m² area at each site and were next to or nearby the previously sampled trees. Forty trees were randomly selected in the three study sites for rhizosphere samples. Douglas-fir and ponderosa pine trees were equally sampled over the three sites.

A major lateral tree root was excavated from the bole out to 35 cm. A 10 g soil sample was collected from the rhizosphere within 3 cm of the root, 10 and 30 cm away from the bole. Eight samples were taken near saplings, and in these cases, only one soil sample was collected. A total of 232 soil samples were collected.

Lab Analyses

**Fungi in Soils**

*Byssochlamys nivea* presence in the rhizosphere soil was determined by placing 2-3 g of non-dried soil into a sterilized 50 mL screw top glass tube. Twenty mL of sterilized de-ionized water were added to each tube, shaken for 1 min, and then heated in a 75º C water bath for 30 min. (Beuchat and Pitt 2002). After heating, each sample was shaken for 1 min, then equally distributed into three 100 x 15 mm petri plates, and swirled with 2% MEA (Malt Extract Agar).

*Umbelopsis* spp. presence in the rhizosphere soil was determined using a soil dilution plate method. A final dilution of 1g:100,000 mL, soil to water was prepared, then one mL of the dilution was equally divided and plated onto two 100 x 15 mm petri plates with 2% MEA then swirled. Ten samples from each study site were randomly selected, for a total of 30 samples across the three study sites.

All samples were incubated at 25º C in the dark for one month. Plates were then examined under a stereo microscope for the presence of *B. nivea* and *Umbelopsis* spp. Identifications were based on morphological characters.

**Growth Temperatures**

The minimum, maximum, and optimal temperatures for growth of the *Byssochlamys* and *Umbelopsis* spp. isolated by Hoff et al. (2004) were determined. One *Byssochlamys* isolate and three *Umbelopsis* isolates (one *Umbelopsis versiformis* Amos & HL Barnett and two *Umbelopsis vinacea* (Dixon-Stew.) Arx) were used. Ten replicates of each isolate were grown in 60 x 15 mm petri plates with 2% MEA. Each of the ten replicates was grown in the dark at 3, 10, 15, 20, 25, 30, and 35º C. The radial growth, in mm, of each isolate was recorded after ten days. Growth rates were expressed as mm/day.

**Antagonisms**

Fungal antagonism was investigated using one *B. nivea* isolate and three *A. ostoyae* isolates. Both species were collected from eastern Washington in previous studies. Five replicates of each of the three different *Byssochlamys-Armillaria* growth pairings were made, as well as five *Armillaria* controls. A section of *A. ostoyae* was aseptically transferred to the middle half of a 100 x 15 mm petri dish with 2% MEA, then were placed in a dark, 25º C incubator for three weeks. After three weeks, *Byssochlamys* sections were placed in the middle of the other half of the petri dishes. Control plates were prepared by growing *A. ostoyae* by itself.

Hyphal interactions among *B. nivea* and *A. ostoyae* isolates were examined using a glass microscope slide method (Riddell 1950). *Byssochlamys* was placed on one side (right) of the coverslip and *Armillaria* was placed on the other side (left) of the cover slip. Each slide was then placed into an empty petri dish and incubated at 25º C in the dark for 14 days. The same three *A. ostoyae* and one *Byssochlamys* isolates were used in the microscope slide pairings. All fungal growth pairings were examined for morphological characters, with close attention directed towards any changes on hyphal morphology, at magnification ranging from 3X to 400X.

**Results**

*Byssochlamys nivea* was isolated from the rhizosphere from 52 percent of the samples, but *Umbelopsis* spp. was not detected.

The optimum growth of *B. nivea*, *U. versiformis*, and *U. vinacea* all occurred at 25º C (Figure 1).

Radial growth of endophytic species differed significantly as a function of temperature (F (2,28) = 11.374; p = 0.000), however, post-hoc Tukey’s HSD tests showed that *U. versiformis* grew significantly more at 25º C than *U. vinacea* and *B. nivea* (p ≤ 0.05). The minimum temperature for growth of all isolates was 3º C. The radial growth of endophytic species at this temperature differed significantly (F (2,37) = 34.166; p = 0.000), with post-hoc Tukey’s HSD tests showing *B. nivea* growing significantly less than *Umbelopsis* spp. (p ≤ 0.05).
Fungal growth at 10º C, 15º C, and 20º C was significantly different among species (F (2,32) = 371.075; p = 0.000, F (2,34) = 221.195; p = 0.000, and F (2,34) = 16.487; p = 0.000, respectively), with post-hoc Tukey’s HSD tests showing *Umbelopsis* spp. growing at a significantly higher rate than *B. nivea* at 10º C and 15º C.

![Graph: Growth rates of Umbelopsis and Byssochlamys isolates at differing temperatures.](image)

**Figure 1**—Growth rates of Umbelopsis and Byssochlamys isolates at differing temperatures. Different letters at the same temperature show statistically different growth rates (ANOVA; p ≤ 0.05). Standard deviation bars are shown.

(p ≤ 0.05). At 20º C, *U. versiformis* grew significantly faster than *U. vinacea* and *B. nivea* isolates (p ≤ 0.05). However, at 30º C *B. nivea* grew significantly faster than *Umbelopsis* spp. (F (2,37) = 690.415; p = 0.000). At 30º C *Umbelopsis* spp. growth had nearly ceased while the *B. nivea* growth did not cease growth until 35º C.

When *Armillaria ostoyae* and *B. nivea* were grown together at 25º C there was no evidence of *B. nivea* inhibiting the growth of *A. ostoyae* (Figure 2). Only in one case was there a significant difference in growth (*A. ostoyae* 286 and *B. nivea*; t = 7.790; p = 0.001; d.f. = 4; two tailed paired t-test) and in this case *A. ostoyae* growth was enhanced relative to controls (Figure 2). No other differences were significant. There was a yellow pigmented interaction zone formed between *A. ostoyae* and *B. nivea* in some of the pairings, but this zone formation did not appear to be related to growth trends. There were also no observable changes in hyphal morphology as a result of the pairings either in the petri dishes or on the microscope slides.

![Graph: Radial growth rates of Armillaria ostoyae alone (control) and A. ostoyae growing with a single isolate of Byssochlamys nivea (antagonism pairing) at 25ºC.](image)

**Figure 2**. Radial growth rates of *Armillaria ostoyae* alone (control) and *A. ostoyae* growing with a single isolate of *Byssochlamys nivea* (antagonism pairing) at 25ºC. There were no statistical differences among *A. ostoyae* 957 and *B. nivea* or among *A. ostoyae* 284. Growth of *A. ostoyae* 286 was enhanced nearly four-fold when grown with *B. nivea* (t = 7.790; p = 0.001; d.f. = 4; two tailed paired t-test). Standard deviation bars are shown.

### Conclusions

1) *Byssochlamys nivea* was commonly found in the soil rhizosphere.

2) The optimum temperature for growth of both fungi is 25º C, but *B. nivea* grew somewhat better at temperatures warmer than 25º C and *Umbelopsis* spp. grew somewhat better at cooler temperatures.

3) *Byssochlamys nivea* did not inhibit the growth of *Armillaria ostoyae* in culture. In fact, it appeared to stimulate the growth of this pathogen in one sample.

### Acknowledgements

This project was funded by the USDA Forest Service and the Joint Fire Science Program. The authors wish to thank Paul Hessburg, Joe Ammirati, Sharon Doty, Brion Salter, Ned Klopfenstein, Nick Brazee, Ryan Hook, Adam Mouton, and Marianne Elliott for their help.

### References


Abstract—Technology transfer is the dissemination of research. While mandated by the USDA Forest Service, little information exists outlining current use and preferences of Forest Service personnel for different technology transfer mediums. This research will assess current and preferred communication mediums of forest health research information through the use of a survey. Effective technology transfer should be built into all research to ensure that new information is adopted into everyday use.

Common terms for technology transfer are adoptions theory, diffusion of innovations, and social marketing. Barriers to effective technology transfer of forest science have been identified by some researchers. These include lack of time, availability of science, conflicting results, lack of trust, and cultural differences between researchers and users.

Forest health research is important to ensure maintenance of healthy forests. Efficient transfer of new research information will help overcome the technology transfer barriers and spread new knowledge of forest health. Therefore, a critical look at technology transfer and the movement of research is important. We are surveying over 250 district-level Forest Service personnel in the Rocky Mountain Region including silviculturists, botanists, foresters, recreation leaders, trail leaders, biologists, timber managers, fire management officers, and fuels planners. This survey will be used to evaluate the technology transfer in two case studies in which research is moved from researcher to user. In addition, this survey will provide valuable tools for transferring future forest health research results to all Forest Service personnel.

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John E. Lundquist is a research pathologist with USDA Forest Service, Rocky Mountain Research Station, Fort Collins, CO 80526
The Role of Armillaria Species in Canopy Gap Formation within the Temperate, Old-Growth Rainforests of the Hoh River Valley, Olympic National Park, Washington

Nicholas J. Brazee

Abstract—Ecologists have long overlooked the impacts that endemic pathogenic fungi have upon disturbance regimes. The impacts of fungi on tree death are not as apparent at the landscape level while fire, insects, and wind are readily observed. Yet, single tree and small group mortality caused by fungi, taken into context at the landscape level, represents the majority of annual tree death in coastal forests. Armillaria is the most common pathogenic fungus causing root rot within the Olympic National Forest, and is very common within the old-growth forests of Olympic National Park. Previous researchers have found five species of Armillaria on the Olympic Peninsula: A. ostoyae, A. sinapina, A. cepistipes, A. nabsnoma, and A. gallica. Despite a wealth of information concerning Armillaria in managed stands, little is known about how Armillaria influences the development of vertical and horizontal stand structure in natural forests. A complex of factors, including major windstorms, southern aspect, hemlock dwarf mistletoe (Arceuthobium tsugense (Rosendahl) Jones), and Armillaria root rot all coalesce to create a mosaic of canopy gaps across the landscape within the Hoh River valley. This has a significant impact upon forest structure, regeneration patterns, plant species diversity, and forest succession. The objective of this study is to determine the spatial distribution of Armillaria spp. in a natural forest system across primarily two forest zones, the Sitka spruce (Picea sitchensis (Bong.) Carr.) and western hemlock (Tsuga heterophylla (Raf.) Sarg.) zones, but also incorporating limited areas of the Pacific silver fir (Abies amabilis Dougl.) zone. By studying the distribution of this fungus in a forest where fire and timber harvesting are absent removes two major disturbance variables that have a substantial impact on the presence and dispersal of this fungus in managed systems. This will help to build a better understanding of how this fungus influences mortality and canopy gap formation, and how human imposed management has altered the presence of Armillaria species on the landscape. By focusing on root rot fungi, this study aims to support the theory that fungi can be a dominant factor in natural disturbance regimes. For this study 100 isolates of Armillaria were collected, primarily from western hemlock, across ten canopy gaps along three trail transects in the Hoh River valley. Five gaps were located in the western hemlock zone, four within the Sitka spruce zone, and one within the Pacific silver fir zone. Five isolates were collected from each gap and five outside each gap in order to establish that Armillaria is ubiquitous across the landscape. RFLP analysis of rDNA will show how the five species of Armillaria present on the Peninsula are distributed across the gaps and the surrounding stands. RAPD analysis will show clone distribution, and shed insight on how multiple species are interacting across the landscape. It is clear that this forest is not dominated by A. ostoyae like many western interior forests, and the majority of blown down trees have extensive root rot. This observation would suggest that less pathogenic species of Armillaria were responsible for high levels of root decay in western hemlock. Whether or not species of Armillaria like A. cepistipes or A. sinapina are capable of causing significant rot in unmanaged system is still not clear. This study will not likely resolve this issue, but raises doubt about our understanding of Armillaria pathogenicity especially in forests where large disturbances have not been operating and root diseases are driving small gap dynamics.


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Forest Fire Intensity Affects the Abundance of Armillaria Root Disease

J.T. Blodgett and J.E. Lundquist

Abstract—Diseases influence forest fires by changing stand structure and generating fuels. Conversely, fires influence the abundance and distribution of fuel-generating diseases, but few studies have examined these relationships. The Black Hills in South Dakota has been the focus of much attention following recent severe fires. Armillaria root disease, caused by Armillaria ostoyae, is arguably the most common disease in this forest. This field study was designed to examine the effects of fire on the abundance and potential spread of Armillaria root disease in ponderosa pine (Pinus ponderosa) forests. Five plots were established in the Jasper Fire area of the Black Hills National Forest, SD that burned 3 years previous to this study. Each plot consisted of four subplots that varied in fire intensity (i.e., low, medium, and high fire intensity, and unburned). Host condition (living or dead) was recorded for 15 ponderosa pine trees per subplot. The root collar of each tree was divided north-south by east-west into quarters, and the abundance of Armillaria rhizomorphs was ranked for each quarter. The abundance of Armillaria rhizomorphs on root collars increased ($P < 0.001$) as fire intensity increased. Pair-wise comparisons were significant among fire intensities except between unburned and low intensity subplots. As expected, host condition worsened ($P < 0.001$) as fire intensity increased with average mortality of 0% for unburned and low fire intensity subplot trees, 59% for medium fire intensity subplot trees, and 100% for high fire intensity subplot trees. Our results show that Armillaria can survive intense fires and can readily colonize roots of trees killed by fire. Therefore, wildfires can increase Armillaria inoculum, which might result in increased future fire-related Armillaria mortality.


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Quantifying Swiss Needle Cast Needle Infection using APS Assess Image Analysis Software

John Browning, Will Littke, Carol Larson

Abstract — Pseudothecia of the Swiss Needle Cast (SNC) pathogen Phaeocryptopus gaeumannii have been quantified by counting their presence in individual stomates on the underside of Douglas-fir needles using a light microscope. The number of stomata plugged by pseudothecia divided by the total stomata area counted is often used to express SNC infection (termed Pseudothecial Density or PD). This method is very time consuming and laborious to undertake on a large number of foliar samples. An alternative method to quantifying SNC infection has been tested using a photo image of the underside of the needle. A digital image is captured using a Digital Microscope similar to the one sold by Spectrum Technologies and capable of 140X magnification. The digital image is processed using APS Assess software to quantify the percentage of stomata plugged with pseudothecia. Digital imaging was compared with visual counts made on the same images. We observed that this method does tend to slightly underestimate total pseudothecial density compared to visual estimates. For this reason a linear regression is used to convert the image analysis estimate to a corresponding visual estimate ($y = 1.4478x + 0.1768$, $R^2 = 0.80$). This method is quick and accurate means to quantify SNC on a large number of samples. Photos also allow you to go back and resample needles. Digital imaging overcomes some of the difficulties experienced in using card mounted needle samples for microscope examination. Another benefit of this technique is that it reduces operator bias and error found in ocular estimates of pseudothecial density, and is relatively simple to learn.
Pruning Blister Rust Cankers to Preserve High-Value Trees

Kelly Sullivan Burns, Amanda Crump, William R. Jacobi, and Brian Howell

Abstract—Limber pine (Pinus flexilis) and Rocky Mountain (RM) bristlecone pine (P. aristata) are especially important in the Rocky Mountain Region because of their unique ecological and cultural characteristics; however, the survival and longevity of important trees is threatened by the exotic, invasive disease, white pine blister rust (WPBR) (causal fungus Cronartium ribicola). Management techniques are available for protecting and prolonging the life of other white pines in the presence of WPBR but these techniques have not been tested on limber and RM bristlecone pine. The purpose of this study is to evaluate the efficacy of several pruning and canker removal treatments in reducing WPBR infections and decreasing mortality in high-value limber and RM bristlecone pines. An additional objective is to assess the various treatments in terms of cost and feasibility.

Tree and WPBR data were collected on approximately 800 trees from two study sites (the Great Sand Dunes National Park and Preserve in southern Colorado and Vedauwoo Campground in southern Wyoming). Treatments implemented included conventional pruning, pruning cankers only, conventional and canker pruning, and no treatment. Post-treatment data collection included treatment implementation time and number of cankers removed. Data is currently being summarized and a preliminary report will be available winter 2005. Both study sites will be re-visited in 2006 for an initial evaluation of treatment effectiveness. Trees will be re-evaluated every 3-5 years thereafter.
Generic Site Mapper
Tony Courter, Frank Sapio, and Judy Adams

Abstract--Generic is defined by two terms ‘universal’ and ‘standard’ both have an important role within Forest Health Protection. The generic site mapper is a package or suite of tools comprising:

1) A computer based data entry form for adding or editing individual records.

2) The ability to pull in data collections from a variety of formats such as excel spread sheet, ascii flat file, or access database.

3) And a mapping functionality to portray these records.

Its relevance is especially important to FHP customers comprising a variety of land ownerships. Although the desire to have standard data in a common format still exists, it rarely happens. With the generic site mapper the data sources can be submitted from state foresters, universities, federal agencies, or any other entity, within their preferred format and the application will display all records on a map seamlessly. The mapping capability is based on a product called Map Objects Lite. This selection was made with the realization that some functionality gained by using an ESRI product would be given up for the ability to share with a larger audience. The maps can be displayed on computers outside the Forest Service, increasing our ability to communicate with external cooperators and partners.

Each project comes in with unique requirements based on geographic area, forest health concerns, customers, and a number of other variables. More important, however, are the commonalities many projects share including: relevant data in different formats, the need to add, or modify records, a requirement for mapping, and the ability to share the results.

From a technology viewpoint there can be and should be standardization across projects. Capitalizing on a universal template will reduce development time, minimize support costs, and provide a common basis for dialogue. The Generic Site Mapper has lived up to its name by offering a universal technology to a broad customer base and setting a standard for project data.


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Dothistromin Production and its Relationship to Genetic Diversity in the Fungal Pathogen *Dothistroma septosporum*

Angie Dale, Kathy Lewis, and Alex Woods

**Abstract**— *Dothistroma septosporum* (Dorog.) Morelet, a foliar fungal pathogen of two and three needle pines, is responsible for a large disease outbreak on lodgepole pine (*Pinus contorta*) in northwest British Columbia. This pathogen has been an important disease agent in exotic pine plantations in the Southern Hemisphere (Gibson, 1972), but has been of little concern in countries of the Northern Hemisphere. The current disease outbreak in B.C. is unique in the fact that a native pine species is being affected, and in the severity and extent of the outbreak. Favourable environmental conditions for disease (Woods and others, 2005), as well as the existence of the teleomorph in B.C. (Funk & Parker, 1966, Parker & Collis, 1966) may have led to a case where sexual reproduction has allowed for rapid evolution of the pathogen population and possibly a more virulent strain of the fungus. The purpose of this study is to explore the genetic structure of *D. septosporum* populations in northwest B.C., to relate population structure to reproductive strategies and to current forest practices, and to relate population genetic structure to toxin producing abilities of the pathogen. Preliminary results have shown genetically distinct individuals both within a single site and between four different sites.

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Fungal Endophytes Isolated from Roots of Douglas-fir and Ponderosa Pine from Prescribed Burn Sites

John R. Goetz III, Jack D. Rogers, Lori M. Carris, Paul Hessburg, Ned B. Klopfenstein, and Mee-Sook Kim

Abstract—Little is known about the diversity and ecological roles of endophytes in woody roots of forest trees in the inland northwestern USA, especially as related to fire ecology. To assess fungal endophytes in large woody roots of Douglas-fir (*Pseudotsuga menziesii*) and ponderosa pine (*Pinus ponderosa*), we conducted a mycobiotic survey of root-core samples from 12 sites in the Okanogan-Wenatchee National Forest of north-central Washington. Each site was subjected to one of four fuels treatments: 1) untreated control; 2) burn only (relatively low intensity surface fire); 3) thin only; and 4) thin and burn. Fungal endophytes were cultured on nutrient media, and were identified by morphology and DNA sequencing of the internal transcribed spacer region (ITS1, 5.8S, ITS2) of rDNA. In total, 594 cores were processed and approximately half yielded one or more fungal isolates. Based on preliminary results, 174 isolates representing 13 genera of hyphomycetes predominate, followed by 35 isolates representing 4 genera of the Ascomycota, and 52 isolates representing 3 genera of the Zygomycota. The identification of isolated fungal endophytes is continuing.
Evaluating the Health of Five-Needle Pines: An Overview from the Pacific Northwest Region with Emphasis on Western White Pine and Sugar Pine in Southwest Oregon

Ellen Michaels Goheen and Donald J. Goheen

Abstract—Five-needle pines are ecologically important components of forest stands in the Pacific Northwest. This poster presents information gathered from four analyses done in 2003 and 2004 to evaluate five-needle pine health including: 1) A Regional assessment of the health and distribution of five-needle pines from available inventory data including FIA (Forest Inventory and Analysis) and CVS (Current Vegetation Survey) plots; 2) A detailed query of pine health from inventory plots across all lands in Southwest Oregon; 3) An in depth survey of 110 forest stands with sugar pine or western white pine components distributed on federal lands throughout Southwest Oregon; and 4) An intensive survey of 100 15 to 25-year-old plantations with western white pine or sugar pine components distributed on federal lands throughout Southwest Oregon.

A query of data from 15,232 FIA, USDA FS R5 and R6 (Forest Service Regions 5 and 6), and USDI Bureau of Land Management (BLM) inventory plots collected from 1991-2000 in Oregon, Washington, and R6 Forest Service lands in California shows western white pine, sugar pine, and/or whitebark pine on 2128 plots (14 percent). Plots with western white pine predominated (58 percent). Data from 519 plots (24 percent) report five-needle pine mortality, data from 232 plots (11 percent) report bark beetle-caused mortality and data from 559 plots (26 percent) indicate the presence of white pine blister rust (caused by the fungus Cronartium ribicola). No five-needle pines were reported on plots in the north and central Oregon Coast Range which is part of western white pine’s historic range. No insect and disease data were collected for trees < 1” DBH.

A query of data from 2,749 FIA, FS R6, and BLM inventory plots collected from 1993-1997 in Coos, Curry, Douglas, Jackson, Josephine, and Lane Counties in Oregon and R6 Forest Service lands in California shows western white pine, sugar pine, and/or whitebark pine occur on 860 plots (31 percent). On plots with five-needle pines plots with sugar pine predominate (64 percent). Only 4 inventory plots contained whitebark pine. Five-needle pine stocking averaged 6 percent of total trees per acre. White pine blister rust was recorded on 234 plots (27 percent), and associated with 74 percent of all dead five-needle pines. Thirty-two percent of live five-needle pine stocking was recorded as infected.

An in-depth survey was done of 110 randomly selected natural stands across Southwest Oregon. Both western white pine and sugar pine were found to be declining in these stands. Regeneration is occurring; however, mortality and disease occur in all size classes, particularly in trees > 10” DBH. High levels of white pine blister rust were observed causing topkill and branch dieback in trees >10”dbh and mortality in trees < 10” DBH. Ribes presence did not appear to be essential for disease development. High levels of mountain pine beetle (Dendroctonus ponderosae)-caused mortality were also observed. Current live basal area thresholds indicating risk of mountain pine beetle attack for sugar pine (150 ft² /ac) and western white pine (130 ft² /ac) were exceeded in 55 percent of surveyed stands. Pine engraver (Ips paraconfusus) infestation was common in western white pine greater than 6” DBH, especially on ultramafic soils. Bark beetle-caused mortality was recorded on 91 plots (10 percent). Bark beetles were associated with 86 percent of all dead five-needle pines.

Sixty-three sugar pine and 43 western white pine plantations were surveyed. Plantations were selected from local databases among those with 10 percent or more stocking of five-needle pines and represented a range of five-needle pine stock types from wild to white pine blister rust-resistant. Average stocking of plantations with western white pine was 234 total trees per acre with an average western white pine stocking of 43 percent. Forty percent of western white pine had white pine blister rust. Average stocking in plantations with sugar pines was 184 total trees per acre with an average sugar pine stocking of 37 percent. Forty-three percent of sugar pines had white pine blister rust.

In summary, survey results indicate: 1) western white pine and sugar pine occur in heavily stocked stands and are at high risk to attack by mountain pine beetle; 2) mountain pine beetle-caused mortality is already wide spread and extensive, affecting larger diameter pines in many areas; 3) white pine blister rust is often associated with mountain pine-beetle attacked trees, and 4) white pine blister rust commonly kills young planted and naturally regenerated five-needle pines, preventing them from replacing the large trees that are being killed.
Phylogeographic Structure of *Armillaria ostoyae* in the Western United States

John W. Hanna, Ned B. Klopfenstein, Mee-Sook Kim, Geral I. McDonald, and James A. Moore

**Abstract**—Direct-PCR was used to obtain sequences of nuclear ribosomal DNA regions (i.e., large subunit, internal transcribed spacer, 5.8S, and intergenic spacer) from *Armillaria ostoyae* genets collected in the western USA. Sequence analysis using Bayesian inference methods defined three phylogenetic groups. Two phylogeographic groups were associated with the Rocky Mountain and Pacific Northwest regions of the USA. Additional analysis of *A. ostoyae* from outside the western USA indicates the presence of a circumboreal group with representation in Utah, USA. Individual genets containing heterogeneous sequence combinations from multiple phylogenetic groups were common in some geographic regions. Heterogeneous sequences indicate intragenomic variation and/or intraspecific hybridization. Hypothetically, phylogenetic groups may have physically converged after long-term geographic isolation. Subsequent hybridization events may have influenced evolution and contributed to variation in ecological behavior of *Armillaria* species.


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Association of *Ophiostoma novo-ulmi* with *Scolytus schevyrewi* Semenov (Scolytidae) in Colorado Elm Trees

W. R. Jacobi, R. D. Koski, T. C. Harrington, and J. J. Witcosky

**Abstract**—The European elm bark beetle, *Scolytus multistriatus*, has been the primary vector of the Dutch elm disease fungus, *Ophiostoma novo-ulmi*, in elm trees in Colorado since the first report of the disease in the state in 1948. An exotic from Asia, the banded elm bark beetle, *Scolytus schevyrewi*, was found in *Ulmus pumila*, Siberian elm, in Colorado in April of 2003; this was the first report of *S. schevyrewi* in North America. *Scolytus schevyrewi* is found throughout much of Colorado, and in at least 21 other states. The similarities in breeding and feeding habits between *S. schevyrewi* and *S. multistriatus* have raised concerns about the ability *S. schevyrewi* to vector *O. novo-ulmi*. The objective of this study was to determine if *O. novo-ulmi* could be cultured from adult *S. schevyrewi* emerged from confirmed Dutch elm disease positive elm trees. *Scolytus schevyrewi* and *S. multistriatus* were reared from caged stem segments of diseased *Ulmus americana*, American elm trees. The beetles were collected, identified, and plated onto selective media. *Pesotum ulmi*, an anamorph of *O. novo-ulmi* was cultured from many of the *S. schevyrewi* and *S. multistriatus* adults.
Abstract—Several resistant gene candidates (RGCs) were isolated in sugar pine (*Pinus lambertiana*) in an effort to determine the nucleotide sequence of the major gene of resistance (*Cr1*) to *Cronartium ribicola*, an introduced fungal pathogen which causes white pine blister rust (WPBR) in the white pines. A complete coding sequence with high similarity to the CC-NBS-LRR class of resistance genes was isolated from a cDNA library constructed from sugar pine resistant seedlings and was found to have no sequence variation in its coding region in 26 sugar pines and in a single western white pine (*P. monticola*). Several RGCs of the TIR-NBS-LRR class of resistance genes that were reported in western white pine elsewhere were cloned in sugar pine and sequenced in megagametophytes from sugar pine tree 5701, which is heterozygous for resistance to WPBR. Three of the five RGC sequences were polymorphic but did not associate with WPBR resistance. Linkage analyses and genetic mapping with these loci is pending.
Persistence of Pinyon Pine Snags and Logs in Southwestern Colorado

H. S. J. Kearns, W. R. Jacobi and D. W. Johnson

Abstract--We utilized the number of years since death to examine the persistence of pinyon pine (*Pinus edulis* Engelm.) standing snags and downed logs in southwestern Colorado pinyon-juniper woodlands. Time since death of pinyon pines killed by bark beetles, black stain root disease, or unknown causes in three study areas in southwestern Colorado was determined through monitoring of permanent plots and dendrochronological cross-dating methods. The structural condition and form of the trees was recorded and related to time since death. Pinyon snags may persist for up to 25 years, with persistence of intact snags averaging 8.4 years and broken snags averaging 13.2 years. Sound logs had been dead a mean of 9.8 years, while more fragmented logs had been dead for a mean of 14.4 years. Extremely fragmented trees had been dead for 16.2 years. There was no statistically significant difference in time since death for snags versus logs in similar condition classes. Pinyon pine form and condition were related to time since death so the date of death can be estimated from this information.


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Armillaria Response Tool (ART)

Mee-Sook Kim, Geral I. McDonald, Thomas M. Rice, David E. Hall, Jane E. Stewart, Jonalea R. Tonn, Philip D. Tanimoto, Paul J. Zambino, and Ned B. Klopfenstein

Abstract—Armillaria Response Tool (ART) is a web-based tool that can estimate Armillaria root disease risk in dry forests of the western USA. It uses habitat types to identify sites with high or low risk potential for developing Armillaria root disease, and indicates how some fuels management treatments may exacerbate Armillaria root disease within high-risk stands. ART guides stand-level choices of appropriate fuels management treatments that should minimize future damage by Armillaria root disease. This web-based tool is part of the Fuels Planning: Science Synthesis and Integration Project (Dr. Russell T. Graham, Program Leader), a pilot project initiated by the USDA Forest Service in response to the need for tools and information useful for planning site-specific fuel treatment projects. More information and a functional version of the tool can be viewed at our web site: http://forest.moscow.fsl.wsu.edu/fuels/art/.
Stand Characteristics and Fuel Loads of Dwarf Mistletoe and Mountain Pine Beetle in Colorado’s Northern Front Range Ponderosa Pine

Jennifer G. Klutsch, Russell D. Beam, William R. Jacobi, and José F. Negrón

Abstract--Dwarf mistletoe and bark beetle infestations are suspected to change fuel complexes and fire hazards. Common assumptions maintain that certain stand characteristics in ponderosa pine (Pinus ponderosa var. scopulorum) infested with dwarf mistletoe (Arceuthobium vaginatum subsp. cryptopodum) or mountain pine beetle (Dendroctonus ponderosae) should alter fire behavior, as compared to un-infested stands. Furthermore, stands with both dwarf mistletoe and mountain pine beetle activity should have different fuel levels and stand characteristics than separately infested stands. The stand structure and fuel loads in the ponderosa pine forests of the Front Range of Colorado were surveyed in areas with dwarf mistletoe and mountain pine beetle infestations. Thirty-one transects, each 1500m in length, were established throughout the Canyon Lakes Ranger District, Roosevelt National Forest through the summer of 2005 to determine the extent and intensity of the dwarf mistletoe and mountain pine beetle populations. Within the 91 tree plots (0.04 hectare) where stand characteristics were recorded, fuel levels were measured using modified Brown’s transects. The data collected will test whether, and to what degree, these disturbances influence stand characteristics. As well, fire behavior models will help determine the potential surface fire behavior, torching potential and independent crown fire potential in infested stands.


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Creating a Database of Condition and Distribution of Whitebark and Limber Pines

Blakey Lockman, Gregg DeNitto, Tony Courter, and Ronda Koski

Abstract —The goal of this project is to create a database of basic plot information on whitebark and limber pines from the numerous surveys and studies that have been completed. FIA plot data is part of the database. This database is not a repository for raw data. The data can be queried to provide a spatial summary of the condition of these two species. Cooperative work is occurring with the US National Park Service towards the creation of an interface for simplifying entry of future survey/study data by individuals.

The first level of the database has basic descriptors of plot/study results that can be queried and GIS-linked. A second level is planned to include a more detailed list of variables with the source information. The user will be able to query these variables using yes/no statements. An example of such a variable and query is “Ribes evaluated, yes/no?” For more detailed information on these second level variables, the user will have to access the publication or get in touch with the contact person.

The results from this effort include a completed interactive database. Maps of the species distribution, the known locations of blister rust, the overall condition of the species, and a map that depicts the obvious gaps in data on the condition of these two species are forthcoming.
Recovery, Detection, and Observation of Phytophthora ramorum from Sapwood of Mature Tanoak

E. Oh, J.L. Parke, and E.M. Hansen

Abstract--The sudden oak death pathogen Phytophthora ramorum causes lethal bole cankers on some members of the Fagaceae. Infection is believed to be restricted to the inner bark and cambium although there are two unpublished reports of isolation from the wood. Bole sections from naturally-infected mature tanoaks were split and cut into 1-cm-thick transverse sections. Sections were photographed and tissue samples were removed from marked sites in the bark, cambium, and wood for isolation on selective medium, scanning electron microscopy (SEM), and diagnostic PCR. SEM revealed both hyphae and chlamydospores in xylem vessels and some hyphae in fiber tracheids near vessels in discolored sapwood. In the vessels, the hyphae mostly grew straight and branched out but hyphal swelling was observed either alone or with chlamydospores. Hyphae were observed in discolored bark but not in discolored pith. P. ramorum was often but not always recovered from brown streaked sapwood. Tyloses appeared to be produced by tanoak in response to infection. Various shapes of tyloses were observed in xylem vessels either alone or with chlamydospores. The frequency of P. ramorum detection was highest with PCR. Also, P. ramorum was detected in sapwood less frequently than from bark samples.
Abstract--Fallen hazardous trees are a valuable source of information for prospective management decisions made by cities, recreation site managers, and homeowners. The International Tree Failure Database (ITFD) offers internet data entry for quantitative details collected from branch, root or trunk failures stored in a database for future analysis.

The ITFD was modeled after a local effort called the California Tree Failure Report Program (CTFRP) initiated in 1987 from the San Francisco Bay Area Hazard Tree Group. Over 200 tree care professionals in California cooperated in gathering details from fallen trees then mailing their forms into the data entry site. The California program database repository housed over 3400 urban tree reports. Management and support of the program are accomplished through the University of California Cooperative Extension.

One of the goals of the ITFD was to capitalize on the many successes credited to the California program and improve upon the limitations. Obviously the scale was increased taking the program from its local geographic area to an international level. Data entry can be accomplished by the cooperators themselves through the internet. Other general information about the program is available to anyone with internet access and includes training schedules, a copy of the data collection form, and a user’s guide. Data entry privileges are password restricted to qualified individuals who have completed a training class.

The entire development of the ITFD has reached this stage in production through several on-going cooperative efforts. Arborists and foresters debated data fields, level of detail, and format design reaching consensus on a single form for the collection of standardized data. Based on the failure, the user determines one of three appropriate pathways for branch, root, or trunk specifics. General information is also requested on the tree, weather conditions, and surrounding landscape.

USDA Forest Service agencies have worked together providing the funds necessary for the development and support to the ITFD. Forest Health Protection has provided financial support for the database design and development efforts, the Urban and Community Forestry is funding the creation of support tools such as the user’s guide, definitions for an on-line help system, and a standard training package.


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Using Ground Penetrating Radar to Estimate Rot Volumes in Living Tree Stems: Exploration of a Non-Destructive Technique and its Application

Michele L. Pruyn, John R. Butnor, David C. Shaw, Mark E. Harmon and Michael G. Ryan

Abstract—Decomposition by saprophytic organisms of wood in living tree stems contributes substantially to disease loss in U.S. forests, causing destruction or decreased value of usable timber and the depletion of carbon reserves in forest stands. Precise methods that predict rot volume are lacking. Our objective was to test and apply Ground Penetrating Radar (GPR) to non-destructively estimate active and inactive rot volumes in living trees. Ten hazard trees of three different species were selected for felling: *Pseudotsuga menziesii*, *Thuja plicata*, and *Tsuga heterophylla*. GPR scans of the bole’s circumference revealed the proportion of rot within. The GPR likely detected changes in the properties of specific gravity and moisture content as the electromagnetic signal moved from solid wood into decay. We then used the GPR in the Wind River Research Natural Area (WA, U.S.A.) to scan four living trees of each species. Tree climbers cooperated with researchers in the canopy crane gondola to collect scans at multiple heights along the tree boles. Using respiration measurements of sound versus decayed wood in tree boles, we will scale-up to the stand level for a more accurate representation of respiratory fluxes from ecosystems. This will improve current flux estimates, which do not include rot respiration. A more precise estimation of rot volumes in forest ecosystems will enable a better prediction of losses to harvestable timber and carbon reserves.


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Sudden Oak Death Survey and Monitoring in Western Washington

Amy Ramsey and Dan Omdal

Abstract—Phytophthora ramorum, also known as Sudden Oak Death, ramorum leaf blight, and ramorum dieback, is responsible for killing tens of thousands of native oak and tanoak trees in California. It has also been discovered in southern Oregon, where efforts are underway to eradicate and prevent the spread of this pathogen northward. While Washington’s native oak species Oregon White Oak (Quercus garryana) is not threatened by P. ramorum, Pacific madrone, huckleberry, rhododendron, and Douglas-fir, among many others, are susceptible hosts. Thirty wildland forest areas and ten aquatic survey plots were monitored and surveyed in the summer of 2005 to detect P. ramorum in western Washington. P. ramorum was not found in Washington’s wildland or aquatic areas in 2005.
DNA Based Detection of Western Gall Rust

Tod D. Ramsfield and Detlev R. Vogler

Abstract—Western gall rust (*Peridermium harknessii* syn. *Endocronartium harknessii*) is a serious pathogen of Monterey Pine (*Pinus radiata*) in its native range, and since exotic forestry in New Zealand is heavily reliant upon *P. radiata*, the pathogen is considered to be a threat to the New Zealand forest industry. As the time between initial infection and spore production can take up to two years, a DNA based marker has been developed that is able to detect the presence of pathogen DNA within galled tissue prior to sporulation. The detection system was initially based on polymorphisms in the internal transcribed spacer region of the ribosomal DNA, but the marker that was developed was unable to distinguish *P. harknessii* from its close relative *Cronartium quercuum*. To increase the specificity of the marker system, the intergenic spacer region of the ribosomal DNA of *P. harknessii* and all four formae specialis of *C. quercuum* was sequenced and compared. This allowed the development of a marker system that was able to distinguish *P. harknessii* from all *C. quercuum* formae specialis with the exception of *C. quercuum* f. sp. *banksianae*. IGS sequence alignment was used to construct a phylogeny to compare *P. harknessii* and *C. quercuum* and this analysis further supported earlier evidence that *C. quercuum* f. sp. *banksianae* is the closest relative of *P. harknessii*.
Fungi Associated With Wood-Stake Decomposition in the Northwestern U.S.A.

Raini C. Rippy, Ned B. Klopfenstein, Mee-Sook Kim, Deborah S. Page-Dumroese, Jack D. Rogers, and Lori M. Carris

Abstract—Fungi associated with wood decomposition have diverse roles that contribute to the physical and chemical components involved in the long-term productivity of forest soils. Standard substrate wood stakes were buried to a depth of 30 cm in the mineral soil and at the forest floor/mineral soil interface on five forested sites in Idaho and Washington to examine fertilizer influences on fungal distribution and wood decomposition rates. Fungi were isolated from these stakes at 6-month intervals. Morphological characterization and ITS rDNA sequencing are being used for fungal identification. During spring 2004, 25 genera of basidiomycetes, 16 genera of ascomycetes and deuteromycetes, and 3 genera of zygomycetes have been tentatively identified.
Potential Impacts of Fuels Treatments on Root Diseases: A Synthesis

Raini C. Rippy, Jane E. Stewart, Paul J. Zambino, Ned B. Klopfenstein, Joanne M. Tirocke, Mee-Sook Kim, and Walter G. Thies

Abstract--Forest structure in the Inland West has changed over the last 100 years as a result of fire suppression, selective harvesting, and introduced pests. Shade tolerant species (e.g., grand fir), which under most circumstances are more susceptible to root diseases than early seral species, have increased in many forests. Heavy accumulations of fuels have put many stands at risk for severe wildfires. Managers are considering fuels treatments, such as thinning, prescribed fire, mowing, mulching, and combinations of these treatments, to potentially reduce wildfire severity. However, it is important to consider future impacts of root diseases before implementing any management activities. A single type of treatment will not be appropriate for all root diseases and forest conditions. Root diseases that can be affected by fuels treatments include: Armillaria root disease, annosus root disease, laminated root rot, black stain root disease, schwinitzii root and butt rot, tomentosus root disease, Rhizina root rot, and stringy butt rot. It is essential to determine presence and significance of each root disease in a stand before considering the appropriateness of fuels treatments. Specific interactions between treatments and root diseases and potential modifications of treatments to minimize specific diseases have been demonstrated. It is essential to determine root disease presence in a stand before considering appropriate fuels treatments. A better understanding of the interactions of fuels treatments with root diseases is needed. Long-term studies could provide more precise information for understanding complex relationships between root diseases, pests, fire, and fuels management.


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Genetic Resistance in Port-Orford-Cedar to \textit{Phytophthora lateralis}: Survival of Seedlings from First Orchard Seed in Greenhouse Testing

Richard A. Sniezko, Kristin Mylecraine, Leslie Elliott, Scott Kolpak, Paul Reeser, and Everett Hansen

\textbf{Abstract}--Port-Orford-cedar (\textit{Chamaecyparis lawsoniana}) is highly susceptible to the non-native, invasive root pathogen \textit{Phytophthora lateralis}, the cause of Port-Orford-cedar root disease. Mortality from this disease has impacted forest ecosystems and has greatly restricted the utilization of Port-Orford-cedar (POC) for restoration and reforestation. Fortunately, a low incidence of genetic resistance is found within some POC populations. In the mid-1990s, the USDA Forest Service and USDI Bureau of Land Management began an operational program to develop genetically resistant populations of POC. Working in conjunction with researchers at Oregon State University, over 10,000 field selections have undergone the first stage of resistance screening, a stem dip test. The second screening stage, a root dip test, is well underway for the top 10\% of parents from the stem dip test. Selections with high survival in the root dip test are placed into containerized seed orchards at Dorena Genetic Resource Center, with separate orchards for each breeding zone. This study represents the first greenhouse test of POC orchard seedlots from the resistance program. The first orchard cone crops were harvested in 2002, and seedlings for this study were root dip tested for resistance in 2004. Three types of seedlots were included in the trial (1) containerized seed orchard (CSO) seedlots, including parents selected from both stem and root dip testing, (2) clone bank (CB) seedlots, including parents selected only from stem dip testing, and (3) Woodsrun (WR) seedlots, representing control seedlots with no selection for resistance. The study design was a randomized complete block with 12 seedlings per replication and 4 replicates (48 total seedlings per family). Seedlings were inoculated by immersing the lower 1 cm of roots in zoospore solution. Mortality was monitored for 12 months. Both CSO and CB seedlots exhibited reduced mortality compared with control WR seedlots.

CSO seedlots from five breeding zones were compared against WR seed. Three seedlots showed significantly lower mortality (33 to 50\%) than the WR mortality seedlots while two seedlots showed a non-significant trend with 13 and 19\% lower mortality. CB seedlots from four breeding zones were also compared against WR seed. All four seedlots showed a non-significant trend with 8 to 25\% lower mortality. The highest mortality level (93.8\%) was in WR from breeding zones 130 and 430, and the lowest mortality level (37.5\%) was in CSO from breeding zone 215.

Initial criteria for inclusion of parents in these first-generation orchards were liberal and survival for at least some orchard lots is expected to increase with further testing and roguing of orchards. As screening results are updated, new orchards will be created for additional breeding zones, resistant parents will be added to existing orchards, and existing orchards will be rogued as necessary. Other ongoing work in the resistance program involves testing new field selections for resistance, breeding to increase levels of resistance, and implementing field trials to examine utility and durability of resistance. The full poster is available on the Dorena Genetic Resource Center website (http://www.fs.fed.us/r6/dorena/publications/poc).
Genetic Characterization of Highly Virulent
_Fusarium oxysporum_ from a Conifer Nursery

Jane E. Stewart, Mee-Sook Kim, Robert L. James, R. Kasten Dumroese, and Ned B. Klopfenstein

Abstract—Root-rot disease caused by _Fusarium oxysporum_ can cause severe loses in conifer nurseries. This fungus commonly occurs in container and bare-root nurseries on healthy and diseased seedlings, conifer seeds, and in nursery soils. Though isolates of the fungus differ in virulence, studies show that pathogenicity and isolate morphology are not correlated. Forty-one isolates of _F. oxysporum_ collected from a conifer nursery were selected for pathogenicity tests and genetic characterization. Amplified Fragment Length Polymorphism (AFLP) and DNA sequencing (mitochondrial small subunit rDNA and nuclear translation elongation factor 1-α) were used for genetic characterization of fungal isolates. Each isolate had a unique AFLP phenotype, and 30 markers were unique to only those highly virulent isolates. In all phylogenetic analyses, highly virulent isolates and non-pathogenic isolates separated into two clades with 100% bootstrap and posterior-probability support. Genetic analyses indicate molecular probes could be developed to differentiate highly virulent from non-pathogenic isolates of _F. oxysporum_. This research will help the development of molecular tools that enable nursery managers to implement timely and appropriate disease management practices.


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Predicted Impacts of Hard Pine Stem Rusts on Lodgepole Pine Dominated Stands in Central British Columbia: Were our Assumptions Valid?

Alex J. Woods

Abstract—In 1997, 30 one-ha stem mapped plots were located in randomly selected juvenile lodgepole pine (Pinus contorta Dougl. Ex Loud.) leading stands aged 15-20 years. The growth and yield model TASS was then used to predict volume losses at rotation. The average loss at rotation due to hard pine rusts in lodgepole pine dominated stands was estimated to be 7.2%. In order to model rust impacts over a rotation three basic modeling assumptions had to be made. This study reviews those three basic assumptions by re-assessing all trees within the 30 plots seven years later. The first assumption was that rust incidence would have stabilized in stands aged 15-20 years. I found that the incidence of both comandra blister rust (Cronartium comandrae Peck) (CBR) and western gall rust (Endocronartium harknessii (J.P. Moore) Y. Hiratsuka) (WGR) continued to increase. The greatest increase in CBR incidence occurred in stands that already had a high incidence while increases in WGR incidence were universally distributed over all 30 plots. The second assumption was that the voids created by rust killed trees would not fill with merchantable sized non-host trees by the time the lodgepole pine crop was ready to harvest. I found that the mean height of interior spruce is just over 1/3 the height of the lodgepole pine crop trees. The mean stocking of interior spruce is 243.7 ± 102.0 sph while that of lodgepole pine was 968.2 ± 196.2 sph. (95% CI). It is unlikely these spruce trees will be of merchantable size prior to harvest of the lodgepole pine crop. The third assumption was that CBR infected trees would die at an annual rate of 5% between the ages of 20 and 40 years. I found that after approximately 1/3 of that time period elapsing, 1/3 of the CBR infected trees were dead. Very few WGR infected trees have died. I also examined the relationship between the abundance of alternate host and the number of CBR infected trees. From this reassessment, I conclude that the three main modeling assumptions used to predict rust impact were valid and that the impact of hard pine rusts in juvenile lodgepole pine dominated stands in central British Columbia is approximately 7.2%.
A Paradigm Shift for White Pine Blister Rust: Non-Ribes Alternate Hosts of Cronartium ribicola in North America

Paul J. Zambino, Bryce A. Richardson, Geral I. McDonald, Ned B. Klopfenstein, and Mee-Sook Kim

Abstract—Naturally occurring Cronartium ribicola infections were discovered in August and September, 2004 on Pedicularis racemosa and Castilleja miniata in a mixed stand of white pine blister rust-infected whitebark pine (Pinus albicaulis) and western white pine (P. monticola) in northern Idaho, at Roman Nose Lakes, ca 30 km west of Bonners Ferry. Infections were confirmed by ITS rDNA sequencing of teliospores. The ability of these species to act as host was confirmed by laboratory inoculations using aeciospores from whitebark pine. Isolates recovered from P. racemosa after artificial inoculations were able to infect Ribes nigrum and were thus not specific to an alternate host genus, and also infected western white pine seedlings. Cronartium ribicola was detected on additional P. racemosa and C. miniata at the site in 2005. Infections were confirmed by ITS sequencing and transfer to R. nigrum. In 2005, exposure of local plants to local inoculum at a second site, ca 200 km to the south, caused infections on P. racemosa, and laboratory inoculations implicated C. rhexifolia from this site as a third non-Ribes alternate host. Identification of these alternate hosts may significantly alter our concepts of blister-rust hazard and epidemiology, particularly for those upper montane to subalpine stands where the newly identified hosts are abundant. The use of these hosts by the introduced pathogen also raises the possibility that fungal adaptation may be one factor allowing change in this dynamic and evolving pathosystem.

Introduction

Until 2004, Ribes spp. (Grossulariaceae) were the only known natural alternate hosts of Cronartium ribicola in North America (McDonald and others In Press). Other plant species (Pedicularis and Castilleja; Orobanchaceae) are alternate hosts for some strains or species of fungi causing blister rust of five-needled pines in Asia but not in Europe (Vogler and Bruns 1998; review in McDonald and others 2005), which is the presumed source for North American blister rust (Leppik 1970; review in Hunt 2003). Hiratsuka and Maruyama (1976) infected and obtained teliospores from C. miniata artificially inoculated with aeciospores from British Columbia, Canada. In contrast, other field and greenhouse inoculations using several species of Orobanchaceae and different sources of aeciospores did not infect non-Ribes hosts (Hunt 1984) or did not cause sporulation (Patton and Spear 1989). Natural infections of non-Ribes alternate hosts by C. ribicola had never been found in North America prior to 2004 (McDonald and others In Press).

This paper reports on studies that were conducted in 2004 (McDonald and others In Press) and 2005 on potential non-Ribes alternate hosts, following the discovery in late August, 2004 of telia typical of the genus Cronartium on four species of Orobanchaceae (Pedicularis racemosa, P. bracteosa, Castilleja miniata, and C. rhexifolia). These infections were in a mixed stand of white pine blister rust-infected whitebark pine (Pinus albicaulis) and western white pine (P. monticola) in northern Idaho (48.634109°N, 116.570817°W, elevation 1800 m), near the Roman Nose Lakes Campground, ca 30 km west of Bonners Ferry. Cronartium ribicola infections were confirmed for some non-Ribes species raising issues of the role of these alternate hosts to white-pine-blister-rust pathosystems of North America.

Materials and Methods

2004 Studies

1. To identify whether infections of Ribes hudsonianum var. petiolare, P. racemosa, P. bracteosa, C. miniata, and C. rhexifolia at the Roman Nose site were caused by C. ribicola, C. coleosporioides, or a hybrid species. Cronartium teliospores from natural infections were tested by direct PCR / DNA sequencing of ITS rDNA using ITS1+ITS4 primers (McDonald and others In Press).


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2. Plants of suspected alternate hosts and control *R. nigrum* cuttings (Heimburger clone) were artificially inoculated (conditions for infection: 100 RH at 19 C for 48 hr) (McDonald and others In Press). *Pedicularis racemosa* and *P. bracteosa* were inoculated with three sources of aeciospores from on-site whitebark pine; *C. miniata* (two plants, from a location near Coeur d’Alene, ID) were inoculated with one of the sources.

3. To test if pathogenicity is retained to *Ribes* and five-needled pines, urediniospores from lab-inoculated *P. racemosa* were used to inoculate *R. nigrum* cuttings (conditions for infection: 100 RH at 20°C for 24 hr), and teliospores from those cuttings were used to infect seedlings of western white pine in the cotyledon stage (conditions for infection: 100 RH at 16°C for 48 hr after basidiospores were cast) (McDonald and others In Press).

4. To test for regular meiosis, germinating teliospores were examined for evidence of four meiotic products using scanning electron microscopy (McDonald and others In Press).

2005 Studies

1. Samples of rust from additional plants of *R. hudsonianum* var. *petiolare*, *P. bracteosa*, and *C. miniata* were sequenced.

2. Samples of rust from *P. racemosa* from Roman Nose were used for direct inoculation of *R. nigrum*, to recover strains for further DNA characterization.

3. In June, aeciospores from western white pine from a second site (Freezeout Saddle, 47.00885°N, 116.00846°W; near Clarkia, ID) were dusted onto local plants of *P. racemosa*, *P. bracteosa*, *C. rhexifolia*, and *C. occidentalis* to allow natural infection processes.

4. Plants of *P. racemosa*, *P. bracteosa*, *C. rhexifolia*, and *C. occidentalis* from the site near Clarkia were removed from the field and inoculated with *C. ribicola* under laboratory conditions.

Results

1. All rust collections were either *C. ribicola* or *C. coleosporioides*, with no hybrids detected at 27 ITS sites that differ between the species.

2. The 11 natural infections tested from *P. racemosa* were all *C. ribicola*. This plant species had frequent infections in both years, and has a morphology that closely resembles the Asian alternate host, *P. resupinata*.

3. The five *P. bracteosa* infections tested were *C. coleosporioides*.

4. *Castilleja miniata* hosted both rusts, with two of seven infections being *C. ribicola*.

5. *Castilleja rhexifolia* was identified as a host by laboratory inoculation. The infected plant produced urediniospores and teliospores identified as *C. ribicola* by ITS sequencing. The one natural infection tested was of *C. coleosporioides*.

6. All aeciospores sources from Roman Nose whitebark pine produced uredinia and telia on potted *P. racemosa* and *C. miniata*, and on leaf cuttings of *R. nigrum*.

7. *Pedicularis racemosa*-infecting isolates grown to produce uredinia and telia on *R. nigrum* caused infections on western white pine seedlings, indicating that these isolates lack specificity to alternate host genus and retain the capacity to complete their lifecycle.

8. Scanning electron microscopy indicated that germinating teliospores bear four sterigmata and basidiospores, suggesting that meiosis is regular and that nuclei migrate to different basidiospores, as expected for *C. ribicola* and other macrocyclic, heterothallic rusts.

9. *Pedicularis racemosa* at the site near Clarkia that had been dusted with natural spores became infected with *C. ribicola*, as confirmed by ITS sequencing, so the ability of this rust fungus to use this host is not restricted to the Roman Nose site (near Bonners Ferry, ID).

10. Most field-inoculated plants of species other than *P. racemosa* were lost from various causes, so ability of *C. ribicola* to infect these hosts could not be determined. Plants in laboratory inoculations also had high mortality, indicating that optimal conditions for plant maintenance and inoculation/infection had not been obtained. More studies will be needed to evaluate regional differences in blister rust infection of potential non-*Ribes* alternate hosts.
11. For the successful *C. miniata* and *C. rhexifolia* inoculations, only one to two leaves or floral bracts of similar age out of the entire plant showed infection.

**Discussion**

These studies have demonstrated that non-*Ribes* species are being used as alternate hosts at an upper montane site with a short, cool growing season. The use of *Pedicularis* and *Castilleja* spp. as alternate hosts in North America could have a major impact on our understanding of disease epidemiology, site-specific risks for pine infection, and naturalization/adaptation in blister-rust pathosystems. Assessment of the importance of these alternate hosts will depend on geographic distributions of such non-*Ribes* alternate hosts, rates of infection, environmental conditions and host developmental physiology that affect windows for infection, and interactions with genetic structure of *C. ribicola*. Testing interactions with rust populations will require artificial inoculations; however, short windows of receptivity of tissues to infection and other characteristics of species in the hemiparasitic Orobanchaceae may make such studies difficult. Negative results from inoculations are not necessarily conclusive.

The lack of previous reports of natural alternate hosts in the Orobanchaceae and the mixed results of previous attempts at artificial inoculation of these species (Hiratsuka and Maruyama 1976; Hunt 1984; Patton and Spear 1989) could have been caused by differences in environmental or experimental conditions, genetic differentiation of rust and/or host populations, local adaptation via either standard genetic mechanisms or phenotypic plasticity, or even a separate, cryptic introduction. Also, prior to the advent of easy identification by molecular techniques, some *C. ribicola* infections on Orobanchaceae may have been mistakenly ascribed to *C. coleosporioides* (see Farr and others 1995 for listed hosts for this species).

ITS sequence data provided no evidence that hybridization between species was the source for *C. ribicola* that utilizes the Orobanchaceae; however, introgression with *C. ribicola* could mask past hybridization events with other Cronartium species. In contrast, Joly and others (2004) and Hamelin and others (2005) have demonstrated that hybrid aecia between *C. ribicola* and *C. comandrae* (the Comandra rust pathogen) occur on limber pine in British Columbia, although they have not determined alternate hosts for these natural hybrids, nor their persistence in nature.

Our reported detection of *C. ribicola* on Orobanchaceae, the finding of natural hybrids between *C. ribicola* and a rust of hard pines (Joly and other 2004; Hamelin and others 2005), and recent expansion of blister rust into new pine hosts and environments indicate that blister-rust pathosystems in North America are dynamic. The blister-rust fungus could be changing and adapting by multiple mechanisms, even as hosts are responding to the presence of the recently introduced pathogen. We expect that the current report is only the first of many shifts in the blister-rust paradigm that will be revealed through more critical examination of rust and host populations, the responses of blister-rust fungi to different environments, and genetic and epigenetic mechanisms of adaptation in *C. ribicola* and its hosts.

**References**


Thirty-three people attended the dwarf mistletoe committee lunch, with Fred Baker serving as Chairman.

**Genetics**

**Resistance of Jeffrey pine to western dwarf mistletoe.**

Genetic Resource Program personnel, with support from Forest Health Protection, in the Pacific Southwest Region (California) continue to pursue sources of ponderosa and Jeffrey pine resistant to western dwarf mistletoe (*Arceuthobium campylopodum*). In March of 2004 an outplanting study was installed to test the genetic resistance of selected Jeffrey pines. The seedlings were planted under heavily infected pines in Nevada Beach Campground which is on the east side of Lake Tahoe at an elevation of 6200 feet. The site has a very well drained and droughty sandy soil.

There were a total of 500 Jeffrey pine test seedlings planted in 10 blocks in the spring of 2004. The spacing was 6-foot square. In cooperation with Dave Fournier, Scott Parsons and the Basin, we've used drip irrigation and Vexar tubing to maintain survival and provide protection. The campground host and Recreation folks have been essential in protection as well. Survival as of August this year (2005) was about 92%, with most of the mortality occurring in two blocks.

There are 10 open pollinated, wild-stand parent trees represented. Five parents were selected for their apparent resistance to dwarf mistletoe and five were selected to act as susceptible controls. All were selected on either the Beckwourth or Milford Ranger Districts of the Plumas National Forest. Block 1 is a demonstration block with the five siblings from each parent tree planted in rows. The remaining blocks are completely randomized.

The strongest comparison will be between the two selection groups: control and resistant. The expected outcome will be for control seedlings to show significantly higher rates of infection than our candidate resistant selections. However, we'd love for some patterns to show up demonstrating varying levels of resistance by seedling families (representing individual selected parent trees). Time will tell.

**Paul Stover, Zone Geneticist, R5, Genetic Resource Program, and John Pronos, Forest Health Protection- Stanislaus National Forest**

**Surveys**

Permanent plots for measuring spread and impact of Douglas-fir dwarf mistletoe in the Southern Oregon Cascades, Pacific Northwest Region: Results of the ten year remeasurement.

Eleven permanent plots to measure spread and impact of Douglas-fir dwarf mistletoe were installed in the Southern Oregon Cascade Mountains on the Rogue River-Siskiyou and Umpqua National Forests in 1992. One plot was dropped in 1997. In 2002 nine of the ten remaining plots were remeasured. A wildfire fire burned through the tenth plot in 2002. It was remeasured in 2003. Data on crown scorch and bole char were collected in this plot to determine whether it will affect survival or dwarf mistletoe infection levels. After ten years heavily infected Douglas-fir had less growth and higher mortality than uninfected or lightly infected Douglas-fir. The number of infected Douglas-fir increased nineteen percent. The majority of newly infected Douglas-fir were within 25 feet of previously infected Douglas-fir. The effects of dwarf mistletoe infection were particularly great in small Douglas-fir. During the ten years between measurements dwarf mistletoe spread into more small diameter (less than 12 inch DBH) and understory Douglas-fir than into large Douglas-fir. Infected small diameter and understory Douglas-fir had less diameter growth and higher mortality than large Douglas-fir. The data will be used to validate the Forest Vegetation Simulator Model. A full report will be available when the analysis is completed. SWOFIDSC-05-03.
**Dwarf mistletoe broom development in mature Douglas-fir trees: A retrospective case study.**

This study was motivated by our continued interest in managing Douglas-fir dwarf mistletoe for wildlife habitat. The objective was to increase understanding of the way dwarf mistletoe brooms develop in relatively large mature Douglas-fir. Specifically, 1) the relationships among broom size, age, number, broom type and broom volume rating, 2) how long the Douglas-fir, particularly heavily infected ones, have survived with the current level of infection, and 3) the sequence of initiation, development and spread of brooms within the crowns. We collected data on thirty infected Douglas-fir on four sites. Cross-sections cut from each infected branch and broom were used to estimate the age of branches, the age when they became infected, and the age of each broom. There was a significant positive relationship between broom size and broom age. The oldest, largest brooms that are used most by wildlife were least common, and young, small brooms were most numerous. The average size of brooms was smaller on heavily infected trees. This suggested that heavy infections might not necessarily provide the greatest number of large brooms useful to wildlife. We also compared ages and volumes of brooms in our study with the volumes of brooms containing spotted owl nests from a study by Martin et al (WIFDWC 1992). The results suggested that under the right conditions brooms suitable for nesting might develop relatively rapidly. SWOFIDSC-04-03.

**Miscellaneous________________________**

The black spruce FIDL is in the final throes of review. Fred Baker, Utah State University

According to Brian Geils, *Mistletoes of North American Conifers*, RMRS-GTR-98 is available. View at:


There were many sightings of the mistletoe man (Bob Mathiasen) in and around Redding while he was on sabbatical last January through May. Several reports of Bigfoot came in during the same five months. Coincidence???? I think not! (See below)
The Hazard Tree Committee met for lunch on Tuesday, September 27 with 48 people in attendance. Four topics were presented and discussed as described below.

Fred Baker and Region 4 FHP have submitted a Special Technology Development Project proposal for FY 2006 to create an on-line instruction course in hazard tree management. The course is aimed at a broad audience to include managers of recreation sites and concessionaires. Fred has considerable experience in developing these modules and showed examples of some that already are available.

Mary Lou Fairweather showed photos of recent tree failures that she was personally involved with. One of the failures occurred during the winter at a cabin near Flagstaff that her family uses, and another took place at a campground while Mary Lou and family were camping.

John Pronos gave an update on the International Tree Failure Database (ITFD). This program currently has over a thousand trained cooperators but only 200 or so new tree failures have been reported since the database began collecting information in October, 2004. Over 4000 tree failure reports from the California Tree Failure Reporting Program have been migrated to the ITFD.

John Pronos described a hazard tree evaluation of a mature giant sequoia that he helped complete. The sequoia had a severe fire scar that was 25 feet wide and 60 feet high. It leaned toward a kiosk at the Big Stump entrance station to Kings Canyon National Park. Park officials closed the entrance station and are trying to decide what further actions to take. It is a challenge to examine trees this large, which have bark up to a foot thick at the base.
The annual meeting of the Nursery Pathology Committee was held during the afternoon of September 26th. There were 12 people in attendance, about half of whom provided information on projects and disease problems involving forest nurseries during the past year. Some of the highlights included:

1) Monitoring *Phytophthora* spp. within irrigation water has become necessary due to termination of chlorination due to high costs at an Oregon nursery.

2) Problems involving *Phytophthora cinnamomi* and *P. lateralis* continue at the Dorena Tree Improvement facility in Oregon.

3) New investigations have been initiated on cypress canker caused by *Seiridium cardinale* on western red cedar in Oregon.

4) Efforts have been continued involving effects of pre-plant soil fumigation on soil pathogen populations at several nurseries.

5) *Cylindrocarpon* (primarily *C. cylindroides*) continues to be a problem at the Weyerhauser Mima Nursery. This disease is especially a problem on bareroot Douglas-fir. Attempts are being made to control this disease with fluorescent pseudomonads.

6) Recent work at the Rocky Mountain Research Station (USDA Forest Service) on nursery isolates of *Fusarium oxysporum* indicates that highly virulent and non-pathogenic isolates are easily identified using molecular markers. This work lays the foundation for developing molecular probes that can be used for pathogen diagnosis in plants as well as estimating soil populations of the pathogen in nursery soil.

7) Dieback disease caused by *Sirococcus conigenus* was very severe in several nurseries on the West Coast this past spring. Damage occurred on both bareroot and container Douglas-fir. Severity of this disease was related to amount and duration of spring moisture.

8) Fusarium root disease, caused primarily by *F. oxysporum*, is a continuing problem in 1-0 bareroot conifers at the Coeur d'Alene Nursery. Soil fumigation with dazomet reduces, but does not eliminate pathogen propagules; pathogen levels tend to increase and seedling infection and mortality often results, especially during the first growing season.

9) Wilt/decline of *Acacia koa* in Hawaii has become a very serious disease, affecting seedlings as well as larger trees. Investigations are ongoing concerning disease etiology, distribution, spread, intensity, and preliminary efforts involving screening different families for disease resistance. The putative cause of the disease is *Fusarium oxysporum*, although disease severity is probably related to site, soil, and moisture conditions.
Twenty-four members attended the Root Disease Committee meeting held Thursday, September 29, 2005 at 0700.

This is Ellen Goheen’s last meeting as committee chair. Ellen has held the position since the Whitefish WIFDWC meeting in 1995 and has asked Brennan Ferguson to accept the Chairmanship for an unspecified length of term based on his willingness to serve. Members in attendance approved Brennan’s appointment.

The meeting format included a presentation by Rona Sturrock on her most recent research on Phellinus weirii, a discussion regarding outcomes of a field excursion to northern California to look at annosus root disease, and a round robin where participants offered results, current studies, and other information related to root disease biology and management in western North America.

An analysis of ITS sequences from almost 100 P. sulphurascens isolates collected in BC reveal the occurrence of two distinct ITS ‘types’. The team’s hypothesis that P. sulphurascens homokaryons could be distinguished from heterokaryons on the basis of having only one vs. both ITS types may not be correct because some heterokaryons could be homoallelic for one ITS type. The team will conduct work to clarify this issue. As part of its research on Phellinus spp. diversity and phylogeny, the team is also collecting and analyzing Phellinus species and P. sulphurascens and P. weirii isolates from other regions of the world including Japan, China and Russia. Finally, Rona has undertaken a review and synthesis about ‘climate change and forest diseases in Canada’. She is in the initial stages of finding and collecting literature and would appreciate ideas and information from others regarding this potentially ‘huge’ subject area.

Discussion_________________

Annosus root disease

Much of the committee meeting discussion centered around various aspects of managing annosus root disease caused by Heterobasidion annosum. A small group of pathologists met in Susanville California in June to view p-type and s-type annosus root disease impacts in the field and to discuss disease management strategies and challenges. This ad-hoc field trip was the brain child of John Guyon (USDA FS Utah) and Blakey Lockman (USDA FS Montana), among others, and was hosted by Bill Woodruff (USDA FS California). A variety of disease conditions were viewed. Permanent plots established by Mike Srago in the 1960s were visited. These plots continue to be monitored. Some of the many challenges to managing annosus root disease discussed were:

Identifying disease thresholds that change recommendations for stump treatments.
Survey timing and techniques.

Managing annosus root disease in mixed species stands, particularly those containing both white fir and other Abies species.

Using Sporax® in true fir stands.

Recommendations for disease treatments in stands on a management trajectory for wide spacing and low stocking.

Annosus root disease management is often the subject of appeals on USDA Forest Service and USDI Bureau of Land Management lands. Appellants object to the use of Sporax® to prevent infection of cut stump surfaces or use concerns about potential disease impacts to prevent salvage or thinning projects. Kerry Britton (USDA FS-WO) reported at the committee meeting that the contract has been awarded for a final Pesticide Risk Assessment for Sporax®; the document is expected to be completed late this fall. Related to issues regarding use of Sporax® in the context of recent changes in environmental assessments, Dave Bakke with the Pacific Southwest Region is preparing a white paper to help guide resource managers in decision making. John Kliejunas (USDA FS California) is doing a study to look at infection by H. annosum of stumps resulting from fire-killed trees. Bill Otrosina is continuing work with Matteo Garbelotto (UC Berkeley) on population structure of H. annosum. In fine WIFDWC tradition, forest pathologists do indeed continue to “give a damn about Fomes annosus.”

Round table

With buses arriving for the field trip, the round table discussion was fairly quick. Kathy Lewis (UNBC) continues a variety of research on Inonotus tomentosus including relative importance of asexual versus sexual reproduction by the pathogen. Many scientists in western North America are actively working on many aspects of Armillaria root disease and an understanding of Armillaria species. Surveys and permanent plot work are being done by Jim Worrell (USDA FS Colorado) and Jim Blodgett (USDA FS South Dakota). Mee Sook Kim and Ned Klopfenstein (USDA FS RMRS Idaho) and their colleagues are looking at AFLP patterns of Armillaria species at the landscape level along with trying to understand smaller scale ecological patterns.

Greg Filip (USDA FS Oregon) announced that the IUFRO meeting of the Root and Butt Rot working party will be held in August 2007 in California and Oregon. This meeting will be followed (in time and geographical sequence) by the IUFRO Phytophthoras in Forest Ecosystems working party meeting.

Thanks for many years of good discussions, productive panels and WIFDWC workshops, Ellen.
Business Meeting Minutes

53rd WIFDWC 2005 Jackson, Wyoming

Mary Lou Fairweather, Presiding Chair

Previous Minutes

2004 Business Meeting Minutes are approved as circulated, moved by Walt Thies, and accepted by the membership by voice vote.

Nominations

The railroad committee, made up of Ellen Goheen, Brian Geils and Alex Woods nominated Kathy Lewis as the 2006 Chairperson of WIFDWC and Marcus Jackson as secretary. John Schwandt will continue as treasurer. Rona Sturrock was nominated to be the new WIFDWC historian and Brennan Ferguson was nominated to be the Achievements Award Chairman. All were accepted by voice vote. Blakey Lockman was selected by Kathy Lewis as the 2006 program chair.

Future Meetings

Alex Woods reported on preparations for the 2006 WIFDWC in Smithers, B.C. The meeting will be held September 11-15, 2006. Mary Lou reported on development of the 2007 meeting to be held in Arizona, which will be held in either Sedona or Flagstaff. She received a vocal support for the idea of holding it in October of that year. Several potential locations for the 2008 meeting were discussed, including the Black Hills of South Dakota, North Dakota, Washington, Durango, Colorado, and Pingree Park, Colorado and Montana. Montana was selected by poll of the membership.

Old Business

Fred Baker reported on a funding shortfall for the CD of the collected past Proceedings of WIFDWC. The pricing and cost of the Proceedings CD was based on all WIFDWC members purchasing the CD, and as of the meeting only a fraction of the WIFDWC membership had purchased the CD.

Treasurer Report

John Schwandt

We had one of the largest meetings ever, with 98 people attending this year’s meeting (including 16 grad students, 7 retirees, and 64 regular members). We also had 11 spouses/guests join us. The table below is a summary of transactions for the WIFDWC account from 4/30/04 to 12/1/2005. For those that need it for travel, our Federal Tax Id. number is: #91-1267879

New Business

Motions

A motion was made to regularly put all future proceedings on the web, similar to what has been done with 2004 Joint Proceedings. The motion was discussed and then tabled in favor of a motion to research how peer-reviewed journals view prepublication in the WIFDWC Proceedings to see if prepublication would inhibit subsequent publication in other Journals. This motion was passed by voice vote and Brian Geils, Bill Jacobi and John Guyon were assigned to a committee to research this question.

A motion was made to simplify the format for the Proceedings to make writing articles for the Proceedings less onerous. The current secretary, John Guyon, expressed a preference to keep the 2005 Proceedings in their current format. The motion was passed by a voice vote and the 2006 Secretary, Marcus Jackson was assigned to prepare a simplified format for the 2006 Proceedings.

A motion was made to accept abstracts of all posters from the poster session for inclusion in the 2005 Proceedings. The abstracts can include an internet link to the full poster if the authors desire to include one. The motion was passed by a voice vote.
2005 Treasurer Report

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<tr>
<td>(Balance from 04 meeting = 100.00)</td>
<td></td>
<td></td>
<td>$2,269.66</td>
</tr>
<tr>
<td><strong>North Am. Tree Failure DB meeting (3/05)</strong></td>
<td>675.12</td>
<td>1,694.54</td>
<td></td>
</tr>
<tr>
<td><strong>Hazard Tree Balance as of 12/1/05</strong></td>
<td>675.12</td>
<td></td>
<td>$1,694.54</td>
</tr>
<tr>
<td><strong>Total Bank Balance as of 12/1/05 (WIFDWC + Hazard Tree)</strong></td>
<td></td>
<td></td>
<td>$11,600.96</td>
</tr>
</tbody>
</table>

A motion was made for the WIFDWC leadership to prepare a letter to the Chief of the USDA Forest Service and current Forest Experiment Station Leadership addressing the current cuts in USDA Forest Service pathology research positions and funding. The motion was discussed and passed by voice vote. Dave Shaw was selected to Chair a committee to prepare this letter, and Greg Fillip, Brian Geils and Bill Jacobi were assigned to assist Dave.

A motion was made to roll the costs of preparing the CD of previous Proceedings into the next year’s registration fees for those who have not purchased a copy of the CD. This motion was discussed and then withdrawn in favor of a motion to roll the cost of the CD into the next 3 year’s registration fees, except for those who have already purchased the CD. Motion passed by voice vote.
This past year WIFDWC lost Norman Eugene Alexander. Norm retired as a faculty member at British Columbia Institute of Technology, BCIT. Norm was known to many of us for his quick wit and sense of humor. He could cut through lots of rhetoric to get to the heart of an issue.

I had the good fortune of corresponding with Norm several times a year by e-mail. His observations were always thought provoking, and I enjoyed discussing issues with him. I would like to share one of the last e-mails from Norm. I hope you enjoy it as much as I did.

Gentlemen:

I was doing a re-read of a book on ecology when I came across this gem. It confirms a suspicion that lurches in my mind, especially after attending a recent forest health conference. I thought you people would be interested. This quotation is taken out of context, as are all quotations, but it came at the end of a long chapter that described the development of ecology and it comes almost as a conclusion.

"Part of the problem arises from what R.P. McIntosh (1980b) calls "hit and run entrants" into ecology from other sciences, especially engineering, mathematics, and physics. They have left behind a trail of models that, as L.B. Slobodkin (1974) observed, "develop biological nonsense with mathematical certainty." E.C. Pielou (1981:17) has stated that "mathematical modeling is rapidly becoming an end unto itself .... As a result the whole body of ecological knowledge and theory has grown top heavy with models."

He concludes the section with:

"But, as Pielou (1981:30) admonishes: too much should not be expected of them. Modeling is only a part, a subordinate part, of ecological research," to be used, as Skellem (1972:27) warns, with 
"enlightenment and eternal vigilance on the part of both ecologists and mathematicians." (Smith, Robert Leo. 1986. Elements of Ecology: Second Edition)
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

<table>
<thead>
<tr>
<th>Year</th>
<th>Chair</th>
<th>Members</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999</td>
<td>J. Byler</td>
<td>W. Littke</td>
</tr>
<tr>
<td>2000</td>
<td>W. Littke</td>
<td>B. van der Kamp</td>
</tr>
<tr>
<td>2001</td>
<td>B. van der Kamp</td>
<td>R. Sturrock</td>
</tr>
<tr>
<td>2002</td>
<td>R. Sturrock</td>
<td>G. Filip</td>
</tr>
<tr>
<td>2003</td>
<td>G. Filip</td>
<td>D. Goheen</td>
</tr>
<tr>
<td>2005</td>
<td>B. Ferguson</td>
<td></td>
</tr>
</tbody>
</table>

* Year elected

Elected to replace “unknown”

Appointed by Chair to filling remaining position.

* The social achievement award retired in 1997.
2005 Recipient: Outstanding Achievement Award

Walter G. Thies

Nomination Narrative

Walt Thies is this year’s recipient of the Western International Forest Disease Work Conference Outstanding Achievement Award for his significant contributions to forest pathology research and the high quality communication of his research results. Except for a few years with USDA Forest Service Insect and Disease Control in Region 6, Walt has spent his entire career as Research Forest Pathologist, Pacific Northwest Research Station and has specialized in studying forest tree root diseases, especially laminated root rot and black stain root disease. Walt has demonstrated a commitment to excellent study design, meticulous, detailed data collection, and careful, complete analysis in the process of substantially advancing our understanding of root pathogen biology and management. He has been particularly noted for his insistence on establishing and maintaining long-term field studies. Walt is an exceptionally hard worker who has never shied away from difficult study sites or challenging research questions.

Walt’s publication record in forestry related journals is impressive as are his contributions to WIFDWC Proceedings over the years. He is especially known for co-authoring, along with Rona Sturrock of the Canadian Forest Service, the publication “Laminated Root Rot in Western North America.” This publication has become the “Phellinus bible” and has been extremely popular with all who are interested in the subject. It has been especially well-received and appreciated by land managers. Thousands of copies of this publication have been distributed by extension specialists and Forest Health Protection Service Centers.

Walt is a very willing and effective communicator with all who seek information on root diseases and especially likes to interact with on-the-ground forest managers. He enjoys discussing the subject with other scientists as well. Such discussions have been described as “very long but very fruitful.”
Past Annual Meeting Locations and Officers

Meetings and Officers, 1953–1989

<table>
<thead>
<tr>
<th>Annual</th>
<th>Year</th>
<th>Location</th>
<th>Chairperson</th>
<th>Secretary – Treasurer</th>
<th>Program Chair</th>
<th>Local Arrangements</th>
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<tbody>
<tr>
<td>1</td>
<td>1953</td>
<td>Victoria, BC</td>
<td>R. Foster</td>
<td></td>
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<tr>
<td>2</td>
<td>1954</td>
<td>Berkeley, CA</td>
<td>W. Wagener</td>
<td>P. Lightle</td>
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<tr>
<td>3</td>
<td>1955</td>
<td>Spokane, WA</td>
<td>V. Nordin</td>
<td>C. Leaphart</td>
<td>G. Thomas</td>
<td></td>
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<tr>
<td>4</td>
<td>1956</td>
<td>El Paso, TX</td>
<td>L. Gill</td>
<td>R. Davidson</td>
<td>V. Nordin</td>
<td></td>
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<tr>
<td>5</td>
<td>1957</td>
<td>Salem, OR</td>
<td>G. Thomas</td>
<td>T. Childs</td>
<td>R. Gilberston</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>1958</td>
<td>Vancouver, BC</td>
<td>J. Kimmey</td>
<td>H. Offord</td>
<td>A. Parker</td>
<td></td>
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<tr>
<td>7</td>
<td>1959</td>
<td>Pullman, WA</td>
<td>H. Offord</td>
<td>R. Foster</td>
<td>C. Shaw</td>
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<tr>
<td>9</td>
<td>1961</td>
<td>Banff, AB</td>
<td>F. Hawksworth</td>
<td>J. Parmeter</td>
<td>A. Molnar</td>
<td>G. Thomas</td>
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<tr>
<td>10</td>
<td>1962</td>
<td>Victoria, BC</td>
<td>J. Parmeter</td>
<td>C. Shaw</td>
<td>K. Shea</td>
<td>R. McMinn</td>
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<tr>
<td>11</td>
<td>1963</td>
<td>Jackson, WY</td>
<td>C. Shaw</td>
<td>J. Bier</td>
<td>R. Scharpf</td>
<td>L. Farmer</td>
</tr>
<tr>
<td>13</td>
<td>1965</td>
<td>Kelowna, BC</td>
<td>J. Bier</td>
<td>H. Whitney</td>
<td>R. Bega</td>
<td>A. Molnar</td>
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<td>14</td>
<td>1966</td>
<td>Bend, OR</td>
<td>C. Leaphart</td>
<td>D. Graham</td>
<td>G. Pentland</td>
<td>D. Graham</td>
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<tr>
<td>15</td>
<td>1967</td>
<td>Santa Fe, NM</td>
<td>A. Molnar</td>
<td>E. Wicker</td>
<td>L. Weir</td>
<td>P. Lightle</td>
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<tr>
<td>18</td>
<td>1970</td>
<td>Harrison Hot Springs, BC</td>
<td>R. Scharpf</td>
<td>H. Toku</td>
<td>A. Harvey</td>
<td>J. Roff</td>
</tr>
<tr>
<td>19</td>
<td>1971</td>
<td>Medford, OR</td>
<td>J. Baranyay</td>
<td>D. Graham</td>
<td>R. Smith</td>
<td>H. Bynum</td>
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<tr>
<td>20</td>
<td>1972</td>
<td>Victoria, BC</td>
<td>P. Lightle</td>
<td>A. McCain</td>
<td>L. Weir</td>
<td>D. Morrison</td>
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<tr>
<td>21</td>
<td>1973</td>
<td>Estes Park, CO</td>
<td>E. Wicker</td>
<td>R. Loomis</td>
<td>R. Gilbertson</td>
<td>J. Laut</td>
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<td>22</td>
<td>1974</td>
<td>Monterey, CA</td>
<td>R. Bega</td>
<td>D. Hocking</td>
<td>J. Parmeter</td>
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<td>23</td>
<td>1975</td>
<td>Missoula, MT</td>
<td>H. Whitney</td>
<td>J. Byler</td>
<td>E. Wicker</td>
<td>O. Dooling</td>
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<td>24</td>
<td>1976</td>
<td>Coos Bay, OR</td>
<td>L. Roth</td>
<td>K. Russell</td>
<td>L. Weir</td>
<td>J. Hadfield</td>
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<tr>
<td>26</td>
<td>1978</td>
<td>Tucson, AZ</td>
<td>R. Smith</td>
<td>D. Drummond</td>
<td>L. Weir</td>
<td>R. Gilbertson</td>
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<td>27</td>
<td>1979</td>
<td>Salem, OR</td>
<td>T. Laurent</td>
<td>T. Hinds</td>
<td>B. van der Kamp</td>
<td>L. Weir</td>
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<td>28</td>
<td>1980</td>
<td>Pingree Park, CO</td>
<td>R. Gilbertson</td>
<td>O. Dooling</td>
<td>J. Laut</td>
<td>M. Schomaker</td>
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<tr>
<td>29</td>
<td>1981</td>
<td>Vernon, BC</td>
<td>L. Weir</td>
<td>C.G. Shaw III</td>
<td>J. Schwandt</td>
<td>D. Morrison</td>
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<tr>
<td>30</td>
<td>1982</td>
<td>Fallen Leaf Lake, CA</td>
<td>W. Bloomberg</td>
<td>W. Jacobi</td>
<td>E. Hansen</td>
<td>F. Cobb</td>
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<td>31</td>
<td>1983</td>
<td>Coeur D’Alene, ID</td>
<td>J. Laut</td>
<td>S. Dubreuil</td>
<td>D. Johnson</td>
<td>J. Schwandt</td>
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<td>32</td>
<td>1984</td>
<td>Taos, NM</td>
<td>T. Hinds</td>
<td>R. Hunt</td>
<td>J. Byler</td>
<td>J. Beatty</td>
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<td>33</td>
<td>1985</td>
<td>Olympia, WA</td>
<td>F. Cobb</td>
<td>W. Thies</td>
<td>R. Edmonds</td>
<td>K. Russell</td>
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<td>34</td>
<td>1986</td>
<td>Juneau, AK</td>
<td>K. Russell</td>
<td>S. Cooley</td>
<td>J. Laut</td>
<td>C.G. Shaw III</td>
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<td>35</td>
<td>1987</td>
<td>Nanaimo, BC</td>
<td>J. Muir</td>
<td>G. DeNitto</td>
<td>J. Beatty</td>
<td>J. Kumi</td>
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<tr>
<td>36</td>
<td>1988</td>
<td>Park City, UT</td>
<td>J. Byler</td>
<td>B. van der Kamp</td>
<td>J. Pronos</td>
<td>F. Baker</td>
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<tr>
<td>37</td>
<td>1989</td>
<td>Bend, OR</td>
<td>D. Goheen</td>
<td>R. James</td>
<td>E. Hansen</td>
<td>A. Kanaskie</td>
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Bylaws were amended in 1989 to split the office of Secretary–Treasurer.
### Meetings and Officers, 1990–2003

<table>
<thead>
<tr>
<th>Annual</th>
<th>Year</th>
<th>Location</th>
<th>Chairperson</th>
<th>Secretary</th>
<th>Treasurer</th>
<th>Historian</th>
<th>Program Chair</th>
<th>Local Arrangements</th>
<th>Web Coordinator</th>
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<tr>
<td>38</td>
<td>1990</td>
<td>Redding, CA</td>
<td>R. Hunt</td>
<td>J. Hoffman</td>
<td>K. Russell</td>
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<td>40</td>
<td>1992</td>
<td>Durango, CO</td>
<td>D. Morrison</td>
<td>S. Frankel</td>
<td>K. Russell</td>
<td></td>
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<td>C.G. Shaw III</td>
<td>P. Angwin</td>
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<tr>
<td>42</td>
<td>1994</td>
<td>Albuquerque, NM</td>
<td>C.G. Shaw III</td>
<td>G. Filip</td>
<td>K. Russell</td>
<td></td>
<td>M. Schultz</td>
<td>D. Conklin</td>
<td>T. Rogers</td>
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<td>43</td>
<td>1995</td>
<td>Whitefish, MT</td>
<td>S. Frankel</td>
<td>R. Mathiasen</td>
<td>K. Russell</td>
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<td>R. Mathiasen</td>
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<td>44</td>
<td>1996</td>
<td>Vernon, BC</td>
<td>A. McCain</td>
<td>J. Beatty</td>
<td>J. Schwandt</td>
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<td>S. Campbell</td>
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<tr>
<td>45</td>
<td>1997</td>
<td>Durango, CO</td>
<td>D. Morrison</td>
<td>S. Frankel</td>
<td>K. Russell</td>
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</table>

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, 1994–2005

<table>
<thead>
<tr>
<th>Committee</th>
<th>Chairperson</th>
<th>Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dwarf Mistletoe</td>
<td>R. Mathiasen</td>
<td>1994–2000</td>
</tr>
<tr>
<td>Rust</td>
<td>J. Schwandt</td>
<td>1994, 2005</td>
</tr>
<tr>
<td>Disease Control</td>
<td>B. James</td>
<td>1995–2002</td>
</tr>
<tr>
<td>Nursery Pathology</td>
<td>B. James</td>
<td>2002–2005</td>
</tr>
</tbody>
</table>

aPete Angwin to become new chair in 2006
bBrennan Ferguson to become new chair in 2006
"Holly Keams to become the new chair in 2006, J.Schwandt was acting chair in 2005
bDisease Control disbanded and Nursery Pathology established in 2002
Judy A. Adams  
Last attended in 2005  
USDA Forest Service-FHTET  
2150 Centre Ave., Bldg A  
Fort Collins, CO 80526 USA  
(970) 295-5846  
jadams04@fs.fed.us

Paul Aho  
Retired-elected HLM in 1984  
Last attended in 1984  
223 W. 30th  
Corvallis, OR 97330 USA

Mike Albers  
Last attended in 2004  
1201 East Hwy. 2  
Grand Rapids, MN 55744 USA  
mike.albers@dnr.state.mn.us

Norm Alexander  
Retired-elected HLM in 1994  
Last attended in 1996  
5972 Glendale Drive  
Chilliwack, BC V2R 3A5 CANADA  
604-824-2156  
normalex@telus.net

Robert Anderson  
Last attended in 2000  
Requested to remain on roster in 2003  
Pacific Corp. Corp. Studies Unit U, University of Hawaii  
3190 Maile Way, Room 159  
Honolulu, 96822 USA  
(808) 956-9428  
robertan@hawaii.edu

Ed Andrews  
Retired—elected HLM in 1977

Stuart "Stuie" Andrews  
Deceased-elected HLM in 1973

Pete Angwin  
Last attended in 2005  
3644 Avtech Parkway  
Redding, CA 96002 USA  
(530) 226-2436  
pangwin@fs.fed.us

John Anhold  
Last attended in 2005  
Southwest Forest Science Complex 2500 S. Pine Knoll Drive  
Flagstaff, AZ 86001 USA  
928-556-2073  
janhold@fs.fed.us

Sue Askow  
Last attended in 2002  
UBC/CFS  
534 Bunker Road  
Victoria, BC V8Z 1M5 CANADA  
250-363-0738  
saskew@pfc.forestry.ca

Fred Baker  
Last attended in 2005  
Dept. of FRWS, Utah State University  
5230 Old Main Hill  
Logan, UT 84322-5230 USA  
(435) 797-2550  
fred.baker@usu.edu

Stan Barras  
Retired-last attended in 1996  
USDA FS-FIDR PO Box 96090  
Washington, DC 20090-6090 USA  
202-205-1596  
sbarras@fs.fed.us

Karen Bartlett  
Last attended in 2002  
School of Occupational and Environmental Hygiene  
2206 East Mall  
Vancouver, BC V6T 1Z3 CANADA  
604-822-6019  
kbartlett@interchange.ubc.ca

Dale Bartos  
Last attended in 1998  
USDA-FS, Rocky Mt. Res. Sta.  
860 North 1200 East  
Logan, UT 84321 USA  
435-755-3567  
dbartos@fs.fed.us

Kendra Baumgartner  
Last attended in 1998  
UC Davis, Dept. of Plant Path.  
One Shields Ave.  
Davis, CA 95616-8680 USA  
530-754-9894  
kbaumgartner@ucdavis.edu

Russell Beam  
Last attended in 2005  
885 Kline Dr.  
Lakewood, CO 80215 USA  
303-205-8575  
rdbeam@lamar.colostate.edu

Jerome Beaty  
Last attended in 2005  
USDA-FS 3160 N.E. 3rd Street  
Prineville, OR 97754 USA  
541-231-8942  
jbeaty@fs.fed.us

Jesse Bedwell  
Deceased-elected HLM in 1966

Robert Bega  
Deceased-elected HLM in 1985

Warren Benedict  
Deceased-elected HLM in 1966

Dale R. Bergdahl  
Requested to remain on roster in 2003  
Dept. of Forestry, Aiken Center  
University of Vermont  
Burlington, VT 05405 USA  
802-656-2517  
dbergdahl@uvm.edu

Frank Betlejewski  
Last attended in 2003  
2606 Old Stage Road  
Central Point, OR 97502 USA  
(541) 858-6127  
fbetlejewski@fs.fed.us

John Bier  
Deceased-elected HLM in 1967

Richard T. "Dick" Bingham  
Retired- elected HLM in 1975  
Last attended in 1975  
1127 American Ridge Dr.  
Kendrick, ID 83537-9504 USA

Peter V. Blents  
Requested to remain on roster in 2003  
Dept. Renewable Resources  
715 General Services Bldg  
Edmonton, AB T6G 2H1 CANADA  
(780) 492-0106  
peter.blents@ualberta.ca

James Blodgett  
Last attended in 2005  
Forest Health Management  
1730 Samco Rd  
Rapid City, SD 57702 USA  
605-394-6191  
jblodgett@fs.fed.us

Bill Bloomberg  
Deceased-elected HLM in 1990

Roy Bloomstrom  
Deceased-elected HLM in 1992

Pierluigi Bonello  
Last attended in 1999  
Dept. of Plant Path., Ohio State U.  
2021 Coffrey Road  
Columbus, Ohio 43210-1087 USA  
614-688-5401  
bonello.2@osu.edu

Bernard Bormann  
Last attended in 2000  
USDA FS, PNW Research Station  
3200 Jefferson Way  
Corvallis, OR 97331 USA  
(541) 750-7323  
bbormann@fs.fed.us

Clive Brasier  
Retired-elected HLM in 2004  
Last attended in 2003  
Forest Research Agency  
Alice Holt Lodge  
Farnham, Surrey GU10 $LH UK  
44 1420 536240  
crive.braisier@forestry.gsi.gov.uk

Nicholas Brazee  
Last attended in 2005  
1714 E. Spring St.  
Seattle, WA 98122 USA  
206-543-1486  
nbrazee@u.washington.edu

August 15, 2005
Kerry Britton
Last attended in 2005
Forest Health Protection
1601 N. Kent St.
Arlington, VA 22209 USA
(703) 605-5347
kbritton01@fs.fed.us

Fred Brooks
Last attended in 2001
American Samoa Community College
PO Box 5319
Pago Pago, AS 96799 USA
684-699-1394
fredbrooks@hotmail.com

John Browning
Last attended in 2005
505 N. Pearl St.
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<td><a href="mailto:welsh@unbc.ca">welsh@unbc.ca</a></td>
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<td>John Wenz</td>
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<td><a href="mailto:wenz@fs.fed.us">wenz@fs.fed.us</a></td>
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<td>Alex Woods</td>
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<td>Paul Zambino</td>
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<td>Wolf Ziller</td>
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**WIFDWC Members**

August 15, 2005
From your humble compiler and your Intermountain Region Host Crew, Peace, and Goodbye.

/s/ John