Proceedings of the 54th Annual Western International Forest Disease Work Conference

Dze L K’ant Friendship Centre
Smithers, British Columbia
October 2 to 6, 2006

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Committee Reports

Dwarf Mistletoe Committee
William Jacobi, Acting Chair

Root Disease Committee
Kathy Lewis, Acting Chair

Blister Rust Committee
Holly Kearns, Chair

Hazard Tree Committee
Pete Angwin, Chair

Business Meeting Minutes, 54th WIFDWC 2006 Smithers, British Columbia
Kathy Lewis, Chair

Past Annual Meeting Locations and Officers

WIFDWC Members

WIFDWC Honorary Life Members

WIFDWC Honorary Life Members (Complete List)

In Memoriam  David Schultz
WIFDWC 2006 Program
Western International Forest Disease Work Conference
October 2—6, 2006; Smithers, British Columbia

Monday, October 2

4:00—8:00  Registration and Social
           Dze L K’ant Friendship Centre (Native Friendship Centre)

Tuesday, October 3

7:00—8:30  Dwarf Mistletoe Committee Breakfast

8:30—9:00  Welcome
           Sybille Haeussler, Bulkley Valley Research Centre, Smithers, BC

9:00—10:15 Regional Reports

10:15—10:45 Refreshment Break

10:45—11:30 Keynote Address: 2005 Outstanding Achievement Award Winner Presentation
           Walt Thies, Pacific Northwest Research Station, Corvallis, OR
           2006 Outstanding Achievement Award Winners Announcement

11:30—12:00 Panel: Student Papers. Angie Dale, Moderator
           Frequency of Southwestern Dwarf Mistletoe and Mountain Pine Beetle in Colorado’s Northern Front
           Range Ponderosa Pine. Russell Beam (Presenter), Jennifer Klutsch, and William Jacobi,
           Colorado State University, Fort Collins, CO; and José Negrón, Rocky Mountain Research
           Station, Fort Collins, CO.
           A Preliminary Evaluation of Fuel Levels in Southwestern Dwarf Mistletoe and Mountain Pine Beetle
           Infested Ponderosa Pine. Jennifer Klutsch (Presenter), Russell Beam, and William Jacobi,
           Colorado State University, Fort Collins, CO; and José Negrón, Rocky Mountain Research
           Station, Fort Collins, CO.

12:00—1:30 Root Disease Committee Lunch

1:30—2:30 Panel: Student Papers Continued
           Technology Transfer and Forest Health. Amanda Crump (Presenter), William Jacobi, Colorado State
           University, Fort Collins, CO; and John Lundquist, Rocky Mountain Research Station, Fort
           Collins, CO.
           Assessment of Colletotrichum gloeosporioides as a Biological Agent for Western Hemlock Dwarf
           Mistletoe (Arceuthobium tsugense). Sue Askew (Presenter) and Bart van der Kamp, University
           of British Columbia, Vancouver, BC; and Simon Shamoun, Pacific Forestry Centre, Victoria,
           BC.
           Magnesium Chloride Dust Suppression Products and Roadside Environments. Betsy Goodrich
           (Presenter), William Jacobi, and Ronda Koski, Colorado State University, Ft. Collins, CO.

2:30—3:00 Refreshment Break

3:00—5:00 Panel: Aftermath of the Largest Mountain Pine Beetle Outbreak in Recorded History: What
           now? Silviculture and forest health issues. Kathy Lewis, Moderator
Silviculture and the Mountain Pine Beetle – What to do? K. David Coates, British Columbia Forest Service, Smithers, BC.

Premonitions of Post-Mountain Pine Beetle Pathogens. Bart van der Kamp, University of British Columbia, Vancouver, BC.

Landscape-level concerns after mountain pine beetle: Warren root collar weevil in regenerating pine stands. Brian Aukema (Presenter) and Matthew Klingenberg, Pacific Forestry Centre and University of Northern British Columbia, Prince George, BC; Carolyn Stevens, British Columbia Ministry of Forests & Range, Burns Lake, BC; and Staffan Lindgren, University of Northern British Columbia, Prince George, BC.

Seedling Tree Survival in Dothistroma and Mountain Pine Beetle Attacked Lodgepole Pine Stands. Frank Doyle, Wildlife Dynamics Consulting, Telkwa, BC.

5:30—7:00 Blister Rust "Happy Hour" Committee Meeting

Wednesday, October 4

7:00—8:30 Hazard Tree Committee Breakfast
8:30—5:00 Field Trip: Banner Mountain/Canyon Creek, McKendrick Pass, Nilkitkwa Provenance Trial, Nichyeskwa Creek, Babine Lake Road
6:30—9:00 Poster Session, Cedar Welsh, Coordinator
    Ice Cream Social

Thursday, October 5

8:00—10:00 Panel: Climate Change: We've Got No Crystal Balls! Rona Sturrock, Moderator
    Certainties and uncertainties of climate change: global change and local impacts. Andreas Hamann, University of Alberta, Edmonton, AB.
    Changing the Climate, Changing the Rules: Global Warming and Insect Disturbance in Western North American Forests. Allan Carroll, Pacific Forestry Centre, Victoria, BC.
    Predicting effects of climate change on Swiss needle cast disease severity in Pacific Northwest forests. Jeffrey Stone and Leonard Coop, Oregon State University, Corvallis, OR.
    Climate Change Effects on Forest Diseases: an Overview. Rona Sturrock, Pacific Forestry Centre, Victoria, BC.

10:00—10:30 Refreshment Break

10:30—12:30 Panel: Foliage Diseases: The Bare-Naked Trees!, Jeffrey Stone, Moderator
    A Brief Summary of Historical Forest Insect and Disease Survey Findings on Pine Needle Casts in British Columbia. Brenda Callan, Pacific Forestry Centre, Victoria, BC.
    Genetic diversity in the fungal pathogen Dothistroma septosporum. Angie Dale and Kathy Lewis, University of Northern British Columbia, Prince George, BC.
    What effects will a changing climate have on lodgepole pine in British Columbia? Alex Woods, British Columbia Forest Service, Smithers, BC; and Greg O’Neill, British Columbia Forest Service, Vernon, BC.

12:45—5:30 Field Trip: Boulder Creek, Hagwilget Bridge, Kispiox Totems, Poplar Park Road, Helen Lake Road, 'Ksan Historical Site

6:00—8:00 Banquet: 'Ksan Historic Village, Hazelton
Friday, October 6

8:00—10:00  Panel: Special Papers, Blakey Lockman, Moderator
Using the New Excel-based Stand Visualization Add-In Software to generate images depicting forest
health issues. Lori Trummer (Presenter), Alaska Region, Forest Health Protection, Anchorage,
AK; Paul Hennon, Alaska Region, Forest Health Protection, Juneau, AK; James McCarter,
University of Washington, Seattle, WA; Robert McGaughey, Pacific Northwest Research
Station, Seattle, WA.
A new Phytophthora causes “Mal del cipres” in the Patagonian Andes of Argentina. Alina Greslebin,
Centro de Investigació n y Extensió n Forestal Andino Patagónico—CIEFAP - Esquel,
Chubut, Argentina; and Everett Hansen (Presenter), Oregon State University, Corvallis, OR.
Growth impacts of western hemlock dwarf mistletoe on western hemlock in an old-growth Douglas-
fir western hemlock forest. David Shaw (Presenter), Manuela Huso, and Howard Bruner,
Oregon State University, Corvallis, OR.
Spread and Development of Phytophthora ramorum in a California Christmas Tree Farm. Gary
Chastagner (Presenter), Kathy Riley, and Norm Dart. Washington State University, Puyallup,
WA.
Molecular Techniques: What Can They Tell Us About Dothistroma Needle Blight? Rosie Bradshaw,
Massey University, Palmerston North, New Zealand.

10:00—12:00  Business Meeting/2006 WIFDWC Meeting Conclusion
Dothistroma Meeting followed WIFDWC through Saturday October 7
Welcome everyone to Smithers for the meeting of the 54th Annual Western International Forest Disease Work Conference. A special welcome to those who have traveled from far away – which is pretty much everyone, but of special note we have folks attending from Britain, Czech Republic, New Zealand, and of course Bill Jacobi and students who drove for four days in a van.

Before we start the meeting I wish to acknowledge that the meeting hall and the town of Smithers is in the traditional territory of the Wet’suwet’en First Nations (People of the Lower Hills) and during our field trip we’ll be crossing into traditional territory of the Gitsan - so thank you to those nations for allowing us to hold our meeting in this beautiful location.

I would like to point out that this year we have 18 people attending from British Columbia Ministry of Forests, which is probably a record number and it would be great (wouldn’t it Harry?), if we could continue having strong attendance at WIFDWC by BC Forest Service folks. There are five consultants, four from the forest industry, 16 US Forest Service, 12 university professors, 14 graduate students, and two Canadian Forest Service attendees for a total of 71 meeting participants.

Thank you to the organizing committee – Alex Woods, Judy Adams, John Schwandt, Marcus Jackson and Blakey Lockman. In addition, thank you Erin Havard, Karen McEwan, Loreen Cavalin, Stefan Zeglan, Dorothy Beneke, Dave Weaver, Alan Banner, Erin Hall, Sarah Berger, and Smithers Senior Secondary for their contributions to local arrangements. And, finally, thank you to Alex’s family for putting up with him over the past month or so.
Keynote Address—2005 Outstanding Achievement Award Winner

Walt Thies

1. Thanks to the Conference. I want to thank the Conference for the honor you bestowed on me at last year’s meeting by presenting me with the 2005 WIFDWC Outstanding Achievement Award. It was a very pleasant surprise. I can think of no greater honor than to be so recognized by colleagues, especially folks whom I respect and enjoy working with.

2. Thanks to Gail. Last year when Don Goheen presented me with this award, he rightly introduced my wife Gail first and credited her with any success I may have had over the years. This is certainly true; she has worked beside me without adequate credit since we were in graduate school. What Don may not have known was that just prior to the meeting we celebrated the 40th anniversary of our trial marriage. More importantly, on that occasion Gail agreed to renew my contract for another 40 years but insisted that at the conclusion of that contract I would have to make a firm commitment.

3. Thanks to Jim, Earl, and Rona. I have many folks to thank for helping me make my career productive and fun; however, if I start naming them all and listing their contributions it will seem that I am trying to stretch this keynote address to an after dinner talk. I want to publicly acknowledge the impact of three individuals. I want to thank Jim Hadfield who first hired me into the Pacific Northwest Region, Insect and Disease Control unit, the precursor of present day Forest Health Protection. This was a good introduction to the Pacific Northwest; I was able to visit nearly every District in the Region and experience the diversity of forests and forest pests. I thank Earl Nelson, who hired me into the PNW Research Station, instilling in me a rather conservative approach to research with great emphasis on keeping research questions well-grounded. And, in British Columbia, my thanks to my fellow root disease researcher (read, laminated root rot), Rona Sturrock. Rona has been a strong sounding board for ideas and, through a keen sense of editing, has helped improve many of my papers. Today, there are so few of us working on root disease research that Rona often gets to do double duty. I send her papers for a peer review and the journals send her my submissions with her changes already included. I am fortunate that she is a discriminating researcher, good editor, a patient colleague, and a valued friend.

While working in Region 6, I had decided early on that laminated root rot (LRR) presented a hopeless situation. In the early 1970s we could do little more than diagnose the disease and then hold the crying towel for the forest manager. Before Earl hired me we discussed research assignments, and I thought we had agreed that I would focus on dwarf mistletoes and definitely not on LRR. So of course my first assignment from Earl was to help Starker Forests install a study of stump removal for controlling LRR; they had a bulldozer in the field ready to go to work and I had a week to plan and get started. Of course one thing led to another on that slippery LRR slope and 31 years later, after expanding stump removal trials, studying effects of fertilization, trying to understand disease impacts on commercial thinning and vice-versa, and a variety of other questions pondered, I am still working on laminated root rot, a direction that I never really changed.

4. Guidance for a Keynote Address. I have struggled with an appropriate structure and theme for this talk. The paper I presented last year, where I detailed some
recent successes in answering questions posed by forest managers, would be good as a keynote address. But too many of you look like you are paying attention so I am afraid I wouldn’t get away with a repeat presentation. In doing so I looked back at past proceedings and realized that for over 50 years this opening talk was given by the chairperson, and I prepared such a talk in 1997 for our meeting just down the road at Prince George. Then I noticed that when Ellen Goheen became chairperson suddenly the opening talk became the responsibility of the award recipient. I know that this is a coincidence, but any of you who have doubted Ellen’s ability to delegate should set those concerns aside.

As I looked for further guidance as to what to say today, I recalled that in presenting the award Don had referred to me as a “model researcher,” I felt greatly complimented, until, while preparing for this talk, found that Webster defined a model as a “small or lesser version of the real thing.” Later I asked Ellen what I should include in a keynote address. She replied that this was my chance to pontificate. So back to Webster’s only to discover that pontificate means “to speak with pompous authority or dogmatism.” Well with the bar of expectation being set that low, this talk may ramble a little.

Although my presentation will not be particularly structured, I will follow the general tradition of these talks and start with a brief personal history. Then I will talk about the reduction in forest pathology research positions, thoughts on WIFDWC, and finally perhaps share some survival skills with the younger members of our conference. Note, I have not suggested that all these thoughts and observations are connected.

5. My history. My history is not very complicated. I was raised just north of St. Louis, Missouri, in the towns of Ferguson and Florissant. My closest relatives are farmers. My father left the farm and moved to the city to drive a school bus and eventually managed a fleet of 90 school buses for our local school district. As was common for depression era young adults, his schooling ended with the 8th grade. Like many of my generation I did yard work and odd jobs to earn money for college and eventually attended the University of Missouri in Columbia where I earned a BS in Forest Management and a commission as a 2nd Lt. in the United States Army. After receiving a temporary deferment to active duty, I attended the University of Wisconsin in Madison, worked with Bob Patton on a root disease in forest tree nurseries, and graduated with a Ph.D. in plant pathology. Bob Patton taught me to do research with an emphasis on keeping field study designs simple, well replicated, and structured so that if our working hypothesis was not correct that we still had reportable results. A declining number of my classmates, including Everett Hansen, are still attending this conference.

I spent three years in the Army in Denver Colorado working on a classified and sensitive project. I want to say upfront that I enjoyed my military time and would repeat it in a heartbeat. Among other things it taught me to find ways to accomplish my perceived mission, sometimes in spite of superiors.

In January 1973, I joined Jim Hadfield and Dave Johnson in Portland in the Insect and Diseases Control branch of the Timber Management Division, Region 6. While I was there, we reorganized and moved to State and Private Forestry. In July 1975, I accepted a position as a research plant pathologist with the PNW Research Station’s Corvallis Lab and have been there since. I plan to retire on January 3, 2007, just 91 days from now. However, I will contribute some time as an emeritus scientist so that I can complete a number of planned papers and two major projects. Perhaps I will be able to report on them at a future conference. We also arranged this emeritus scientist status so that Gail would not have to immediately and wholly give up arranging her life around my work at the Station. The emeritus position allows me to slowly transition into my new full time job as a woodworking Grandpa. It has long been my intent that, when I die, someone will have to turn off my wood lathe rather than my office lights.

6. Staffing in the Forest Service Forest Pathology Research. While I was not the last pathologist hired by the PNW Station, in general, our total numbers have drifted down. When I arrived at the Corvallis Lab the pathology project there had five scientists, three technicians, and one microbiologist, and all were permanent full-time positions -- mine is the only original position remaining! My understanding is that when I retire in January my position will be closed. The one constant in our society and the world in general is change; nothing remains fixed. Those who look only to the past or the present are certain to miss the future. Last year when I reported on the number of national USDA Forest Service pathology positions there were 13 full-time equivalent (FTE) forest pathology research positions. I conducted the survey again this fall and found that while no positions were lost, shifts in assignments have resulted in the loss of an aggregate 1.5 FTE. In January, when my position is vacated, one more position will be lost. This is a
dramatic reduction and one that I personally think is a mistake, but it is not an issue that I take personally. It is like watching a glacier inching towards your house. I believe that the tenure of the upper level administrators in our Washington Office, excluding the clerks and those who get the real work done, is about 6-7 years. This same rate of turnover occurs at the PNW Research Station. This means that 7 generations of administrators have come and gone during my career. Clearly no one person or group of people bears a disproportionate responsibility for the decline in forest pathology research positions. The only connection I would make is that this decline started about the time Terry Shaw III was hired into the Forest Service. I think there has just been a shift away from a consideration of pest management issues to other important things, such as the shape of toilet seats in campgrounds or the potential economic impact of endangered slugs on the stock market in 2010. Perhaps as Paul Hennon once suggested, we have done such a good job of integrating with other disciplines that we have become invisible. Or perhaps, as I believe, the very openness that we western pathologists treasure (that is, our willingness to share and debate our ideas, data, and conclusions) has contributed to the decline. Forest tree disease research, by its very nature is a long-term enterprise and the results may be in use long before being published in a refereed journal. Examples from my own career are publication of the stump removal and fertilization study reported after 25 years of observation and the stump fumigation study after 16 years of observation. Both studies had been well discussed with colleagues before they were initiated, interim data were presented to various conferences, including this one, and both were the subject of several field workshops. By the time the results were published in refereed journals, everyone who needed to know the results already knew, few reprints were requested, and most who used the information and passed along the results accepted it as “common knowledge” and might be hard pressed to know where it came from. Furthermore, and more important for our administrators to realize, is that it can be difficult to connect management decisions to the research work we have done. Still, if I could do it all over again, I would not change the way my research results were disseminated. I believe that we must remain open and free to discuss studies at every stage. It is vitally important that our pest management practitioners be informed and able to share, with land managers, the best information available even if it’s awaiting official publication.

It is a conundrum – researchers must continue to share openly as new information evolves and at the same time, be able to demonstrate to administrators the value of studies requiring decades to complete. This at a time when the norm is instant gratification and most administrators consider “long term” to be three years.

I find it curious that our upper level administrators do not seem to recognize that there has been a decline in forest pathology and entomology research capability. In a recent review of Region 6 FHP, when I presented evidence of this decline the head of the review team replied “So what?” Rather than expressing a callous indifference to the decline of pathology research capability, what he really was asking for was a response that he could give to policymakers or congress at budget time. Unfortunately, I feel that too many of our administrators have a vision that unfortunately stretches no further than the next budget cycle. There seems to be a belief that forest health and many other problems can be solved in a year or two if we just throw enough money at them. I find this shortsighted in a profession where resource managers are dealing with a crop with an 80- to 200-year cycle, and where making the wrong choices may take a long time to correct or could easily result in starting over.

I do not have a solution. But I think we need to avoid painting the picture as gloom and doom. I am convinced that there will always be some pathologist and entomologist research positions; we do not lack for researchable questions.

7. Thoughts on WIFDWC. If WIFDWC did not exist, we would have to invent it. This is without a doubt the most successful and perhaps important gathering of forest pest managers in the world. This loose confederation of individuals, all engaged in a similar enterprise but with very different individual goals, has provided an unparalleled opportunity for the exchange of ideas and problem solving. WIFDWC’s very basis encourages the mixing of new, eager students just “discovering” forest pathology with the lifetime members who are its pioneers. Our history is important to us, and we take great delight in those meetings where the original members (or those nearly that) can join in discussions. The nature of the meeting is still much the same as the first meeting I attended in 1973 at Estes Park, although perhaps a little less raucous. In recent years, I don’t recall food fights, anyone passing out at the banquet, or formal letters from a city inviting us not to return. I have noticed in recent years a shift towards more formal papers, less debate of ideas, and shorter coffee breaks. I submit that the informal discussions and interactions
are every bit as productive and perhaps more so than the formal papers. Even though my supervisors may think that coffee breaks are a waste of time, I believe that the time I have “wasted” has been well spent! Perhaps in future years the organizers can begin a trend of expanding the coffee breaks until the formal papers become an endangered entity.

8. **Suggestions to young professionals:** We must each evolve our own job survival skills, but I will share a few that I have used. I will try to refrain from mentioning what to avoid as I suspect that many of you would prefer to make your own mistakes. Further, advice is said to be worth what you pay for it, and it puts things in perspective to recognize that this advice is free.

A. **Identify your customer** or primary audience, keep a focus on who you are and who you are helping. My focus has been on the foresters or forest land managers. If your focus is just to please your supervisor or impress higher administrators, then you will eventually just move into administration yourself. When you retire, you will likely have the satisfaction of knowing that you left as much impression on natural resource management as a stick leaves when you pull it out of a bucket of water: Perhaps a few ripples but no lasting mark or impression.

B. **Be true to yourself.** In the end, you are the one that has to be pleased with what you have done. If you are not then you will eventually quit, or worse, quit trying. Have fun with your job. Life may not be the party we had hoped for, but as long as we are here we might as well dance.

C. **Stay focused on getting the job done** that you think needs doing. Last year Don Goheen suggested that there may have been times when I worked a little close to the edge of our rules and regulations, and I admit that I may have pushed the envelope a few times. Sometimes you just have to get around the road blocks.

D. **Keep your own importance in perspective.** Our profession, more than most, builds on the efforts of our predecessors. If I can see further today, it is only because I have stood on the shoulders of giants such as Frank Hawksworth, Bob Gilbertson, Lew Roth, Gordon Wallis, Duncan Morrison and many others. I have also been helped and supported by many people. I have a Powerpoint presentation to illustrate my point. This small dot represents the impact of Walt Thies on the world. Let’s add dots for the 62 researchers who I have worked with cooperatively with during my career. Add to it dots for the 93 land managers who worked directly with us over the years and then the approximately 176 support staff who came and went or stayed during my time at the Forestry Sciences Laboratory. And add the approximately 286 temporary and term employees who worked on my projects during my time in Corvallis. Looking at it this way, it is not hard to ask “Where’s Walt?” To me it is a little embarrassing to accept recognition for accomplishments that have really been done by so many. I won’t belabor that further, because it is the nature of the work that we do.

9. **Summary.** Let me summarize this talk with the five points that I think were necessary for me to have a satisfying career. Perhaps these will resonate with some of you.

1. Marry well.
2. Have a clear image of who you are working for.
3. Keep and support this conference and its members.
4. Keep a perspective on your own importance in the work place.
5. Last and perhaps most important, marry well.

I now return the podium to the chairperson, who is sure to have far more illuminating subjects to share with us. Thank you for your kind attention today and your collegiality over many years.
Panel: Student Papers
Angie Dale, Moderator

Program

Frequency of Southwestern Dwarf Mistletoe and Mountain Pine Beetle in Colorado’s Northern Front Range Ponderosa Pine. Russell Beam (Presenter), Jennifer Klutsch, and William Jacobi, Colorado State University, Fort Collins, CO; and José Negrón, Rocky Mountain Research Station, Fort Collins, CO.

A Preliminary Evaluation of Fuel Levels in Southwestern Dwarf Mistletoe and Mountain Pine Beetle Infested Ponderosa Pine. Jennifer Klutsch (Presenter), Russell Beam, and William Jacobi, Colorado State University, Fort Collins, CO; and José Negrón, Rocky Mountain Research Station, Fort Collins, CO.

Technology Transfer and Forest Health. Amanda Crump (Presenter), William Jacobi, Colorado State University, Fort Collins, CO; and John Lundquist, Rocky Mountain Research Station, Fort Collins, CO.

Assessment of Colletotrichum gloeosporioides as a Biological Agent for Western Hemlock Dwarf Mistletoe (Arceuthobium tsugense). Sue Askew (Presenter) and Bart van der Kamp, University of British Columbia, Vancouver, BC; and Simon Shamoun, Pacific Forestry Centre, Victoria, BC.

Magnesium Chloride Dust Suppression Products and Roadside Environments. Betsy Goodrich (Presenter), William Jacobi, and Ronda Koski, Colorado State University, Ft. Collins, CO.
Frequency of Southwestern Dwarf Mistletoe and Mountain Pine Beetle in Colorado’s Northern Front Range Ponderosa Pine

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

A random ground survey was conducted to determine the frequency and distribution of Southwestern dwarf mistletoe (*Arceuthobium vaginatum* subsp. *cryptopodum*), mountain pine beetle (*Dendroctonus ponderosae*), and *Ips* spp. within Colorado’s Front Range ponderosa pine (*Pinus ponderosa* var. *scopulorum*). Stand structure canopy closure estimates were also recorded with greater than 80% of the forest floor receiving sunlight classified as open and less than 80% sunlight classified as closed. A total of 32, 1500 m extent transects, were located in randomly selected ponderosa pine dominated polygons from the USDA Forest Service Common Vegetative Unit for Roosevelt National Forest. Every 50m along the transect an 11.35 m radius plot (0.04 ha) was conducted quantifying the incidence and severity of Southwestern dwarf mistletoe, mountain pine beetle, and *Ips* spp. Within the study area stand structure was approximately 65% open and 35% closed. There was a significantly higher occurrence of mountain pine beetle and *Ips* spp. in plots that had dwarf mistletoe than those plots without dwarf mistletoe. The highest incidence of bark beetles occurred in plots with the highest severity of dwarf mistletoe. These results strengthen the idea of bark beetles utilizing pockets of stressed trees in endemic populations.


Russell D. Beam and Jennifer G. Klutsch are graduate students and William R. Jacobi is a professor in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, Colorado 80525-1177. José F. Negrón is a research entomologist with the Rocky Mountain Research Station, Ft. Collins, Colorado 80526-2098.
A Preliminary Evaluation of Fuel Levels in Southwestern Dwarf Mistletoe and Mountain Pine Beetle Infested Ponderosa Pine

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

Stand structure and downed woody debris in ponderosa pine forests (*Pinus ponderosa* var. *scopulorum*) of the northern Front Range of Colorado were measured to assess the difference in fuel levels and potential fire behavior due to Southwestern dwarf mistletoe (*Arceuthobium vaginatum* subsp. *cryptopodum*) and mountain pine beetle (*Dendroctonus ponderosae*) infestations. The fuel attributes were measured in 2005 and 2006 on 150 plots (0.04 hectare) with modified Brown’s planar transects throughout the Canyon Lakes Ranger District, Roosevelt National Forest. Based on ANOVA, dwarf mistletoe intensity, canopy cover, and average tree decay status of mountain pine beetle infestation were shown to contribute a significant amount of variability in duff depth, 10 hr time lag fuel and 1000 hr time lag fuel patterns. For example, there was a greater 1000 hr time lag fuel load in plots with an older infestation of mountain pine beetle, as determined by the average tree decay class, when compared to un-infested plots. Further analysis will assess the contribution of environmental and stand attributes to fire hazard within stands experiencing either southwestern dwarf mistletoe or mountain pine beetle infestations.


Jennifer G. Klutsch and Russell D. Beam are graduate students and William R. Jacobi is a professor in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, Colorado 80525-1177.
José F. Negrón is a research entomologist with the Rocky Mountain Research Station, Ft. Collins, Colorado 80526-2098.
Technology Transfer in Forest Health

Amanda Crump, William R. Jacobi, and John E. Lundquist

Technology transfer is the movement of research to a user. Most technology transfer research has addressed the transfer of scientific research to the public and private landowners. This study quantifies the usage and preferences of technology transfer of forest health information to Region Two, United States Forest Service employees. Populations of researchers, intermediaries, and users in the Rocky Mountain Region were surveyed, interviewed, and observed. Respondents use a variety of media to stay current on forest health research findings. While the respondents utilize computer media to learn about forest health research, they would prefer to use face-to-face methods. Respondents believe that forest health research is important but find some of the current technology transfer media difficult to use. Survey respondents and interviewees relied heavily on face-to-face interactions to help them make forest management decisions, especially when those decisions needed to be based on the best and most current scientific findings. Most people in this study identified lack of time as the largest barrier to staying current on forest health research. Thus, informational products should be easy to understand, require limited time to find, and utilize face-to-face media to effectively transfer forest health information.


Amanda Crump is a graduate student and Bill Jacobi is a professor in the Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins, CO 80523-1177.

John Lundquist was a Research Plant Pathologist, Rocky Mountain Research Station, Fort Collins, CO 80526.
<table>
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<tr>
<th>Mike McWilliams</th>
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<td>Rosie Bradshaw</td>
<td>Bob Edmonds</td>
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Assessment of *Colletotrichum gloeosporioides* as a Biological Agent for Western Hemlock Dwarf Mistletoe (*Arceuthobium tsugense*)

**Sue Askew, Simon F. Shamoun and Bart van der Kamp**

**Abstract** — Changes in the B.C. forest timber harvest systems have reduced the number of methods for the control of western hemlock dwarf mistletoe (HDM) using conventional silviculture practices. An inundative biological control strategy may provide an alternative method of control. This approach includes the use of the endemic parasitic fungi *Colletotrichum gloeosporioides* (*C.g.*). PFC isolate 2415 was selected as a lead isolate based on pathogenicity tests on detached dwarf mistletoe shoots. A field trial consisting of healthy dwarf mistletoe infections was used to determine the efficacy of two *C.g.* formulations. The formulations used in this project were the ‘Stabileze’ method and sucrose and gelatin suspension. Four months after application of *C.g.* formulations, there was a significant reduction in the percentage of diseased berries. Comparing the two formulations, no significant differences in the effectiveness of the two different formulations were noted after 4 months. The reduction in healthy berries indicates that *C.g.* may effectively interrupt the HDM lifecycle and therefore reduce the spread of HDM.

**Introduction**

The traditional approach to dwarf mistletoe management on the coast of BC, consisting of large clearcuts with either mistletoe eradication or regeneration with non-host species, has worked well. However, recent concerns about habitat diversity and sustainable forests by the consumers of wood products have resulted in a change from clear-cut practices to variable retention (Mitchell and Beese 2002). Variable retention (VR) consists of retaining patches, small groups and individual trees, at harvest, and in BC coastal forests many of these will carry hemlock dwarf mistletoe. Hemlock regeneration is common with VR, and a large part of the harvested area is within reach of overhead sources of dwarf mistletoe seed. Variable retention, therefore, can be expected to result in serious dwarf mistletoe damage to regenerating stands. In this study we explored the potential for biological control of hemlock dwarf mistletoe following variable retention harvesting. Such control could also be useful in riparian reserves and other sensitive ecosystems. We focused on a native fungal parasite of hemlock dwarf mistletoe shoots and berries, *Colletotrichum gloeosporioides*, and an inundative control strategy. The ultimate goal of this research is to reduce mistletoe seed production substantially following a single application of the parasite. The research objectives of this study were:

- To select a lead isolate through pathogenicity screening of several coastal BC isolates of *C. gloeosporioides*;
- To describe the response of *C. gloeosporioides* lineal growth and conidia germination to temperature; and
- To compare the efficacy of two formulations *C. gloeosporioides* (*Stabileze* and a gelatin and sucrose formulation) in a field situation.

**Methods and Materials**

**Production of *C. gloeosporioides* Isolates**

Diseased *A. tsugense* shoots, fruits and swellings were collected from different locations from Vancouver Island and cultured and maintained in the Pacific Forestry Centre (PFC) culture collection. Five isolates from three different locations in B.C. were described and screened to select one for a field trial.
Conidia were produced as follows. Twenty grams of millet in 20 mL distilled water was autoclaved twice for 20 minutes at 121°C and inoculated with six mycelial plugs from PDA cultures of the parasite. Two replicates of each isolate were prepared. Flasks were maintained for 10 days under 12 hours light and 12 hours dark a day at 20 to 23°C and agitated daily. The millet substrate was washed with 100-mL sterile distilled water by placing the flasks on a rotary shaker at 50 rpm for 60 minutes. The contents of each flask were filtered through sterile cheesecloth into two falcon tubes and centrifuged at 2400 ×g for 30 minutes. The supernant was decanted from the falcon tubes and the conidia re-suspended in 10 mL of sterile distilled water. The contents of two falcon tubes originating from the same flask were then combined.

The conidia suspension was stored at 4°C and screened for bacterial contamination by plating 500 μL of conidia suspension on potato dextrose media and growing it at 25°C for 3 days. The conidia were used within one week of harvesting. Conidia concentrations were determined using a haemacytometer.

In vitro Virulence of Isolates

Virulence was screened using detached hemlock branches infected by hemlock dwarf mistletoe and bearing mistletoe shoots. Male and female (with berries) samples were collected at the Spider Lake field site (described below) in early August 2002. Branches were placed in rock wool cubes that were pre-soaked overnight in a hydroponic solution (2 mL/L of Thrive Alive B-1, Techafloa Plant Products, Vancouver, BC).

Mistletoe shoots were sprayed to runoff with a 10^6 conidia/mL water suspensions using four replicates (infected branches) for each of the five isolates. A control treatment consisted of a water spray.

Inoculated branches were placed in a germination tray. Excess moisture in the bottom of the germination trays was removed, and the trays were kept in a Precision® (Ohio, USA) environmental growth chamber at 18 hour days at 15°C and 6-hour nights at 15°C and monitored weekly for one month.

A diseased shoot was defined as any shoot showing symptoms of *C. gloeosporioides*. Typical diseased symptoms of *C. gloeosporioides* on *A. tsugense* included small necrotic brown or black lesions that progress from the tip of the shoot to the base of the shoot. Initial disease symptoms were typically brown to black lesions near the tip of the shoot. The disease progressed on the dwarf mistletoe shoot by necrosis to complete blackening of shoot (Kope and Shamoun 2000). Infected dwarf mistletoe berries turned from a healthy blue/green colour to black or black purple and then developed acervuli and became mummified (Kope and Shamoun 2000). The extent of infection was expressed as the percentage of shoots and berries infected after one month.

Growth Characteristics of *C. gloeosporioides*

The mycelial growth rate of the 5 PFC isolates was determined for each of the following temperatures 4, 10, 15, 20, 25, and 30°C. A mycelial plug (7.5 cm) of *C. gloeosporioides* was placed in the centre of a PDA plate and incubated at each temperature. Three replicates were used for each of the isolates. The diameter of each colony was measured along two pre-determined lines at right angles to each other. The mycelial growth for each plate was the average of the measurements along the two pre-determined lines. Plates were measured every fourth day for 2 weeks or until the fastest growing colony at a particular temperature reached the edge of the PDA plate.

Conidia Germination at Varying Temperatures

Conidia were germinated on 1.5% water agar plates (three replicates per isolate) at 4, 10, 15, 20, 25, 30, and 35°C using 500 μL of a 1.0 x 10^5 per mL conidia suspension per plate. Germinated conidia were defined as conidia with a germ tube length exceeding the diameter of a conidium. Counts of 100 conidia per plate were made at 2, 4, 6, 8, 9, 10, 12, and 24 hours. Germination was expressed as a percentage after 24 hours and as number of hours to reach 50% germination. The latter was achieved by fitting various curves to the germination-over-time data set and estimating the time to 50% germination.

Field Trial

Spider Lake site description—A field site was selected at Spider Lake near Qualicum, BC in the Coastal Douglas-fir Zone (GPS: 49° 21′ 43″ N 124° 37′ 42″ W, elevation 51 m.) The stand (approximately 2 ha) consisted mainly of young infected hemlock with many mistletoe infection between 1 and 2 m of the ground under a scattered canopy of older, heavily infected hemlock.
Both *C. gloeosporioides* and *Neonectaria neomacrospera* (identified by culturing) and acervuli were present on *A. tsugense* shoots and infected bark respectively in and around the research site.

**Formulation description**—Stabileze’ was formulated as follows: 5 grams of Water Lock B-204 (Grain processing Corporation, Muscatine, Iowa, USA) water absorbent starch was mixed with 5 mL of unrefined corn oil (Spectrum oil) heated in a microwave oven at high power for 1 minute and cooled to room temperature. Twenty mL of a 1.0 x 10⁷ conidia/mL suspension of isolate PFC 2415 were added to the cooled starch/oil mixture and mixed with a metal spatula. The mixture was allowed to stand to fully absorb the 20 mL liquid. Twenty grams of Confectioner’s powdered sugar (Roger’s sugar, Vancouver B.C., Canada) was added followed by 7 grams Hi Sil 233 (PPG Industries Inc, Pittsburgh, P.A., USA) hydrated silica. The formulation was completely mixed until uniform crumbs of approximately 0.5 cm were formed. The mixture was spread 3-4 mm thick on aluminum foil, and allowed to dry for 48 hours (Quimby et al. 1999).

The sucrose-gelatin formulation was prepared by heating 500 mL of sterile distilled water to 60°C and adding 2.5 grams of gelatin (Sigma, Oakville, Ontario, Canada) and maintaining the mixture at 40°C until the gelatin granules were entirely dissolved. Then 10 g of sucrose were added to the mixture. The mixture was then cooled to room temperature and a concentrated fungal suspension of PFC isolate 2415 was added to yield a final concentration of 1.0 x10⁶ conidia/mL.

**Experimental design**—The experimental design was a complete random design. A total of 169 clean (showing no evidence of fungal parasites) individual *A. tsugense* infections were randomly assigned to the treatments and controls (Table 1).

A Hobo™ data logger (Onset, Bourne, MA, USA) was installed near the centre of the plot to record temperature and relative humidity. Dwarf mistletoe infections were sprayed to run-off with the appropriate suspension. After application was completed, the suspensions used were sprayed on PDA plates to check conidia viability. Treatments were applied on August 29, 2002. The weather during the inoculation was sunny with periods of overcast and a maximum temperature of 24°C coinciding with the minimum relative humidity of 52.8% at 3:35 PM. The weather during the following week remained dry and variably cloudy with temperatures around 20°C throughout the day and night temperatures as low as 7.0°C.

**Assessments**—Each dwarf mistletoe infection was assessed at 6, 15, 28, 63, 102, 145 and 240 days after treatment. At each assessment the following data were recorded: swelling diameter, number of mistletoe buds (shoots <0.5 cm in length), number of shoots, number of diseased shoots, number of berries and the number of diseased berries. At the first and last assessment additional measurements included swelling length, distance between the most distal and proximal shoots, sex, and maximum shoot length. Five swellings for each treatment were photographed at every assessment.

**Verification of *C. gloeosporioides* in the field**—One month after treatment, samples of diseased shoots were collected randomly from the 5 treatments. These samples were surface sterilized by placing the diseased materials in 95 % ethanol for 2 minutes, followed by placing the parts in 10% sodium hypochlorite for 2 minutes, followed by 3 2-minute washes of sterile distilled water. The surface sterilized pieces were then transferred to potato dextrose agar (PDA, Difco Laboratories, Detroit, USA) and incubated for approximately 10 days at 20°C. *C. gloeosporioides* colonies were identified based on morphological characteristics such as conidiophores, conidia and the mycelium as described by Kope and Shamoun (2000).

**Results**

**Conidia Production of *C. gloeosporioides* Isolates**

There was no practical difference between conidia production on millet of the five isolates. Only the most

### Table 1—Treatments and number of replicates used at the Spider Lake field trial.

<table>
<thead>
<tr>
<th>Treatment</th>
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<td>1.  Stabileze suspension</td>
<td>36</td>
</tr>
<tr>
<td>2.  Sucrose Gelatin suspension</td>
<td>36</td>
</tr>
<tr>
<td>3.  Stabileze control formulated without conidia</td>
<td>35</td>
</tr>
<tr>
<td>4.  Sucrose and gelatin control Formulated without conidia</td>
<td>35</td>
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<td>5.  Water spray control</td>
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productive (PFC 2415) and the least productive (PFC 4059) were significantly different.

**In vitro Virulence Test**

Three weeks after inoculation there was no significant difference in percent of shoots or berries infected between the five PFC isolates. PFC 2415 showed the highest percentage of disease shoots and berries.

**Effect of Temperature on Lineal Growth Rate**

Maximum lineal growth occurred between 15 and 25 °C and the optimum temperature varied significantly between isolates. Maximum lineal growth for PFC 4060, 4059, and 2280 occurred at 25°C; for PFC 2415 at 20°C and for PFC 4058 at 15°C.

**Effect of Temperature on Conidia Germination**

Optimum temperature for total germination after 24 hours ranged from 15 to 25°C. At 15°C, PFC 4058 and 2415 exhibited a significantly higher conidia germination percentage than PFC isolates 4060, 2280 and 4058. No differences in conidia germination were detected at 20 and 25°C. At 35°C, PFC 4058 had significant lower conidia germination than the other isolates.

At 4 °C, PFC 4058 conidia germinated significantly faster than those of PFC 4060 and 4059 while PFC 2415 and 2280 did not differ from any of the isolates.

At the other temperatures there was no significant difference in the number of hours to reach 50% germination. However at these temperatures PFC 2415 always exhibited the fastest germination.

**Field Trial**

**The Spider Lake Field Trial**—On the basis of the highest virulence rating in the *in vitro* virulence test, isolate PFC 2415 was selected for the field trial. One month after treatments were applied symptoms of *C. gloeosporioides* on *A. tsugense* shoots collected from the Spider Lake site appeared as a brown to black necrotic regions. Mature infections enlarged and girdled the dwarf mistletoe shoot, resulting in death of the distal portion of the shoot. Some dwarf mistletoe shoots turned black and the entire shoot was killed. Infected *A. tsugense* berries turned from green-blue to purple black and eventually became mummified.

**Dwarf Mistletoe Berries**—The main dispersal of berries occurred between September 27 and November 5, 2002. At the November assessment only a few berries remained attached to the shoots, and almost all of these were diseased. The treatments with *C. gloeosporioides* showed a significant reduction in the number of healthy berries compared to controls. There was no significant difference between the two formulations.

**Dwarf Mistletoe Shoots**—Diseased shoots are quickly shed, and hence analysis of the effect of treatments on shoot health was based on the number of healthy shoots remaining at each observation time, expressed as a percentage of the number of healthy shoots for each swelling at the start of the study. The number of healthy shoots declined steadily over the course of the experiment for both treatments and controls, such that by the last observation eight months after treatment the remaining healthy shoots comprised <20 percent of the number of shoots at the start. At the November and December observations, the percentage of healthy shoots remaining was significantly lower for the *C. gloeosporioides* treatments than for the controls. There were no significant differences at the other observation times. In November the Stabilize formulation showed the greatest reduction in healthy shoots, and in December the Sucrose-Gelatin showed the greatest reduction. The differences between the Stabileze and Sucrose-Gelatin formulations were not statistically significant at any observation time.

**Discussion**

Ideally, selection of an inundative biological control agent is based on the most aggressive and virulent isolate and its ability to survive in field conditions with no impact on non-target plants (Templeton 1982; Charudattan 1989; Watson 1998). A major objective of the studies described in this paper was to select a single isolate for further extensive field testing. The differences in mycelial growth, conidia production, and percent conidia germination however, were so small that none of the isolates could be rejected on that basis. The most important criterion for selection is undoubtedly virulence. The virulence was tested directly *in vitro*. It was also tested indirectly as rate of germination (quick germination presumably being associated with greater virulence) and linear mycelial growth rate (again greater rates of growth presumably being associated with greater virulence). The direct
tests of virulence suggested variation between isolates although statistical tests failed to prove this conclusively. Nevertheless PFC 2415 is most likely to be the most virulent of the isolates. In the case of germination rate and linear growth rate there was an interaction between isolate and temperature – the best isolate depended on the temperature at which the test was done. This suggests that eventually different isolates might be used for different locations. The selection of an isolate was based on prioritizing the screening results for each PFC isolate tested. For the purpose of further study the results of the direct virulence tests were judged to outweigh other considerations, and so isolate PFC 2415 were selected as the lead isolate.

Reducing the spread of dwarf mistletoe can be achieved by 1) reducing berries 2) reducing shoots and 3) killing the endophytic system of the dwarf mistletoe plant (Ramsfield 2002; Shamoun et al. 2003). In this field trial, the mean percentage of healthy berries was reduced for both the Stabileze treatment and sucrose/gelatin treatment when compared with their respective control between 28 and 63 days after the application of treatments.

The primary and secondary cycles of infection by *C. gloeosporioides* that infects dwarf mistletoe is relatively unknown. In this study, acervuli were first observed in the field 4-8 weeks after the application of the inoculum treatments. The substantial increase in the number of diseased berries occurred between 28 and 63 days while diseased shoots occurred between 15 and 28 days. The time required to form acervuli (1-2 months after application of treatments) runs parallel with the increase in number of diseased berries and reduction in the number of healthy shoots which could indicate that secondary *C. gloeosporioides* infection can occur after 28 days. Studies on berry crops and fruit crops such as citrus fruit revealed that prolonged periods of wetness or following rainfall by rainfall promoted primary *C. gloeosporioides* infection (Avenlino et al. 2004). With blueberries and strawberries infected by *C. gloeosporioides* researchers have confirmed that secondary infection occurs after rainy, or misty or cool conditions (Freeman et al. 1998; Hildebrand et al. 2005). Makowsi and Mortensen (1998) have demonstrated with the biological control agent *C. gloeosporioides* that infects mallow, secondary infection (acervuli are capable of reforming) can occur after 5-7 days in favourable conditions. In the race of *C. gloeosporioides* that attacks blueberries and strawberries, spore dispersal peaked with the flowering and early fruit development (Wharton et al. 2004; Hildebrand et al. 2005). With *C. gloeosporioides* that attacks almonds, mummified fruit represents the main source of conidia for infection (Adskaveg et al. 1998; Wharton et al. 2004). For the *C. gloeosporioides* that infects hemlock dwarf mistletoe, the primary and secondary infection, sources of inoculum, spread and persistence in the field has not been comprehensively investigated. Future research into the epidemiology of *C. gloeosporioides* will be important for the further development of this biological control.

The percentage and number of healthy dwarf mistletoe shoots decreased over the 240 day assessment period for both inoculum treatments and controls. The number of healthy shoots in the inoculum treatments was not statistically significantly different from their respective control treatments. Reductions in healthy dwarf mistletoe shoots occurred for both inoculum treatments, 28 to 102 days after the application of the treatments. A reduction in shoots was also noted during the last (after 240 days) assessment percent loss. The Stabileze formulation inoculum treatment reduced the percentage of healthy mistletoe shoots more rapidly (63 days after treatments were applied) than the sucrose and gelatin inoculum treatment (102 days after treatments were applied).

The loss of shoots from the control treatments may have been product of high levels *C. gloeosporioides* in the natural background inoculums and/or by secondary infections initiated by application of the inoculum treatments. In some cases, the control treatments were located adjacent to inoculum treatments increasing the possibility of secondary infection on the controls.

The reduction in mean percentage of healthy dwarf mistletoe shoots by the inoculum treatments may have been masked by decline in healthy shoots in all treatments after the November assessment date. Other possible explanation for lack of differences between treatments and controls in healthy dwarf mistletoe shoots includes 1) secondary *C. gloeosporioides* infection that may have spread to the control treatments, 2) a high variation in number of diseased shoots within and between treatments, and 3) some buds may have developed into dwarf mistletoe shoots after August 29, 2002. Such shoots would not have been part of the count of healthy shoots at the start of the study.

Possible causes contributing to dwarf mistletoe shoot loss during this assessment period include: 1) handling of experimental units at each assessment period and 2) environmental stress on the site that reduced the vigour of dwarf mistletoe plant resulting in shoot loss.
To increase the reduction of dwarf mistletoe aerial shoots and berries by *C. gloeosporioides* future research should include: screening large number of hemlock dwarf mistletoe *C. gloeosporioides* isolates from different geographical location to select more virulent isolates; establishing the optimum concentration of the conidia for effective dwarf mistletoe infection, selecting the most compatible formulation and to determine combinations of other biological control agents to increase the efficacy of *C. gloeosporioides*.

Data collected from this study indicated that one time application of this biological control agent decreased the number of dwarf mistletoe berries and may have some influence in the reduction of the number of healthy dwarf mistletoe shoots. Further studies are required to determine the effect of secondary infection and persistence of *C. gloeosporioides* in field conditions.

To develop *C. gloeosporioides* into effective and possibly a commercially available biological control product for dwarf mistletoe control there are number of challenges that need to be addressed. Theses challenges include determining *C. gloeosporioides* lifecycle on dwarf mistletoe such as methods of conidia spread, secondary infection and latency tendencies (if they exists) and the location of the overwintering stage of the conidia (upper or lower canopy or shoot litter found on the ground). DNA from the jellyfish is inserted into *C. gloeosporioides* can be used to detect plant-microbe interactions with this biological control agent. The green fluorescent protein (GFP) is a protein from the jellyfish *Aequorea victoria* that fluoresces green when exposed to blue light using a fluorescent microscope (Maor et al. 1998). Conidia and hyphae of GFP-expressing fungal isolates can be identified by fluorescence microscopy in intact tissues or tissue sections allowing for a distinction between inoculum that is applied and background inoculum (Maor et al. 1998; Robinson and Sharon 1999).

**Acknowledgements**

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**References**


Figure 1—Detached *A. tsugense* swellings with healthy shoots three weeks after treatment - Control (sterile distilled water).

Figure 2—Detached *A. tsugense* swelling three weeks after treatment with PFC 2415. Note collapsed brown shoots.

Figure 3—Swelling 2758 on August 29, 2002 before treatments were applied.

Figure 4—Swelling 2758 on Nov 5, 63 days after treatment with the Sucrose-Gelatin formulated with PFC 2415. Note the blackened diseased berries.
Magnesium chloride dust suppression products and roadside environments

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

Magnesium chloride (MgCl₂) is applied to gravel roads during summer months for dust suppression and road stabilization. Research quantifying the impacts of MgCl₂ on vegetation is limited, and potential impacts of dust suppressants on roadside systems are studied even less. This project investigates vegetation and soil properties along gravel roads in Larimer and Grand Counties, Colorado, USA. Objectives of this study are to determine movement and spatial distribution of MgCl₂-based dust suppression compounds from treated gravel roads into roadside systems, and to determine if road maintenance procedures are related to plant damage along gravel roads.

1 Chloride moved downslope from roads between 10 and 20 feet, on average. Magnesium ions were significantly higher close to and downslope from the road edge, but did not displace other important soil cations, such as calcium or potassium. Ions taken up from soils can accumulate in leaves and cause toxicity and osmotic stress in trees, leading to dehydration injury typical of drought. Trees in our plots uptake and accumulate high levels of chloride in leaf tissue downslope from the road, but are not deficient in essential plant nutrients such as calcium or potassium. Trees displayed varied amounts of foliar damage and correlations between foliar ion content and crown damage is weak but significant.


Betsy Goodrich is a graduate student, Ronda Koski is a Research Associate, and Bill Jacobi is a professor in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO 80525-1177.
Panel: Aftermath of the Largest Mountain Pine Beetle Outbreak in Recorded History: What now? Silviculture and forest health issues

Kathy Lewis, Moderator

Program

Silviculture and the Mountain Pine Beetle – What to do? K. David Coates, British Columbia Forest Service, Smithers, BC.

Premonitions of Post-Mountain Pine Beetle Pathogens. Bart van der Kamp, University of British Columbia, Vancouver, BC.

Landscape-level concerns after mountain pine beetle: Warren root collar weevil in regenerating pine stands. Brian Aukema (Presenter) and Matthew Klingenberg, Pacific Forestry Centre and University of Northern British Columbia, Prince George, BC; Carolyn Stevens, British Columbia Ministry of Forests & Range, Burns Lake, BC; and Staffan Lindgren, University of Northern British Columbia, Prince George, BC.

Seedling Tree Survival in Dothistroma and Mountain Pine Beetle Attacked Lodgepole Pine Stands. Frank Doyle, Wildlife Dynamics Consulting, Telkwa, BC.
The magnitude and extent of the current mountain pine beetle outbreak in British Columbia requires thoughtful planning to recover value from the impacted timber while maintaining other values and reducing impacts to future timber supplies. Currently, the epidemic covers over 9 million hectares and has killed about 300 million m$^3$ of pine volume. About 50% of harvestable timber in central BC is lodgepole pine.

Historically, clearcutting followed by prompt planting has been the dominant management practice in pine-leading stand types. The silvicultural strategies we employ during the salvage will profoundly affect mid-term (20-40 years) timber supply for affected communities. We know that total salvage and planting will not help communities faced with mid-term timber supply shortages.

In order to make informed decisions about management of stands and landscapes affected by the pine beetle we need better information on the abundance and extent of structure in lodgepole pine stand types. We currently have limited information on the abundance of seedlings and saplings (understory trees) in pine-leading stand types. Understory trees in pine-leading stands are generally dominated by species not susceptible to the mountain pine beetle, or if they are pine, are too small to be attacked, and hence will survive the epidemic. In addition to understory trees, non-pine sub-canopy and canopy trees can contribute to structure in pine-leading stand types. Collectively, we will call seedlings, saplings, sub-canopy and canopy trees that will likely survive a pine beetle attack “secondary structure”.

In a study undertaken for the Chief Forester of BC (Coates et al. 2006), we specifically addressed the following questions: (1) how abundant is secondary structure in pine-leading stands? (2) is secondary structure similar across biogeoclimatic units (SBSdk, SBSdw2, SBSdw3, SBSmc2 and SBSmc3) in pine-dominated stands? (3) can stands with good secondary structure reasonably be expected to reach harvestable volumes in the mid-term?

We found that pine-leading stands have considerable variability in secondary structure across north central BC. Approximately 20-30% of these stands have sufficient secondary structure today to reasonably expect a mid-term harvest opportunity if simply left unsalvaged. Approximately 40-50% of pine-leading stands in north central BC have sufficient understory densities to be stocked without further silvicultural intervention. We have insufficient data to predict if they would be considered stocked with well-spaced acceptable species. Such understory trees, if protected, may reduce rotations by 10-30 years compared to complete salvage and planting. Approximately 20-25% of pine-leading stands in north central BC had poor secondary structure. These stands are prime candidates for total salvage and planting.

There was some variability by ecological units with secondary structure being by far the lowest across the SBSdk subzone compared to other biogeoclimatic units. The SBSmc2 has an excellent combination of understory stocking and minimum thresholds of overstory basal area that can release and provide mid-term harvest opportunities.

In conclusion, there appears to be considerable potential to reduce the impact to mid-term timber supply and enhance biodiversity values, hydrologic recovery, visual quality and wildlife habitat by strategically protecting certain pine-leading stand types from immediate harvest and/or protecting secondary structure during salvage operations.

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References

Premonitions of Post-MPB Pathogens

B.J. van der Kamp

In this talk I will restrict myself to reflections on the likely problems faced by lodgepole pine regeneration following stand destroying attacks by mountain pine beetle (MPB). I will not consider MPB-killed stands that are salvaged and regenerated by planting. Such new stands will face a set of risk factors that are well known after many years of experience with lodgepole pine regeneration following logging or wildfire. It should also be recognized, that several other tree species will probably play significant roles in regeneration following MPB, including Douglas-fir, subalpine fir, interior spruce, and aspen.

Let me start with calling attention to a bit of an anomaly: Lodgepole pine is a ‘successful’ species. It is the dominant tree of the high, dry plateaus east of the BC Coast Mountains and elsewhere, but it is also found in many other places from coastal bogs and rock bluffs right up to the timberline in the Rockies. Perhaps it is this very success that has attracted so many pathogens. A perusal of known pathogens by species shows 24 foliage pathogens (more than any other BC conifer), and 26 necrotrophic ascomycetous pathogens of stems and branches (surpassed only by Douglas-fir and western hemlock in BC). Then there are 4 stem rusts (no other BC conifer has more), a dwarf mistletoe, an assortment of root diseases and decays, and some odds and ends like seed and seedling diseases. It all reminds one of that doggerel by Ogden Nash about the turtle:

The turtle lives ‘twixt plated decks which practically conceal its sex
I think it clever of the turtle
In such a fix to be so fertile.

(For our British visitors, I can assure you that the last two lines do indeed rhyme if read by a Texan.)

And so I offer this variant for your edification:

Lodgepole pine’s beset by pests
Of foliage bark and all the rest
I think it clever of the pine
In such a fix to do just fine

Two phenomena are usually considered when explaining the success of the species in the face of such an assault. Great genetic diversity at all scales, and abundant regeneration. I believe we would do well to remember these, and especially in the face of great uncertainties about climate, as well as the uncertainties about the responses to conditions following MPB (as apposed to stand destroying fires or logging), and to continue to rely on these for both the short and long term survival and flourishing of the species. The uncertainty that we face is, of course, whether the characteristics of the species that have served to maintain it as the dominant forest cover over large areas will be sufficient to allow it to maintain that dominant position in the face of new challenges (rapid climate change, human intervention such as 50-odd years of fire control, and no doubt others). The massive MPB outbreak, an order of magnitude greater than any previously described outbreak of the pest, is perhaps an indication that things are not well, and that further troubles may appear.

I will approach my analysis by discussing various groups of diseases in turn, starting with foliage diseases. The typical pattern for most foliage diseases, most of the time, is relatively short outbreaks (2-5 years) with serious defoliation (but little if any mortality) followed by long periods of virtually undetectable levels. Such outbreaks occur on scales of a few hectares to region-wide. In all but the most severe cases, variation in resistance is obvious. Some trees remain essentially uninfected, and certainly undamaged even during outbreaks, and, on the other hand, some trees carry detectable infection even during intervening periods. Typically too, young trees are much more severely affected than older trees.

Who knows how these outbreaks get started. Presumably optimal weather conditions for one a few consecutive years allow massive buildups of inoculum, which then takes a few years to decline even under less favourable conditions. Secondary pathogens may also


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play a significant role. *Lophodermella concolor* combined with *Hendersonia acciola* is common in BC and a good example of a typical outbreak pattern. But it doesn’t always work that way, as we will see on one of the field tours. (*Dothistroma*). If I’m right about climatic triggers for outbreaks, then we may be in for some nasty surprises.

It’s probably worthwhile to distinguish between those foliage pathogens that spread primarily by ascospores, and those spread by conidia, the former having a short spore dispersal period, typically midsummer, and the latter ready to release conidia whenever decent conditions occur (wet and not too cold).

Finally we need to remember that the environment for young pine in post MPB stands is different from that following fire or logging, with (probably) higher humidity and lower windspeeds. Those conditions are likely to favour foliage diseases.

When it comes to necrotrophic pathogens of bark, the total list of species found on lodgepole pine is long, but for the moment few are serious, and most are restricted to stressed trees or tree parts. The only one that causes widespread damage is *Atropellis piniphilla*. Again, uncertainty about future conditions, and the possibility of severe stress in response to climate change may change this. There are certainly enough candidates.

Richard Reich has done some interesting work with *Elytroderma* recently. Apparently it invades and maintains itself in bark, causing long narrow swellings. Much remains to be learned here, about the infection process, the long-term results of infection both on tree survival, increment, and wood quality.

The three main stem rusts of lodgepole pine, western gall, stalactiform and comandra rust are all most common on young trees. They are also all subject to what Roger Peterson called ‘wave years’. In the last decade, we’ve had quite a number of such wave years for gall rust here up north. The others also appear to be on the rise, particularly comandra rust. In young stands in the pine areas just east of here (Smithers, BC), comandra rust is now much more common that when I first surveyed the area in 1980. In the case of comandra rust I believe we need to learn a good deal more about the ecology of its alternate host (strictly *Geocaulon lividum* (=*Comandra livida*) here up north) if we want to understand what is going on.

Stalactiform rust can be found throughout the BC range of lodgepole, but not usually in numbers that seriously threaten productivity. The two rusts differ in the way their mycelium grows in bark. Comandra rust grows at about the same rate longitudinally and laterally, resulting in a diamond-shaped canker and fairly rapid girdling of the stem axis on which it occurs. Hence infected trees tend to die quickly, and infections on branches need to be fairly close to the bole in order to reach it before the branch dies. Stalactiform rust mycelium, on the other hand, has a very low rate of lateral growth. Cankers are long and narrow, and girdling takes many years. Infected branches remain alive much longer than in the case of comandra rust, increasing the probability of infections reaching the bole. In cases where the rust reaches the bole from a branch, the rust may never girdle. Such infection can develop into cankers that are several meters long. They can be mistaken for *Atropellis* infections, but older stalactiform cankers almost always exhibit rodent gnawing, and *Atropellis* never.

How do we guard against damage? In the longer term it is certainly possible to breed for resistance to any one or perhaps all three rusts. However I’m not convinced that is wise and it certainly won’t help for the current MPB situation. The current genetic structure of the pine population is the result of long exposure to these rusts, and in my view represents a dynamic equilibrium state that we can learn to live with. True, climate change may throw a monkey wrench into the works, but that remains to be seen. In the short term, the best approach is to establish and maintain high stocking levels until the danger of major damage by these rusts is past, probably around age 15, although that will vary a bit from site to site. Of course, admixtures of other species will provide extra insurance.

The aftermath of the MPB outbreak will bring a new situation for dwarf mistletoe (*A. americanum*). Currently mistletoe occurs throughout the pine region to the south of us here (Smithers, BC), and Peters out quickly as you go north from here. While one can find stands with high DMR ratings, they are not common and mostly restricted to riparian and wet areas, and sometimes situations where the current stand has arisen after cool fires allowing survival of scattered tall infected trees. (Nowadays such stands tend to be preserved as wildlife patches). Large areas of even-aged stands are essentially free of mistletoe, and not subject to invasion by the parasite by lateral spread from infected areas. Long distance spread into such stands, mainly via birds, does occur, but that does not result in damaging levels of mistletoe before maturity, and most such infection foci go unnoticed. A good part of the MPB-killed area will probably never be
harvested, but rather left alone to develop into a new stand. Under these circumstances, scattered small DM foci can develop into significant mistletoe infection. There is not a great deal that can be done about this. When these new stands are eventually harvested, mistletoe can be eliminated again, but in the meantime there will be some damage.

In summary:

- Diseases are not going to disappear, and in all likelihood will be worse than what we see after wildfire or clearcut logging, and for at least two reasons:
  a) conditions in post-MPB stands are likely more favourable for pathogens;
  b) climate change, which will be more drastic here than farther south, is much more likely to than not to favour diseases.

So what can we do? Opportunities to intervene are limited. Much will depend on whether a significant bio-energy industry develops to make use of all the dead, deteriorating timber. If it does, much of the affected area will be roaded and harvested, but if not, the post-MPB stands will mostly be left to develop without intervention.

- Monitor examples of early post-MPB stands to provide early warning of possible problems.
- Favour species mixtures (pine with spruce, Douglas-fir, and hardwoods (aspen)).
- Maintain the natural genetic variability of pine.
- Favour higher densities, particularly for younger stands.
- Pay particular attention to stands that will likely be at harvestable age within the next 50 years or so. Such stands are critical for maintenance of a wood supply for northern BC communities.
Photo Contest Winners

Signs

1st Place Judges Award and Peoples Choice Award
*Armillaria ostoyae* fan by Alan Kanaskie

2nd Place Judges Award
*Pucciniastrum goeppertianum* on evergreen huckleberry by Jeff Stone

3rd Place Judges Award
*Polyporus squamosus* by Angel Saavedra
Landscape-level Concerns after Mountain Pine Beetle: Warren root collar weevil in regenerating pine stands

Brian H. Aukema, Matthew D. Klingenberg, Carolyn Stevens, and B. Staffan Lindgren

The outbreak of mountain pine beetle (*Dendroctonus ponderosae* Hopkins; Coleoptera: Scolytidae; alt. Curculionidae: Scolytinae) in British Columbia, Canada currently covers 8.7 million ha, although the majority of the outbreak in the northern interior of the province has run its course (Aukema et al. 2006). Salvage operations will continue at unprecedented rates throughout the next decade, transforming the landscape into a mosaic of numerous patches of regenerating stands adjacent to killed timber. In the current context of mountain pine beetle disturbance, a significant threat to regeneration is the Warren root collar weevil (*Hylobius warreni* Wood; Coleoptera: Curculionidae) both concentrating in and migrating from residual stands.

The Warren root collar weevil is prevalent in lodgepole pine and mixed lodgepole pine/spruce stands throughout British Columbia. Larvae develop and feed subcortically on the root collars of trees. Large diameter trees typically tolerate feeding, although the feeding wounds may predispose surviving trees to root pathogens. Larvae frequently girdle and kill small diameter trees, and cumulative mortality rates in past studies have ranged as high as 16% (Schroff et al. 2006). Adults may live up to five years, and females lay 25-30 eggs per year. Of these, approximately 25% typically survive to the damaging larval stages (Cerezke 1994).

Salvage logging of stands killed by mountain pine beetle might act to decrease endemic resident weevil populations, but in fact, the opposite may be true. Mortality due to mountain pine beetle will decrease available hosts, and weevils may concentrate on the remaining live stems. After salvage harvesting, larvae will continue to develop in residual stumps (Cerezke 1994), while adults may migrate to nearby regenerating stands, such as within clearcuts. The extent of the latter activity is unclear. Aerial surveys in the Nadina Forest District of British Columbia have noted up to 40% mortality in regeneration openings, frequently within 100m of stands killed by mountain pine beetle. We are currently quantifying this migration and subsequent mortality to subsequent regeneration.

References


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Carolyn Stevens is with the Nadina Forest District, British Columbia Ministry of Forests & Range, Burns Lake, BC V0J 1E0.

Staffan Lindgren is with the University of Northern British Columbia, Prince George, BC V2N 4Z9.
Photo Contest Winners
Symptoms

1st Place Judges Award and People’s Choice Award
*Cristulariella depraedens* on bigleaf maple by Jeff Stone

2nd Place Judges Award
*Dothistroma septosporum* on lodgepole pine
by Dave Weaver

3rd Place Judges Award
*Phytophthora cambivora* canker
by Angel Saavedra
Throughout many areas in northwestern British Columbia, many young-mid seral Lodgepole Pine (*Pinus contorta*) stands have been attacked by Dothistroma needle blight (Woods et al. 2005), while mid seral to mature stands have been attacked by the Mountain Pine Beetle (*Dendroctonus ponderosae*). These attacks are killing a high a percentage of the trees, and where the host species is the dominant tree within the stand, other studies have shown that this results in increased shrub and herb growth. In the attacked stands (Picture 1 and 2) the increased shrub and herb growth is attributed to an increase in light levels as the trees are defoliated, and possibly also due to an increase in the availability of nutrients and water, as a result of the reduced competition for these resources with the death of the trees.

Unfortunately the resulting stand conditions appear to provide ideal conditions for a known herbivore of seedling trees, the Snowshoe Hare (*Lepus americanus*) (Hodges and Sinclair 2005). Hare numbers increase in response to the availability of food and cover, and the dead or dying stands are providing both of these requirements, with the dead trees providing the required over-story cover. In such conditions, under-planting within Dothistroma attacked stands has resulted in a high losses or damage to spruce, cedar, hemlock and sub-alpine fir seedlings, as a direct result of hare browsing.

Under-planting trials have now been established within Dothistroma attacked stands in the Skeena-Stikine and Kalum Forest Districts in northwestern British Columbia, to determine if the high seedling tree losses can be reduced through pre-planting silvicultural treatments, or by planting seedling trees that are less attractive to hares. A report from the initial phase of this work will be available in the spring.

**References**


Figure 1. Dothistroma attacked pine stand.
Photo Contest Winners
Forest Pathologists

First Place Judges' Award and Tied People's Choice Award
Lew Roth Passing Pathology Wisdom to the Next Generation
by Blakey Lockman

Second Place Judges' Award
Everett Hansen in Meditation
by Mike McWilliams

Third Place Judges' Award
Borys Tkacz Contemplating "Danger: tree men at work"
by Everett Hansen
Panel: Climate Change: We've Got No Crystal Balls!
Rona Sturrock, Moderator

Program

Certainties and uncertainties of climate change: global change and local impacts. Andreas Hamann, University of Alberta, Edmonton, AB.

Changing the Climate, Changing the Rules: Global Warming and Insect Disturbance in Western North American Forests (Paper not submitted). Allan Carroll, Pacific Forestry Centre, Victoria, BC.

Predicting effects of climate change on Swiss needle cast disease severity in Pacific Northwest forests. Jeffrey Stone and Leonard Coop, Oregon State University, Corvallis, OR.

Climate Change Effects on Forest Diseases: an Overview. Rona Sturrock, Pacific Forestry Centre, Victoria, BC.
Certainties and Uncertainties of Climate Change: Global change and local impacts

Andreas Hamann

Abstract—This presentation investigates recent climate trends, taking a historical perspective of the Earth's climate over the last century, millennium, half-million years, and half-billion years. The range of projections of climate change over the next century is summarized, focusing on certainties and uncertainties. What is the reason for conflicting climate change predictions? Should we make changes to forest management practices and policies based on predictions by climate modelers? What is the evidence that directional warming observed over the last 50 years will continue and is attributable to human activities? Finally, recent research is presented that demonstrates ecological impacts of recent climate trends in western Canada, highlighting Dothistroma needle blight and mountain pine beetle outbreaks in British Columbia.

Natural Climate Oscillations____

Recent advances in paleoclimatology have revealed a picture of oscillatory climate change operating at multiple time scales and spatial scales. Long term glaciation and deglaciation cycles are worldwide phenomena with global temperature differences of 10-15°C (Petit et al. 1999). Nested within, century to millennia scale anomalies can have continental-scale effects, often beginning and ending extremely abruptly with temperature changes that can regionally be as large as 5-10°C. These anomalies are increasingly understood as cyclical events, although the mechanisms that drive these changes are still debated (Bond et al. 2001). Finally, inter-annual to decadal cycles, such as El-Niño anomalies or the Pacific Decadal Oscillation have significant regional effects on weather patterns (Mantua and Hare 2002). Worldwide evidence indicates that glacial cycles resulted in sweeping species and ecosystem displacements, changes in community composition, and large fluctuation in species population size (Jackson and Overpeck 2000). Century to millennia scale variation in climate has also been linked to major ecological impacts, implying a significant vulnerability of North American ecosystems to warming and associated drought conditions (Booth et al. 2005). Even comparatively small fluctuations associated with decadal cycles have shown clear vegetation response, for example in the form of repeated invasions of alpine meadows (Millar et al. 2004).

Anthropogenic Climate Warming

Global mean annual temperature has increased by approximately 0.6°C over the last hundred years (Folland et al. 2001, IPCC 2001). This is by far exceeds climate fluctuations observed over the last 2000 years (Mann and Jones 2003, Osborn and Briffa 2006) and has generally been attributed to an increase in anthropogenic greenhouse gases, particularly CO₂ (Crowley 2000). It has long been recognized that global climate change as a result of greenhouse gas emissions constitutes a major threat to the earth’s biodiversity (Peters and Darling 1985). Severe and rapid climate change in the past has resulted in major redistribution of species (Alley et al. 2003) and ‘runaway’ greenhouse gas effect at the end of the Permian period, 248 million years ago, has presumably caused the most severe mass extinction event in earth’s history (Benton and Twitchett 2003). In this context, predicted global warming of 3-5°C by the end of the century appears alarming. Even a moderate increase around 2°C predicted over the next 50 years would result in approximately a quarter of all species being on a path to extinction (Thomas et al. 2004). Several review papers suggest that directional global climate change has already affected species (Walther et al. 2002, Parmesan and Yohe 2003, Root et al. 2003, Walther 2004), including severe populations declines and local extirpations (Pounds et al. 1999). If warming trends continue as observed, climate change could potentially surpass land conversion and other forms of habitat destruction by humans as a threat to biodiversity and may even pose a problem for common species that are currently under no threat.

Local Climate Change Impacts__

Biological response is particularly prevalent in the northern sub-boreal, boreal, and sub-arctic ecosystems
where the warming signal is strongest (Zhou et al. 2001, Bogaert et al. 2002, Lloyd et al. 2002, Lloyd and Fastie 2003). Over the past decade, British Columbia (49°-60° latitude) has experienced a warming trend that approximately matches climate change predictions from general circulation models published in the mid 1990s (Johns et al. 1997, Mote 2003). This relatively small increase in mean annual temperature, approximately 0.7 °C for British Columbia, already appears to have had remarkable economic and ecological impacts. The current mountain pine beetle epidemic is the largest documented for this species spreading over 4.1 million ha (Ebata 2004). This outbreak may be partially caused by lack of low winter minimum temperatures, which would normally reduce the populations of this native insect pest by killing the larvae, and by the beetle’s temperature controlled development cycles and lack of diapause (Powell et al. 2000, Logan and Powell 2001, Carroll et al. 2004). A minor native disease, Dothistroma needle blight, which is starting to cause widespread damage in northeast British Columbia, has been causally linked to increased frequency of warm and moist conditions that were experimentally found to be favorable for infections by this fungus (Woods et al. 2005). In the southern and interior of British Columbia, substantial reforestation failures and high frequency of wildfires were attributed to drought in combination with record temperatures, particularly in 1998 and 2003 (Filmon 2004). Although natural variability in climate and non-climatic factors may play a role in these incidents, the current problems observed in British Columbia are consistent with an observed warming signal and illustrate that mitigation and adaptation strategies are urgently needed in the event that future projections of climate modelers turn out to be accurate.

References


Predicting Effects of Climate Change on Swiss Needle Cast Disease Severity in Pacific Northwest Forests

Jeffrey K. Stone and Leonard B. Coop

Abstract—Swiss needle cast disease of Douglas-fir is caused by the ascomycete Phaeocryptopus gaeumannii. Symptoms are foliage chlorosis and premature needle abscission due to occlusion of stomata by the ascocarps of the pathogen, resulting in impaired needle gas exchange. Growth losses of 20-50% due to Swiss needle cast have been reported for about 150,000 ha of Douglas-fir plantations in western Oregon since 1996. Climate factors correlated with pathogen abundance are winter temperature and spring leaf wetness. A model for predicting disease severity based on these factors accounts for 77% and 78% of the variation in one- and two-year-old needles, respectively, for western Oregon sites. A trend of temperatures increasing by 0.2-0.4 °C during the winter months and spring precipitation increasing by 0.7-1.5 cm per decade since 1970 suggests that regional climate trends are influencing the current distribution and severity of Swiss needle cast disease. Forecasts for the Pacific Northwest region predict continued increases in temperatures during winter months of about 0.4 °C per decade through 2050, suggesting that the severity and distribution of Swiss needle cast is likely to increase in the coming decades as a result of climate change, with significant consequences for Pacific Northwest forests.

Background

Swiss needle cast disease of Douglas-fir is caused by the ascomycete Phaeocryptopus gaeumannii (Rohde) Petrak. The disease, and the fungus that causes it, were first described from Douglas-fir plantations in Switzerland and Germany in 1925, and soon afterward reported from various locations in Europe, the British Isles and northeastern North America (Boyce 1940, Peace 1962). The causal agent, P. gaeumannii, was found to be abundant on foliage of diseased trees and was determined to be distinct from any previously described foliage fungi from coniferous hosts. Subsequent surveys of Douglas-fir in the western United States found the pathogen was widespread throughout the Pacific Northwest region, where it had escaped notice because of its inconspicuous habit and negligible effect on its host. Boyce (1940), considered P. gaeumannii widespread but harmless on Douglas-fir in western North America, and probably indigenous to the Pacific Northwest, where “...the fungus has been found at such widely separated localities in British Columbia, Washington and Oregon that it must be considered generally distributed, although harmless, in the Douglas-fir region of the Pacific Coast.”

Figure 1—Symptoms of Swiss needle cast, premature defoliation, loss of older needles.
Since around 1990, unusually severe and persistent symptoms of Swiss needle cast have been observed in Douglas-fir forest plantations in western Oregon, particularly near the town of Tillamook (Hansen and others 2000). Annual aerial surveys conducted since 1996 by the Oregon Dept of Forestry have documented the disease on about 150,000 ha of forest land in the Oregon Coast Range (Figure 2). Unlike Boyce’s (1940) characterization of the pathogen on native Douglas-fir as being inconspicuous and harmless, the fungus is abundant, trees frequently are defoliated of all but current-year needles, and attached foliage is often severely chlorotic (Figure 1). Growth reductions of 20 - 50 % due to Swiss needle cast have been measured in the affected area (Maguire and others 2002). The severity of the problem in Oregon has brought renewed interest in understanding the biology of the pathogen and epidemiology of Swiss needle cast disease. In particular, research has focused on understanding why an inconspicuous, insignificant native pathogen has become a significant forest health problem.

Aerial surveys for Swiss needle cast conducted by the Oregon Department of Forestry have classified patches of Swiss needle cast severity based on foliage discoloration, characterizing the discoloration as being “severe” or “moderate”. The affected area as determined by the aerial survey lies along the entire length of the Oregon coast, extending inland about 40 km, with most symptoms occurring within 30 km of the coast. The crest of the Coast Range forms the approximate eastern edge of the affected area. The aerial survey covers about 1.2 million ha of coastal forest, with the symptomatic area comprising between 50 – 160 thousand ha (Figure 2).

Infection Cycle and Mechanism of Pathogenicity

Ascospores of Phaeocryptopus gaeumannii mature and are released during early May through late July, coinciding with bud break and shoot elongation of Douglas-fir. Ascospores are the only infective propagule, there is no conidial anamorph. Newly emerging foliage of Douglas-fir is most susceptible to infection. Ascocarp (pseudothecia) primordia begin to form in substomatal chambers at 4 – 9 mo following infection. Internal colonization of needles continues as long as they remain attached, so numbers of ascocarps increase as needles age. Normally, fruiting bodies of the fungus are more abundant on needles aged three years or older, and are sparse or absent on younger foliage (Boyce 1940, Hood 1982, Stone and others 2007a). In recent years however, trees having abundant fruiting bodies on current-year needles have been commonly observed in forest plantations along the Oregon coast, with older foliage being prematurely abscised due to the disease (Hansen and others 2000).

The ascocarp primordia completely occupy the substomatal space, thereby rendering the stoma nonfunctional. Occlusion of the stomata by pseudothecia of P. gaeumannii impedes gas exchange and regulation of transpiration, causing impaired photosynthetic activity, and is considered the primary mechanism of pathogenicity (Manter and others 2000, 2003). Estimates of the effect of P. gaeumannii on CO₂ assimilation indicate that occlusion of 25% of stomata results in negative needle carbon budgets, i.e. respiration exceeds assimilation, on an annual basis (Figures 3, 4) (Manter and others 2003).

The abundance of pseudothecia is also highly correlated with needle abscission because of the effect on CO₂ assimilation. It has been suggested that foliage abscission occurs when needles switch from being carbon sources to carbon sinks (Cannell and Morgan 1990). The mechanism of pathogenicity of P. gaeumannii, therefore, can be accounted for by the physical blockage of the stomata and interference with photosynthetic gas exchange. The proportion of stomata occupied by pseudothecia on attached needles seldom exceeds 50%, suggesting that most needles are abscised before more than half the stomata are occluded by pseudothecia, regardless of needle age (Hansen and others 2000). Because the physiological effects of the disease (impaired CO₂ uptake and photosynthesis) are quantitatively related to the abundance of the pathogen (proportion of stomata occluded by ascocarps), pathogen ascocarp abundance is a suitable response variable for assessing effects of climatic factors on disease.

Observations on Climate and Disease Severity

Disease severity (foliage retention, discoloration, crown sparseness, pathogen abundance) has also been monitored annually in permanent plots in the Oregon Coast Range (Hansen and others 2000) equipped with temperature and leaf wetness dataloggers. Foliage retention and abundance of P. gaeumannii ascocarps on one- and two-year-old needles have been monitored annually since 1996 in 9 to 12 Douglas-fir stands, initially aged 12-15 yr. Study sites were selected to represent a range of elevations, distance from maritime influence, and disease severity. Within the area of
Figure 2—Annual variation in surveyed SNC area in western Oregon 1996 - 2006 (A) and combined disease severity survey scores for western Oregon (severity scores for all 11 years summed and superimposed on map, B). Legend signifies 1=disease moderate for single year to 22=disease severe all 11 years of survey. Circed areas highlight regions where chronic SNC has been observed. Source data: Oregon Department of Forestry.

severe disease, symptom severity is variable, but all Douglas-fir show some effects of the disease compared to healthy stands on the eastern slope of the Coast Range and in the Cascade Range. Normal needle retention in healthy Coastal form Douglas-fir is about four years. Within the epidemic area, needle retention varies from about 1.5 to 2.6 years (Hansen and others 2000). Although symptom severity for all sites varies from year to year, relative disease severity is fairly consistent. Disease tends to be more severe nearer the coast, at lower elevations, and on southern aspect slopes, gradually diminishing to the east (Hansen and others 2000, Manter and others 2003, Rosso and Hansen 2003).

The Swiss needle cast epidemic area (Figure 2) corresponds approximately to the Sitka spruce vegetation zone, a narrow strip of coastal forest characterized by elevations generally below 150 m, proximity to the ocean, a moderate climate and a distinct forest type (Franklin and Dyrness 1973). Although Douglas-fir is considered the early seral dominant in the western hemlock vegetation zone, which borders the Sitka spruce zone to the east, its occurrence within the Sitka spruce zone is more sporadic. There Douglas-fir occurs mainly in mixtures with Sitka spruce and western hemlock, but normally not as pure stands as is typical of early post-fire succession in the western hemlock zone (Franklin and Dyrness, 1973).
Figure 3—*Phaeocryptopus gaeumannii* on the underside of Douglas-fir needles with pseudothecia (fruiting bodies) emerging through stomata. A, B. Pseudothecia aligned along stomatal rows. C. Cross section through a stoma showing obstruction of the opening by the pseudothecium (p) between the guard cells (gc).

![Figure 3](image)

Figure 4—Relationship of net carbon dioxide uptake ($A_{net}$) per unit leaf area to [A] *P. gaeumannii* pseudothecia abundance (Disease index, %) and [B] needle retention (%). Taken from Manter and others (2003).

![Figure 4](image)

It has long been suspected that local climate plays a key role in the pathogenicity of *P. gaeumannii*. Boyce (1940) suggested that seasonal patterns in local climate could differentially affect fungal growth and development, and this might explain the greater virulence of *P. gaeumannii* in Europe and the eastern U.S. compared to the area where both *P. gaeumannii* and Douglas-fir are native. A relationship between disease severity and local climate has also been observed in the Pacific Northwest. Hood (1982) found more *P. gaeumannii* in southern British Columbia and western Washington in coastal forests of Vancouver Island and the Olympic Peninsula, with lower levels in the rain shadow of eastern Vancouver Island and the interior, and attributed the difference mainly to precipitation patterns. More severe disease symptoms and greater fungal colonization are commonly observed on lower elevation sites near the coast,
suggesting the possible involvement of maritime fog (Rosso and Hansen 2003).

**Experimental Approaches to Understanding Climate-Disease Interactions**

Because the most severe disease has been observed in sites within the low elevation coastal fog zone, the presence of free water on needle surfaces during the summer has been considered a possible factor affecting disease severity (Hansen and others 2000, Rosso and Hansen 2003). Other investigators have noted a relationship between precipitation patterns and Swiss needle cast severity in the Pacific Northwest (Hood 1982, McDermott and Robinson 1989). Manter and others (2005) attempted to investigate the relative effects of individual climate factors on *P. gaeumannii* abundance experimentally. A factorial design was used to compare the effect of post-inoculation incubation conditions under two levels each of drip irrigation, shade, and intermittent mist on *P. gaeumannii* colonization. Seedling trees were exposed to inoculum in a diseased forest stand, then randomized and maintained under the different post inoculation treatments. Abundance of *P. gaeumannii* ascocarps on foliage was determined monthly. The different post-inoculation conditions resulted in significant differences in *P. gaeumannii* development. But contrary to the expected result, abundance of *P. gaeumannii* was negatively correlated with shade and mist, which was interpreted as being due to their indirect effects on temperature, rather than direct effect of shade or leaf wetness, whereas irrigation had no effect.

**Modeling Swiss Needle Cast**

The finding that small differences in temperature could affect rates of needle colonization and fungal development over the 11-month incubation period of *P. gaeumannii* prompted us to examine the relationship between temperature and *P. gaeumannii* abundance data from field sites in western Oregon. Average daily temperature and cumulative leaf wetness hours were separated into three-month groups, corresponding to major phases in the infection cycle, and subjected to stepwise regression against *P. gaeumannii* distribution data to identify climate factors for use in a disease prediction model. Consistently strong correlations were found between winter (Dec-Feb) mean daily temperature and infection in both one- and two-year-old needles ($R^2 = 0.75 – 0.92$), and this was the only climate variable with $R^2$ values above 0.5. The best-fit climate model included winter mean daily temperature and cumulative spring leaf wetness hours, $R^2 = 0.78$ and 0.77 for one- and two-year-old needles, respectively. When this model was tested against infection data for different sites in different years, a significant 1:1 relationship was found ($R^2 = 0.79$, Figure 5).

![Figure 5](image_url)

**Figure 5**—Best-fit model for predicting *P. gaeumannii* abundance using only climate variables (winter temperature and spring leaf wetness), predicted vs. observed values. From Manter and others 2005.

A more general model was sought that could be used to predict geographic variation in Swiss needle cast severity with the aid of spatial climate models. However, because leaf wetness is not readily available in public meteorological databases, alternative models were tried. Winter temperature was the best single predictor of *P. gaeumannii* abundance in the western Coast Range, but its relationship with infection level varied by year. The abundance of *P. gaeumannii* and severity of Swiss needle cast for a particular site in a given year are not independent of previous year disease severity for the site. Abundance of *P. gaeumannii* in one-year-old needles is partly determined by the amount of inoculum present and the number of ascospores that initiate infection on a needle. Because ascospore infection of needles in their second year is negligible (Stone and others 2007a, Hood and Kershaw 1975), abundance of *P. gaeumannii* on two-year-old needles is partly determined by the degree of colonization present in needles at the beginning of their second year. Therefore two disease components, the amount of *P.
*P. gaeumannii* in two-year-old and one-year-old needles for the previous year, were added to the temperature model to predict *P. gaeumannii* abundance in one- and two-year-old needles for the year of interest. Disease predictions generated by this model had a significant 1:1 relationship \((R^2 = 0.812)\) when compared with observed values in the validation data set (Manter and others 2005).

The temperature/infection model described above was then run over several iterations with temperature held constant over a range of values \((2\) to \(10^\circ\) C). After five iterations, the infection level reaches a stable asymptote for any value of winter temperature. The maximum infection value is a function of winter temperature, regardless of initial infection level, and represents the point of equilibrium between temperature, colonization and inoculum production. A plot of the equilibrium infection level against temperature shows that between 3.77 and 8.90\(^\circ\) C, the range of average winter temperatures measured at coastal study sites, infection levels vary from zero to about 15\% in one-year-old needles and between 10 to about 40\% in two-year-old needles (Figure 6).

![Figure 6](image)

**Figure 6**—A. Simulation of *P. gaeumannii* infection index over time as predicted by the combined winter temperature-infection model, with temperature held constant \((5.13^\circ\) C) and initial infection index of 1\%, and B. Relationship between final equilibrium infection level and winter temperature for one (solid circles) and two-year-old (open circles) needles.

This model also was accurate for predicting disease levels for sites in the western Coast Range \((R = 0.85)\) but was less accurate when infection data from sites from the east slope of the Coast Range and Willamette Valley were included \((R^2 = 0.70)\). Observed infection levels for the inland sites were less than predicted by the equilibrium temperature model, and this is likely due to the comparative dryness of the inland sites. The model that included a term for spring leaf wetness was more accurate in predicting infection levels for the inland sites than the equilibrium temperature model, illustrating that the relative importance of different climate factors can vary spatially. The simple equilibrium temperature model appears suitable for predicting disease levels in the western Coast Range, where spring surface moisture on foliage is probably rarely limiting, but additional parameters are necessary for disease prediction on more inland sites.

The distribution of *P. gaeumannii* also was investigated in New Zealand in 2005 (Stone and others 2007b). Foliage was collected and abundance of *P. gaeumannii* pseudothecia determined for one- and two-year-old foliage from 16 Douglas-fir plantations in the North and South Islands. *P. gaeumannii* was first reported in New Zealand in 1959, and is now
considered established throughout the country (Hood and others, 1990). The study was undertaken to characterize the spatial variation in disease severity and *P. gaeumannii* abundance in New Zealand, and to determine whether climate factors correlated with variation in disease severity in western North America are also correlated with disease distribution in New Zealand. Winter temperature was the best explanatory variable for predicting the abundance of *P. gaeumannii* throughout New Zealand. Univariate models using August minimum temperature or June average temperature both had similar predictive values (*R*² = 0.81, *P* = 0.0001). Both variables were positively correlated with *P. gaeumannii* abundance. These results suggest that variation in Swiss needle cast severity in New Zealand is being influenced by climate factors similar to those identified for the disease in western Oregon, and reinforce the conclusion that winter temperature is a key factor influencing spatial variation in disease severity. When the New Zealand *P. gaeumannii* distribution data were plotted using average winter temperature to compare directly to the western Oregon model of Manter and others (2005) there was no discernable difference (Stone and others 2007b).

The sensitivity of *P. gaeumannii* to relatively small temperature differences helps to explain patterns of spatial variation in Swiss needle cast severity, and suggests that recent increases in Swiss needle cast severity have been influenced by regional climate trends. Over the past century, average temperatures in the Pacific Northwest region have increased by about 0.8°C, with more warming occurring during winter months (Mote and others 2003). Average temperatures for the period Jan-Mar have increased by approximately 0.2 to 0.4°C per decade since 1966 in coastal Oregon and Washington (NOAA 2005). An increase in average winter temperature of 1°C corresponds to an increase in infection index (proportion of stomata occluded) of 3% for one-year-old needles and 6% for two-year-old needles, based on the equilibrium temperature model of Manter and others (2005). Spring precipitation has also increased on average by about 1.6 to 2.6 cm per decade since 1966 (NOAA 2005).

These regional climate trends suggest that over the past few decades conditions become more favorable for growth of *P. gaeumannii*, and have contributed to increasing Swiss needle cast severity. Predictions for continued regional warming of about 0.4°C per decade to 2050, together with increasing (+ 2 to 4%) spring precipitation (Mote and others 2003), suggest that conditions in the region will continue to be favorable for Swiss needle cast development, and could result in expansion of the area affected by the disease beyond the western Coast Range of Oregon. A goal of ongoing research is to develop an improved disease prediction model to investigate the interactions between climate and Swiss needle cast. An expanded SNC disease prediction model will be designed to incorporate long-term climate trend forecasts to enable site-specific short- and long-term disease risk predictions, growth impact predictions, and add various climate change model datasets to allow examination of disease development trends under different climate scenarios.

**Conclusions**

Improved understanding of the effects of climate factors on *P. gaeumannii* abundance now helps to clarify the underlying causes for recently observed increases in Swiss needle cast in the western Coast Range. Previous observations on the regional distribution of *P. gaeumannii* in the Pacific Northwest have suggested a connection between *P. gaeumannii* abundance and spring rainfall (Hood 1982). In the western Coast Range of Oregon, where spring precipitation is abundant, winter temperature has been found to be a highly reproducible predictor of the spatial variation in abundance of *P. gaeumannii* and resulting Swiss needle cast severity, presumably because of its effect on fungal growth. Winter temperature alone is not a satisfactory predictor of *P. gaeumannii* abundance region-wide or in areas where spring precipitation is not as abundant, such as the Willamette Valley, or Oregon Cascade Range, as shown by Manter and others (2005). The predictive disease model described here, therefore, is applicable for predicting spatial variation in *P. gaeumannii* abundance and Swiss needle cast severity only for the western slope of the Oregon Coast Range. Within this area there have been few historical reports of Swiss needle cast, and the disease has been considered an insignificant forest health issue. The natural distribution of Douglas-fir in the western Coast Range has undoubtedly been influenced by *P. gaeumannii* and Swiss needle cast, along with other disturbance agents. The effect of chronic, profuse *P. gaeumannii* colonization of Douglas-fir foliage is to reduce growth rates of affected trees relative to competing species, such as spruce and hemlock. Normally faster growing than western hemlock, Douglas-fir is an inferior competitor where Swiss needle cast disease pressure is high. In the coastal lowlands and interior valleys of the western Coast Range, seasonal climatic conditions are the most favorable for *P. gaeumannii* growth and
reproduction. In these areas, a distinct natural forest type has historically been dominated by western hemlock and Sitka spruce, with Douglas-fir occurring only sporadically. Douglas-fir gradually becomes more abundant in natural forests at higher elevations and further inland, as the Sitka spruce zone gradually merges into the western hemlock vegetation zone, where Douglas-fir is a successional dominant, and where climatic conditions are less favorable for _P. gaeumannii_ growth. This leads to the conclusion that the Sitka spruce vegetation zone occurs as a consequence not only of favorable habitat for Sitka spruce and western hemlock, but also because of the inhibition of their main competitor, Douglas-fir, due to Swiss needle cast disease. This scenario also suggests that the severity of Swiss needle cast in the region may be the result of recent forest management in the western Coast Range, where Douglas-fir has been strongly favored in forest plantations because of its greater economic value, increasing the abundance of the host species in the area most favorable for growth and reproduction of the pathogen. As noted above, however, recent climate trends also are likely to have contributed to current Swiss needle cast severity. Furthermore, forecasts of future climate trends for the Pacific Northwest suggest a probable expansion of the area affected by severe Swiss needle cast beyond the Pacific Northwest suggest a probable expansion of the area affected by severe Swiss needle cast beyond the western Coast Range as winter temperatures and spring precipitation continue to increase, resulting in greater disease pressure on Douglas-fir stands further inland.

**Acknowledgements**

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**Literature Cited**


Climate Change Effects on Forest Diseases: an overview
Rona N. Sturrock

Introduction

Global climate change (CC) does occur naturally but the rate and magnitude of change we are seeing today is believed to be both faster and larger than has ever occurred before. A broad spectrum of scientists believes that this unprecedented change in the Earth’s climate is due largely to human activities. Fossil fuel combustion, deforestation, and animal production have all contributed to significantly increased concentrations of greenhouse gases (principally carbon dioxide (CO$_2$), methane (CH$_4$), and nitrous oxide (N$_2$O)) in the Earth’s atmosphere since the time of the Industrial Revolution about 200 years ago. An increase in the atmosphere’s heat-trapping (i.e., greenhouse) gases means that the Earth’s surface is being increasingly warmed. Global climate change and its associated effects, including increased air temperatures, more severe weather, and rising sea levels, will have an impact on all species, including those occurring in the world’s forests.

About one third of the Earth’s land area is covered by forests. Half of these forests occur in the tropics and half in the temperate and boreal regions of the world. Healthy, sustainable forests are critical to the economic, aesthetic and spiritual health of humans. Indeed, forests are vital to the ebb and flow of carbon in many of the world’s ecosystems. Boreal forests, occupying about 10% of the Earth’s land cover, are expected to undergo some of the most extreme CC-induced changes. Boreal forests could decrease by up to 50%, whereas tropical and temperate forests may actually expand by up to 20% (Krankina et al. 1997).

This paper provides a summary of some of the principle potential effects of climate change on forest species and forest pathogens (figure 1), forest species - pathogen interactions, and the management of these proposed effects.

Potential Climate Change Effects and Host Responses

- Plant species ranges will shift and forest tree species compositions will change. Due to their long-lived nature, slow rate of migration and existing fragmented populations (due to land use patterns), many forest plant populations will not have sufficient time to adapt to altered climates. Forest species favoured under a quickly changing climate will be those already well-adapted to disturbance, plus those species with high migration rates and early sexual maturity.
- Overall, the productivity of forests may improve due to increased CO$_2$ and increased N levels (due to higher mineralization and decomposition) but this may be offset in many cases by the detrimental effects of drought and increased occurrence of disturbance (e.g., fire).
- Changes in interspecific competition among tree species (under elevated CO$_2$) could result in the development of novel forest types.
- Increasingly warmer and drier climate of some regions will begin to influence forest ecosystems as existing stands become less adapted to prevailing conditions; this may result in episodes of regional ‘forest decline’.
- CC is a serious threat to some rare plant species, which are often characterized by specialized environmental requirements and low genetic diversity.
- Conservation of biodiversity may be threatened by CC, especially where CC alters habitat critical to species’ survival.
Figure 1—Diagram illustrating the complex relations between human activities, climate change effects, forest host species and pathogens and other factors in forest ecosystems.

Potential Climate Change Effects and Pathogen Responses

- Pathogen distribution changes caused by CC are likely closely tied to shifts in host distribution. Thus, CC may induce the latitudinal extension of those pathogens requiring warm temperatures.
- One outcome from pathogens expanding their ranges and contacting ‘new’ hosts and/or vectors may mean that new pathosystems will emerge.
- CC may amplify the impact and aggressiveness of pathogens; it may also change the status of weak/opportunistic pathogens such that they are able to infect and damage stressed tree hosts.
- Wetter springs in some regions may result in increased foliage diseases
- Increased drought stress of hosts in many regions may mean increased mortality from root pathogens.
- Interactions between pathogens may change.
- CC may alter the balance between pathogens and their natural enemies.
- When combined with CC, trends in increasing invasions by non-native pathogens means that new epidemics may occur.

Potential Climate Change Effects and the Host X Pathogen Interaction

- Almost every study and review of CC effects on forests has a common caveat – that the complexity of forest ecosystem relations and the paucity of predictive scientific data means that it is difficult to predict the effects of CC
on many forest factors, including host-pathogen interactions’
- CC could “alter stages and rates of development of [the] pathogen[s], modify host resistance, and result in changes in the physiology of host-pathogen interactions.” Coakley et al. 1999
- The relative impact of CC will likely be different for native versus introduced host and pathogen systems; native, co-evolved systems are likely better ecologically buffered and therefore will likely be more inherently stable under the pressures of CC.
- The fitness of beneficial and/or protective mycorrhizal fungi may be affected by CC effects on, for example, soil temperatures and soil moisture.
- Soil microbes: climate-induced effects on the abundance and diversity of soil microbial communities may influence ability of ecosystems to adapt to changing conditions.

Case Studies_________________

The effects of climate change on forest tree diseases can be predicted to some extent by evaluating the existing roles of climate and weather fluctuations on pathogen survival and behaviour. Results of such evaluations, conducted for the most prevalent and economically important diseases (including proposed high risk alien pathogens) of forest tree species in a given region, are available in tabulated form or as case studies in several publications (e.g., Lonsdale and Gibbs 2002, Boland et al. 2004). Included here are summarized case studies for two important forest pathogens and for the condition called ‘forest decline’.

1. Phytophthora cinnamomi

Current condition
- Very wide host range; causes root and stem-base diseases of conifers and hardwoods throughout the world.
- Pathogenic activity greatest at 25-30°C but can grow at 5°C; particularly destructive in sub-tropical and Mediterranean climates.

Predicted Future
- Based on a model developed by Brasier and Scott (1994) (considered to predict the effects of CC with reasonable confidence because of its use of high quality data on the activity and distribution of the fungus), P. cinnamomi is predicted to increase its activity in temperate zones in the N and S hemispheres (i.e., to have considerable northward expansion and a decrease in the tropics). Under changing climatic conditions the fungus is expected to cause more damage to existing urban and forest tree hosts and to expand the number of species it can infect.
- Also predicted is a similar trend for many invasive tree-infecting Phytophthoras (e.g., alder Phytophthora and P. ramorum)

2. Armillaria spp.

Current condition
- Very wide host range; Armillaria spp. cause root diseases of conifers, hardwoods and shrubs.
- Establishes many infection points; host responses (e.g., callusing) often slow down Armillaria spp.

Predicted Future
- Highly pathogenic Armillaria spp. may be assisted by the impairment of host tolerance caused by CC-induced stress; this may result in lower pathogenic species also becoming more successful on stressed trees.
- As site conditions become drier, increased cumulative growth reduction and mortality losses in BC could increase from 32% (present state) to as high as 67% (stressed state in 100-year-old Douglas-fir stands (Cruickshank and Morrison 2003).

3. Forest Declines

Current condition
- Forest declines represent one of the few examples where a strong association between CC and disease incidence/severity has been established for several forest species (e.g., ash, birch, balsam fir and maples in Europe and North America) and proposed for others (e.g., yellow-cedar decline in Alaska and cypress decline (mal del cipres) in Argentina).

Predicted Future
- In a changing climate with increased temperatures, evapotranspiration, and extreme weather events, there will be an increase in the frequency and severity of stress factors, which may lead to more frequent forest declines.
Managing and Mitigating Climate Change Effects on Forest Diseases

Management and mitigation of CC effects on forest diseases will require consideration of several possible approaches by researchers, forest and pest managers, and politicians/policy makers alike. Some of the key approaches are outlined below.

Acquisition and/or Synthesis of Basic Knowledge

- There is a need to improve understanding of the biology and epidemiology of native and non-native pathogens; what is their adaptability to CC effects?
- Gene expression studies of hosts and/or pathosystems should be conducted to unravel the effects of multiple stressors.
- There is a need to utilize new techniques that enhance identification and development of host tolerance/resistance.
- Diagnostic arrays should be applied to monitor changes in pathogen populations over time and space.

Genetic Management of Forest Species

- A forward-looking program is needed to respond to CC.
- There should be a determination of how much help is required by each species.
- Climate-based seed zones need to be updated.
- Widely adapted and diverse nursery stock should be deployed/encouraged.
- Breeding programs should promote genetic diversity, pest resistance, tolerance to environmental stresses, etc.

Modeling

- Closer links between empirical and modeling studies could support more rapid progress in understanding CC effects.
- Models are potentially powerful predictive tools but they have at least three continuing problems (Scherm 2004): 1) model inputs may have a high degree uncertainty (e.g., disease distribution), 2) non-linear relationships between climatic variables and epidemiological responses are common, which means there may be insufficient data for clear prediction, and 3) the potential for adaptation by plants and pathogens is often ignored in models.
- There is a need for region and species/pathosystem specific spatial and quantitative data and for the combining of CC models with epidemiological or population-dynamic models.

Monitoring

- Existing monitoring efforts should be expanded and improved because of an expected increase in the number of new, introduced plant diseases.
- There is a need to determine the likelihood/risk of the arrival and subsequent spread of new diseases (use models; McKenney et al. 2003).
- Additional controls to prevent the arrival of new pathogens should be developed and implemented.
- There is a need to increase the development and/or utilization of diagnostic tools, adequate strategies, and personnel to detect pathogens.

Modification of Silvicultural Systems, Regeneration and Stand Management Practices

- Under CC, we will need to maintain forest vigour during a progressive disequilibrium between forest vegetation and climate. This will require systems (e.g., ‘early’ harvest, thinning, partial cutting, underplanting) that address the management and regeneration of declining stands.
- Forest managers must be prepared for novel forest types requiring new silvicultural approaches.
- Extensive artificial regeneration may be required to assist the migration of tree species and/or genotypes; what is the timing required for each species?
- The use of plant mixtures and planting of native trees, which are better ecologically buffered, should be encouraged.
- Forest managers should be prepared for even greater success of weedy competitors; also, they should try to maintain/encourage the
abundance and diversity of soil microbial communities, including mycorrhizae.

**Summary and General Conclusions**

There is consensus among climatologists and most others (some politicians) that global warming is occurring and is linked to human activity.

- It is difficult to generalize about the effects of CC on forests/forest diseases since the effects will tend to be different for different pathosystems in different locations.
- Forest species will shift their ranges - some may disappear and some new forest types may emerge.
- CC is likely to be broadly detrimental to tree health and will favour some very damaging pathogens (Brasier 2005).
- CC can have a positive, negative, or neutral impact on individual pathosystems because of the specific nature of host-pathogen interactions; this means that there are no real rule(s) of them regarding specific CC impact assessments.
- Host-pathogen interactions involving non-native hosts and invasive pathogens are likely at highest risk to the effects of CC.
- Approaches to managing and mitigating the effects of CC on forest diseases include the acquisition and/or synthesis of basic knowledge about tree species and pathosystems, the development of better models that will yield more accurate predictions and increase the certainty of what they predict, forward looking and dynamic breeding and forest management practices, and the development of tools and policies to prevent the arrival of invasive pathogens, or to deal with them, if they do arrive.

**Literature Cited and Some Selected Key References**


Panel: Foliage Diseases: The bare-naked trees!

Jeff Stone, Moderator

Program

A Brief Summary of Historical Forest Insect and Disease Survey Findings on Pine Needle Casts in British Columbia (Paper not submitted). Brenda Callan, Pacific Forestry Centre, Victoria, BC.


Genetic diversity in the fungal pathogen Dothistroma septosporum. Angie Dale and Kathy Lewis, University of Northern British Columbia, Prince George, BC.

What effects will a changing climate have on lodgepole pine in British Columbia? Alex Woods, British Columbia Forest Service, Smithers, BC; and Greg O’Neill, British Columbia Forest Service, Vernon, BC.
Foliar Diseases of Pine – The New Zealand Experience

Lindsay S. Bulman

Introduction

The great majority of New Zealand plantation forest area, over 90% of the total 1.7 million ha, is comprised of *Pinus radiata*. This species is relatively healthy and does not suffer many of the serious root rots, rusts, and insect pests that are problematic in other parts of the world. Radiata pine suffers from three significant foliar diseases – Dothistroma needle blight, Cyclaneusma needle cast and physiological needle blight (PNB). This article summarizes work done in New Zealand on these three diseases.

Dothistroma Needle Blight

Dothistroma needle blight was first discovered in New Zealand in the early 1960s, and was formally identified as *Dothistroma pini* Hulbary in 1964. The sexual stage of the fungus (*Mycosphaerella pini* Rostrup ex Monk) is not found in New Zealand. Immediately, a research programme was started to examine:

- Infection process
- Infection period, effect of light and weather
- Optimal spray regimes
- Host susceptibility and response

Gadgil (1967, 1974, and 1977) found that prolonged needle wetness is needed to initiate and continue infection – continuous wetness at 20°C/12°C day/night temperature resulted in appearance of stromata after 14 days. After conidia germinate, hyphae grow haphazardly, penetrate stomata, and advance only in disrupted mesophyll. Bassett (1970) showed that dothistromin is present. Infection period is summer, when temperatures are over 16°C. Infection can occur at 7°C but wet conditions and many spores (i.e. >6 million per seedling induced 15% disease at warmer temperatures) are needed.

The role of dothistromin is uncertain. A relationship between pathogenicity or virulence in the field has never been established. Production of dothistromin in culture is extremely variable (Bradshaw and others 2000). Franich and others (1986) proposed that lesion development is caused by benzoic acid production in response to dothistromin. Significant differences between lesion length induced by doses of dothistromin on resistant and susceptible trees were found, but such relationships were weak.

Environment plays a critical role in disease development. A combination of consecutive wet summers, trees growing closely together, and no aerial application of fungicide has resulted in tree mortality in New Zealand. In one experiment mortality reached 12% but effects of Dothistroma needle blight were confounded with *Armillaria* root rot (Woollens and Hayward 1984). New Zealand does not have the variety of damaging bark beetles and root diseases that can attack and kill trees weakened by Dothistroma needle blight. There is a strong relationship between summer rainfall and disease. Temperatures in spring and summer in New Zealand are high enough to promote infection and are not limiting. Figure 1 shows the relationship using data gathered from a large forest growing in the central North Island of New Zealand.

![Figure 1—Relationship between summer rainfall and disease.](image-url)
From October 2003 to August 2005 lesion and stromatal development was monitored monthly on radiata pine trees at three locations. The monitoring proved to be problematic because the work was tedious and counting individual stromata on individual needles was difficult. Not only that, as disease developed on one cohort of needles those needles fell off and had to be replaced by healthier ones. This disrupted monitoring the progress of numbers of stromata and lesions significantly. However, preliminary results indicate that the primary infection period is in late spring, followed by another in late summer or early autumn. This late infection period was unexpected. Models based on our knowledge of disease development and the empirical data gathered in the monitoring experiment are now being developed.

Dothistroma needle blight causes significant economic loss in New Zealand (see figure 2). Volume growth loss is proportional to average disease level (Van der Pas 1981) and annual loss is in the order of $NZ24 million per year.

![Figure 2](image_url)  
**Figure 2**—Relationship between diameter growth and Dothistroma disease level.

However, aerial spraying copper fungicide provides effective control. The fungicide kills conidia-bearing stromata and thus reduces inoculum. It also protects foliage from fresh infection. It is the only example in the world of successful control of a forest pathogen by aerial application of fungicide. Control has also been achieved by the development of resistant breeds where gains of 10 to 12% have been reported experimentally (Carson 1989).

Dothistroma needle blight can be controlled by cultural techniques. Stands grown on pulpwood regimes (660 to 1630 stems per ha) were sprayed on average 5.5 times during the period when they were susceptible to the disease (when trees were aged between 1- to 15 years-old). In contrast, stands managed on a clearwood regime (lower branches pruned and stocked at 300 to 350 stems per ha) were sprayed far less frequently. About 75% of these stands in a forest growing in similar conditions to the forest managed on pulpwod regimes were sprayed zero to 2 times during the susceptible period.

A great deal of research has been carried out on Dothistroma needle blight but there are still things to learn. For instance, the relationship between dothistromin production and virulence or pathogenicity in the field is unknown. Dothistroma should be treated as a biosecurity risk even though the fungus already exists in New Zealand. Like all forest pathogens, we need to maintain a watch for all new subspecies, races, formae speciales, even if the species is already present (Gadgil and others 2002). The factors that induce hyphae to penetrate stomata are unknown. We have good data on performance of resistant breed in field trials but performance when planted in large contiguous areas has not been monitored.

**Cyclaneusma Needle Cast**

Cyclaneusma needle cast, caused by *Cyclaneusma minus* (Butin) DiCosmo *et al.*, affects susceptible radiata pine individuals scattered among non-susceptible individuals. The same individuals are affected year after year, where one-year-old needles turn yellow in spring and are readily cast. A less severe needle cast often occurs in autumn. The main infection period is autumn and early winter and infected needles are cast about six months afterward. Cyclaneusma colonises needles first, followed by many fungi such as *Lophodermium* spp (along with *Cyclaneusma* the most common species) and *Strasseria*, *Hendersonia*, *Cladosporium*, and others.

There are at least two types of *Cyclaneusma minus* in New Zealand, based on morphological characteristics. One type is more common than the other, but no association between type and pathogenicity has been seen. Disease incidence and severity is worse on moist sites (gullies or high altitude sites prone to mist). Disease is most severe in trees ages between 6 and 15 years old, with trees younger than 5 years and older than 20 years very rarely affected.

Cyclaneusma causes significant growth loss where an average disease severity of 80% will result in a volume increment loss of 60%. Control methods include breeding. This should offer a good opportunity because disease occurs on the same individuals year after year. The breeding programme has selected for
good needle retention so by default over the years has selected against Cyclaneusma needle cast. The other control method available is to selectively remove susceptible individuals during thinning operations. However, this method has some operational difficulties because the disease is only apparent over a short time of year and doesn’t show until age 6, by which time the first thinning and pruning operations have usually taken place.

Research priorities include identification of high risk sites so control measures can be carried out, determining the fungal genetic population variation, and determining what induces Cyclaneusma to move from an endophyte to a pathogen in some individuals. The fungus can be readily isolated from needles of susceptible and resistant trees during the peak disease expression period.

Research on Cyclaneusma needle cast in New Zealand is summarised in Bulman and Gadgil 2001.

**Physiological Needle Blight**

A disorder, now named Physiological needle blight (PNB) has been recorded in New Zealand since the early 1980s. Over the last 10 years, sporadic outbreaks have been recorded in certain regions every 2 to 3 years. Almost exclusively trees older than 14 years are affected. Distribution is patchy within a stand and defoliation is usually severe (Figure 3). Often 80% of the crown will be affected.

Needles droop and wilt, but remain firmly attached to the branch. Many fungal isolations from affected needles were carried out and only non-pathogenic fungi were obtained. Similar symptoms were induced in glasshouse studies where foliage of age cuttings was kept continuously moist for over 8 weeks. Applications of broad-spectrum fungicide reduced the incidence of needle death. Fungi colonising the needles were also reduced but were not eliminated. It is probable that the natural saprophytic fungal colonisers accelerate death of needles in a severely stressed state. An association between reports of outbreaks and above average rainfall and raindays in June and July was found after extracting forest health reports. This finding was supported by results of aerial surveys undertaken in 2002 and 2003. Thus, experimental work has indicated that the disorder is physiological in origin although the physiological nature of the needle breakdown is not fully understood.

Research in progress involves pre-dawn water potential measurements to determine root function. Results were not conclusive, but may have indicated that root waterlogging may be contributing to the disease. The age effect may be due to increased resistance and decreased water conductance with tree age.

Work on PNB is ongoing.

**References**


Bradshaw, Rosie E.; Ganley, Rebecca J.; Jones, W. T.; Dyer, P. S. 2000. High levels of dothistromin toxin produced by the forest pathogen *Dothistroma pini*. Mycological research 104(3) 325-332.


Gadgil, Peter D. 1977. Duration of leaf wetness periods and infection of *Pinus radiata* by...


The Status of Red Band Needle Blight in Great Britain
Anna Brown

Abstract—Since the late 1990s, the incidence of red band needle blight in Britain has increased very significantly. The disease was initially reported in the East Anglia Forest District in one stand of Corsican pine (Pinus nigra ssp. laricio) in 1999 and since then it has become a serious disease problem throughout the forest district. Annual surveys are ongoing to assess the extent and severity of the disease. Results indicate that the disease affected a total of 8,860 hectares (60%) of the Corsican pine crop in 2003 and by 2005, this figure has risen to 10,198 hectares (73%). In 2006, a survey of all Corsican pine stands in Britain under the age of thirty years revealed that the disease was present in the majority of Corsican pine growing districts with 71% of Corsican pine stands found to be infected. The threat this disease poses to lodgepole pine (Pinus contorta latifolia) which is grown mainly in Scotland is also of concern.

Background_________________

Red Band Needle Blight (RBNB) is an economically important tree disease (Gibson, 1974) with a worldwide distribution which is believed to have been aided by the introduction of contaminated exotic pines (Bradshaw, 2004). As the disease has the potential to cause severe damage to susceptible hosts, it is listed under EC plant health legislation. The causal agent is Mycosphaerella pini E. Rostrup apud Munk (Synonym Scirrhia pini Funk & A. K. Parker) which is the teleomorph of the Ascomycete fungus often referred to as “Red Band Needle Blight”. More often, the pathogen is found as the anamorph which until recently was known as Dothistroma pini Hulbary (synonym Dothistroma septospora Morelet (Dorog.), Cytosporina septospora G. Dorogueine). However, recent molecular work by Barnes and others (2004) has found two separate species can cause the disease and have suggested that the epithet D. pini should be applied to the species restricted to North-central America, while Dothistroma septosporum used for the species found elsewhere.

Historical Disease Status_______

RBNB was first recorded in UK in 1954 (Murray and Batko, 1962) on a range of pine species at Wareham nursery, Dorset. Disease outbreaks reoccurred
sporadically at the same location until 1966 and there were two occurrences in Wales on plantation trees in 1958 and 1989. However, apart from these records there were no reports of the disease until three outbreaks in the late 1990s. Since the late 1990’s, the incidence of the disease in the UK has increased markedly, particularly on Corsican pine (Pinus nigra spp. laricio). Between 1999 and 2002 there were 22 reports of the disease in Great Britain (see Figure 1). Five of these were on privately owned Corsican pine plantations. The remaining 17 reports were from the Forestry Commission estate, 11 of which occurred in forest blocks within the same forest district, East Anglia. In addition to Corsican pine, the disease was also reported on Ponderosa pine (Pinus ponderosa) in Bedegbury pinetum, located in Kent, east England, and on Jeffreys pine (Pinus jeffreyi) at Forest Research, near Edinburgh, Scotland.

**Figure 1**—Distribution of Red Band Needle Blight up until 2002. White = red band outbreaks pre 1995, black = red band outbreaks 1989-2002 on the Forestry Commission estate, grey = red band outbreaks post 1989-2002 on private forestry estates.

**Current Disease Status**

**Extent and severity in the East Anglia Forest District 2003 –2005**

The East Anglia Forest District is the largest Forestry Commission Corsican pine growing area in Great Britain. Of a total forest area of 25,000 hectares, approximately 14,000 hectares is Corsican pine. The number of reports of RBNB in the East Anglia Forest District resulted in extensive surveys of the Corsican pine between June and August, in 2003, 2004 and 2005 to determine the extent and severity of the disease. Fifty trees in 119 stands (a total of 5,950 trees) were individually assessed for a range of parameters including the percentage of crown infected. Based on the number of infected trees and the levels of crown infection, predictive values have been estimated to give an indication of the extent and severity of infection within the district.

Based on this information, it was estimated that in 2003, 61% (8,684 hectares) of the total area planted with Corsican pine was infected with RBNB. By 2004 this figure had risen to 66% (9,186 hectares) and by 2005, 73% (10,198 hectares) was infected (Table 1). Mean crown infection also increased significantly between 2003 and 2005 from 27% to 33% ($p=0.001$; Table 1). Age of the trees was found to influence the probability of infection occurring ($p=0.001$) with all Corsican pine under the age of 10, and 99% aged 11-30 likely to be infected. In contrast, only 55% of trees more than 30 years old were infected although it should be noted that the disease was observed on trees older than 70 years. Age was also found to have a significant effect on disease severity ($p=0.001$), with trees aged between 11 and 30 showing the greatest levels of infection. In 2003, mortality was only recorded in 1% of stands that underwent assessment, whereas by 2005 mortality was observed in 14% of the 119 stands assessed (Table 1).
### Table 1—Predicted area (percentage and hectares) of infected Corsican pine, the percentage of crown infection and percentage of stands with mortality in the East Anglia Forest District.

<table>
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<tr>
<th>Year</th>
<th>Total Area (Ha)</th>
<th>% Area infected</th>
<th>Area Infected (Ha)</th>
<th>Mean % Crown Infection</th>
<th>% of Stands with Mortality</th>
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### Extent and severity in Great Britain 2006

The total area of Corsican pine growing on the Forestry Commission estate was calculated at 33,952 hectares in 2006. In the same year all Corsican pine stands under the age of 30 years were assessed for RBNB in Great Britain. This survey comprised 2,928 stands covering 10,574 hectares within all eleven forest districts in England, three out of four districts in Wales and six out of fourteen in Scotland. Due to the scale of the survey the disease was assessed as either being present or absent within a stand but not quantified for severity. The survey revealed that the disease was present in all forest districts surveyed in England and Wales and half of those assessed in Scotland (Figure 2). Overall, the disease was found to be present in 71% of the stands assessed covering a total of 7,051 hectares. Based on the results of the East Anglia survey where 55% of the Corsican pine over the age of 30 years was found to be infected, it can be inferred that across Great Britain a further 12,858 hectares are likely to be infected bringing the total affected by disease to 19,909 hectares. This is approximately 60% of Corsican pine crop in Great Britain. Red Band Needle Blight was also reported on a further 13 pine species in Great Britain during this survey, with lodgepole pine the second most frequently infected species.

### Discussion

The extent and severity of red band needle blight on Corsican Pine in East Anglia Forest District has increased significantly since the first reports in 1999. In addition, surveys carried out across Great Britain in 2006 have shown that the extent of the disease is much more widespread than previously understood, with the majority of Corsican pine stands under the age of thirty showing signs of infection. The reason for this rapid increase in disease occurrence and severity is likely to be twofold. Firstly, red band needle blight has been identified on pine plants in several nurseries in England since 2004 and, as has been the case in other countries (Bradshaw 2004), the movement of infected stock is likely to have aided the long distance dispersal of the disease. Secondly, as with British Columbia (Woods, 2005), climatic conditions since the late 1990’s have been favourable to the disease in Great Britain with an increased incidence of warm and wet springs and summers.

Red Band Needle Blight is likely to have a significant impact on British forestry over the coming years. Until this current disease outbreak, Corsican pine was increasingly the species of choice; its form and timber properties encouraged widespread planting and it was considered as a key species for the future in much of England because of climate change predictions (Broadmeadow, 2005). Reports from the 2006 survey of the disease on lodgepole pine are also of great concern in the context of the damage being caused to this species in British Columbia (Woods, 2005). There is approximately 73,000 hectares of lodgepole pine on the Forestry Commission estate, more than double that of Corsican pine, so the potential for economic losses are considerable.
Acknowledgements

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References


Genetic Diversity in the Fungal Pathogen *Dothistroma septosporum*

Angie Dale and Kathy Lewis

A widespread, severe outbreak of *Dothistroma septosporum* on native lodgepole pine in northwest British Columbia has prompted several questions about the factors contributing to disease including the genetic structure of the fungus. The existence of both the anamorph and teleomorph in B.C. forests has led to the assumption that the fungus is able to sexually reproduce and has the ability to rapidly evolve, possibly into more virulent strains.

The purpose of this study was to explore the genetic structure of 21 *Dothistroma septosporum* populations from 3 different biogeoclimatic subzones, in both remote natural stands and plantations. Population genetic structure was analyzed using data from amplified fragment length polymorphism (AFLP) and from 4 microsatellite loci. Preliminary analysis on 2 populations using analysis of molecular variance showed a significant difference between the populations (phi PT 0.44, probability 0.001). There were no clones identified in the two populations. There was no diversity found in the 4 microsatellite loci.

The high level of genetic diversity in the AFLPs and the lack of clonal structure suggests that sexual reproduction and ascospore dispersal may play a more important role in the spread of the fungus than once thought and should taken into account in silviculture planning.


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What effects will a changing climate have on lodgepole pine in British Columbia?

Alex Woods and Greg O’Neill

Abstract—The foliar compliment of evergreen conifer trees is dependent on the crown ratio and foliar longevity. Variation in foliage longevity is one of the important traits that often favours the genus *Pinus* over its competitors. Healthy lodgepole pine trees throughout British Columbia, Canada typically retain needles for 4 to 5 years. Foliar diseases have a profound influence on foliar longevity and crown ratio, and lodgepole pine is susceptible to a large suite of foliar pathogens. We assessed the foliar longevity, live crown percent and mortality of lodgepole pine trees at 25 lodgepole pine provenance test sites in central BC, and correlated these values with changes in climate at each site between the decade of the 1920s and the 1990s. We found strong relationships between increases in August minimum temperatures and live crown percent ($R = -0.75$) and mortality ($R = 0.75$). Sites in Region 7 of the trial (Robson Valley) consistently have the least foliage and have consistently experienced the greatest increases in August minimum temperature and July precipitation, and the greatest decreases in May maximum temperature. Region 9 (Nechako Plateau) sites consistently have the most foliage and have consistently experienced the least change in August minimum temperature, July precipitation, and greatest increases in May maximum temperature. Future changes in climate in conjunction with foliar pathogens could have profound effects on the health of lodgepole pine in BC.

Introduction_________________

Climate change could influence plant disease by altering biological processes of the pathogen, host, or disease spreading organism (Harvell et al. 2002). In some instances, changes in precipitation regimes, though more difficult to predict than temperature (Barron 1995), could have the larger influence on forest productivity of the two climatic factors. Increases in summer precipitation could benefit some tree species (Nigh et al. 2004) but could also have unexpected impacts on tree growth if changes in moisture regime favours foliar pathogens (Woods et al. 2005). Foliar disease fungi may be some of the more responsive forest disease organisms to climate change (Woods et al. 2005).

The foliar compliment of evergreen conifer trees is dependent on the crown ratio (Koch 1996), the ratio of crown length to total tree height, and foliar longevity. Longevity of foliage plays an important role in the overall life history of a tree (Schoettle and Fahey 1994). Variation in foliage longevity is one of the important traits that often favours the genus *Pinus* over its competitors (Schoettle and Fahey 1994).

Foliar pathogens can have a profound influence on foliage longevity and therefore, on the competitive advantage of a species. Foliar diseases result in premature leaf mortality causing leaves to be shed prior to the end of their productive lives which leads to a loss of photosynthetic capacity and nutrients (Schoettle and Fahey 1994). Many foliar pathogens tend to attack needles in the lowest portions of tree crowns first, which often results in reduced crown ratios in infected trees. Lodgepole pine (*Pinus contorta* var. *latifolia* Dougl. ex Loud.) is host to a suite of foliar pathogens (Sinclair and Lyon 2005). These foliar diseases, under the influence of climate change, could potentially reduce any competitive advantage that a species like lodgepole pine may have had.

Until recently, foliar diseases in natural forests have generally had a minor impact due to high levels of host resistance (Harrington and Wingfield 1998). Woods et al. (2005) have reported on a shift to this generalization occurring in northwest BC, where Dothistroma needle blight (*Dothistroma septosporum* Dorog. (Morlet)) is killing mature, native lodgepole pine, an unprecedented phenomenon they suggest is linked to climate change. Woods et al (2005) suggest that perhaps an environmental threshold for Dothistroma needle blight has been surpassed in northwest British Columbia allowing the foliar disease to overcome a previously established balance between host and pathogen.

The objectives of this study were to: (1) determine what if any significant changes in climatic variables...
have occurred between the period 1991-2000 and the decade of 1920s in the study area using the climate model ClimateBC v2.2 (Wang et al. 2006), (2) determine if any changes found in the previous objective could have had a significant influence on foliar diseases and thus the foliar longevity and foliar compliment in the Illingworth lodgepole pine provenance trial (Illingworth 1978), and (3) assess the risk to lodgepole pine management posed by foliar diseases in central BC in relation to any changes to the current climatic conditions.

**Methods**

**Illingworth Lodgepole pine Provenance Trial**

In 1974 field test sites were established at 60 sites (Fig 1a) with three-year-old seedlings grown from seeds collected from 142 natural populations of lodgepole pine (Fig 1b) (Illingworth 1978).

**Figure 1a and b**—The Illingworth lodgepole pine provenance trial (EP657.06) showing site locations (1a, below) and provenance locations throughout the range of lodgepole pine (1b, right). In this study we examined the trial sites circled in 1a (from Ying et al. 1985).

To facilitate the location of test sites, twelve broad biogeoclimatic regions were delineated, primarily on the basis of latitude and moisture regime (Ying et al 1985). The Illingworth trial is considered one of the most comprehensive provenance tests in the world (Wang et al. 2006). In this study we examined the 25 sites of regions 7-11 of the provenance trial. Each trial site contained two replicates of 60 families, 9 trees per replicate per family, for a total of 1080 trees per site.

**Foliar Compliment Assessment**—We assessed trees for the extent of their foliar compliment as follows (Fig 2):

- First, we estimated the crown ratio, the extent of live crown to the nearest 5% by visualizing a line perpendicular to the stem at the lowest point of the crown occupied by full green internodes on > ¼ of the branches.
- Second, we recorded the number of years of needle retention (internodes) with > 50% green needles from a representative branch in the middle of the live crown as estimated in step one. In cases where only half of the current year's needles were present, the internode was recorded as 1/2 rather than 1.
By assessing a branch from the middle of the live crown we accounted for the fact that foliar disease damage is typically more severe in the lower portions of the crown. We assumed that a branch from the middle of the crown would represent the average number of years of needle retention for the live crown.

**Foliar Disease Assessment**—All 25 sites of the Illingworth trial that we assessed for foliar compliment were also examined for the presence of foliar pathogens.

**Figure 2**—Contrasting examples of lodgepole pine foliage estimates. The first tree has 100% live crown and 4+ years of needle retention, while the tree in the photo below it has 25% live crown and only 2 years of needle retention.

**Statistical Analysis**—All statistical analyses were conducted using SAS (SAS Institute Inc., 1989). We calculated mean live crown %, and mean needle longevity (nodes) as well as the proportion of trees that had died since a previous assessment (1994) for each of 25 sites, (response traits). We considered only climatically local provenances, (MAT ± 2.0 °C, MAP ± 200 mm) so that we did not confound our results with grossly off-site provenances that would be more susceptible to foliar disease.

We used Environment Canada climate data from weather stations located nearby each of the 25 provenance trial sites. We examined correlations between response traits and changes in a suite of climatic variables between the decade of the 1990s and the 1920s using Climate BC v2.2 (Wang et al. 2006).

We also examined daily weather records (Environment Canada weather data) for climate stations throughout BC to determine the frequency of warm rain events consisting of three or more consecutive days of rain with high temperatures above 18°C. Such climatic conditions have been associated with sharp increases in foliar disease incidence and severity (Peterson 1973, Gadgil 1977, Woods et al. 2005).

**Results**

We found large differences in the longevity of foliage throughout the 25 sites we assessed with sites in the central interior of BC such as Dog Creek, generally retaining considerably more foliage than sites, such as Dave Henry, in the Robson Valley (Figure 3).

**Figure 3**—Frequency distribution of trees by representative branch needle longevity at the Dog Creek plot (below) and the Dave Henry plot (next page) of the Illingworth lodgepole pine provenance trial.
Among all trees in the 25 sites we assessed, the most frequently observed needle longevity of representative branches within the live crown was only one year (Figure 4).

There was considerable variability in mean crown ratio or percent live crown across the 25 sites (Figure 5) with the three lowest mean live crown percentages found in Region 7 (Goat R, Holmes R, and Dave Henry). Crown ratios for individual trees across the 25 sites combined ranged from less than 5% up to 100%, with 50% live crown being the most frequently encountered value (Figure 6). Over 79% of the surviving trees we examined had live crown percentages of less than 70%. Close to 27% of all trees planted on the 25 sites combined have died since the trial was installed (Figure 6).

Figure 4—Frequency distribution of trees by representative branch needle longevity at 25 sites of the Illingworth lodgepole pine provenance trial, combined.

Figure 5—Mean percent live crown per site for 25 sites of the Illingworth lodgepole pine provenance trial as assessed in 2003.
**Figure 6**—Frequency distribution of all trees by live crown percentage class for 25 sites of the Illingworth lodgepole pine provenance trial as assessed in 2003.

We found relatively strong relationships between a number of climatic variables derived by the climate interpolation model ClimateBC v2.2 (Wang et al. 2006) and all three response traits (Table 1). Increases in minimum temperatures in August between the decade of the 1920s and the 1990s was one of the most highly correlated climatic variables with needle longevity (-), live crown (-) and mortality associated with foliar disease (+). Increases in May maximum temperature was also strongly correlated with needle longevity (+), live crown (+) and mortality associated with foliar disease (-).

**Table 1**—Correlation coefficients associated with a suite of climatic variables* derived from changes between average climatic conditions in the decade of the 1920s and average conditions in the 1990s at each trial site as determined by the model Climate BC, compared to the response traits NODES (needle longevity) LC 2003 (live crown in 2003) and DOTHMORT (mortality associated with foliar diseases).

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* TMN08_S_d = Change in average min. temp. in August between 1920s and 1990s at a given site  
* TMX_sm_S_d = Change in average max temp. for July, August and Sept between 1920s-1990s.  
* TMX06_S_d = Change in mean maximum temp. in June between 1920s and 1990s.  
* PPT07_S_d = Change in mean July precipitation between the 1920s and the 1990s.
When plotted, the relationships between response traits and the strongly correlated climatic variables revealed distinct geographic groupings (Figure 7 a-d). Sites in Region 7 of the trial including Goat River, Holmes River, Dave Henry and Valemount consistently clustered together as did sites in Region 9 including Fraser Lake, Dog Creek, Ootsa Lake and Decker Lake. The two clusters were consistently at opposite extremes. The greatest increases in minimum August temperatures and July precipitation occurred in Region 7 where the proportion of trees killed by foliar disease was greatest and live crown values were the least. The reverse of these relationships occurred in Region 9.

**Figure 7 a-d**—Plotted relationships between changes in minimum August temperatures, May maximum temperature and July precipitation between average values in the 1920s compared to the 1990s using ClimateBC v2.2, with respect to Live Crown in 2003 and mortality associated with foliar disease since the previous trial assessments in 1994.

Environment of Canada daily weather records for climate stations throughout BC indicate an increasing trend in the frequency weather events consisting of three or more consecutive days of warm rain (>18C) (Figure 8). Part of this trend could be explained by the increase in climate stations collecting data throughout the province of BC. Part of this trend is, however, due to real increases in the frequency of warm rain events as clearly illustrated at long term weather stations like Ft. St. James BC (Figure 9).
Figure 8—Average frequency of weather events per decade consisting of 3 or more consecutive days of rain in which average maximum temperatures exceed 18°C for all weather stations throughout British Columbia.

Figure 9—Frequency of warm rain events consisting of 3 or more consecutive days of rain with temperature highs exceeding 18°C in Ft. St. James BC 1900-2002 (left) and 4 or more consecutive days of rain with highs above 18°C in Quesnel BC 1950-2002 (right).

Discussion

Critchfield (1957) observed a wide range in age of lodgepole pine (*Pinus contorta* var. *latifolia* Dougl. ex Loud.) needles in British Columbia of 2 to 10 years with an average of 4.3 years. The overall mean needle longevity in our results was only 1.8 years based on a sample of 19632 live trees across 25 sites. Over 8% of all trees in the 25 sites combined that were considered healthy at age 20 have died in the past 10 years, largely we believe due to foliar disease.

Johnson and Pollack (1990) found mean crown ratios ranging from 0.727 to 0.881 for planting espacements of 1.83 m to 3.66 m, respectively, in 20-year-old
lodgepole pine stands in central British Columbia. The crown ratio for many tree species including lodgepole pine is dependent on stand density (Roydhhouse et al. 1985, Johnstone and Pollack 1990). Roydhhouse et al. (1985) found crown ratios of 0.80 in unmanaged 20-year-old lodgepole pine stands in south central BC at stocking densities of 5000 stems/ha but as low as 0.51 for stands with 150,000 stems/ha. We found mean crown ratios or percent live crown as low as 34% for both the Goat River and the Holmes River sites. The highest mean percent live crown for any site we assessed was 70%. All of the Illingworth trial plots were planted at an espacement 2.4 m or approximately 1600 stems/ha. At that density the crown ratios or percent live crown should have been close to 70% at all sites (Johnson and Pollack 1990) in the absence of foliar diseases. Dothistroma needle blight was present at the majority of the 25 sites and abundant at Region 7 sites.

Weather events consisting of several consecutive days of rain above temperature thresholds of 16-20 C have been associated with sharp increases in Dothistroma needle blight incidence of infection and severity of attack (Peterson 1973, Gadgil 1977, Woods et al. 2005). We believe an explanation for TMN08_S_d being so influential for foliage retention and by association foliar diseases such as Dothistroma, is that it approximates cloud covered, higher humidity nights. Such an explanation fits with the biology of the pathogen (Peterson 1973, Gadgil 1977). If the influence of the variable TMN08_S_d was simply due to increased temperatures in August then we should have seen higher correlations with TMX08. We did not find this.

We believe the positive correlations between both years of needle retention (nodes), and live crown, with increased summer maximum temperatures (i.e. TMX06_S_d, TMX07_S_d, and TMX sm_S_d) and the negative correlation between Dothistroma caused mortality and the same summer maximum temperature variables are also consistent with pathogen biology. Live crown and needle longevity would benefit from high maximum temperatures in the summer because those conditions typically occur in conjunction with dry sunny weather. Dothistroma-caused mortality should be correlated with cooler maximum temperatures in summer and higher minimum temperatures again because of cloud cover and higher humidity.

Contrary to the claim of Wang et al (2006) that the relationships between lodgepole pine survival and climate variables are generally weak, we found strong relationships between changes in several climatic variables and foliar disease caused mortality. Wang et al (2006) used 20-year height measurements of the Illingworth trial (Illingworth 1978) to support their conclusions while our results are based on an assessment of the same trial at age 30. It seems apparent that in that 10-year period between assessments the host/pathogen relationship between lodgepole pine and foliar diseases including Dothistroma needle blight has been altered. Such changes to host pathogen relationships as a result of climate change have been predicted (Coakley et al. 1999, Dale et al. 2001, Harvell et al. 2002) and documented (Brazier 1996, Woods et al. 2005). Our results further illustrate how quickly and unpredictably climate change may affect a host species growth and survival if those climate changes favour a pest or pathogen.

Our results also illustrate how essential it is to use the most current data available when making predictions about climate change impacts on forest productivity. Over the 10-year period from the 1994 Illingworth trial assessment to our 2003 assessment, growing conditions and host/pest relationships have changed. Using 20-year volume measurements as their basis for prediction, Wang et al (2006) concluded that a climate change scenario consisting of an increase in mean annual temperature (MAT) of 2°C along with a 3.6% increase in mean annual precipitation (MAP) could result in a 25% increase in productivity. (Estimated increases are 7% for local, native forests, 25% for forests planted with seed adapted to future climates) The same authors acknowledge that their growth response functions do not include increased pest activity. That omission is key. Documented increases in pest and pathogen activity in British Columbia attributed in part to climate change (Carroll et al. 2004, Woods et al. 2005) are having a devastating impact on the forests of this province including some sites of the Illingworth trial (Figure 10). The Goat River plot was formerly considered one of the most productive sites of the 60 installations in the Illingworth trial (C.Ying pers comm.). Prior to the mountain pine beetle attack of 2003/2004, Dothistroma needle blight and possibly other foliar diseases had killed over 150 trees that had been considered “thrifty” in 1994, accounting for close to 50% of all mortality that had occurred at that site since 1974.

The severity of damage due to Dothistroma needle blight in plantations in northwest BC, Canada (Woods et al 2005) has lead to lodgepole pine no longer being considered a preferred species for forest management in the interior cedar hemlock (ICH) zone (Pajar et al.
serious consequences for lodgepole pine in BC. Although such consequences are a setback to forest management in the area, there is a wealth of other tree species to manage for in that biogeoclimatic zone. Over time, losses due to the Dothistroma epidemic in the northwest ICH will diminish as the dying lodgepole pine plantations are converted to non-susceptible species either through natural ingress or management intervention. A real concern however, is that the Dothistroma epidemic, so far concentrated in the ICH zone, could spread to the sub-boreal spruce (SBS) zone (Pojar et al. 1987) where lodgepole pine dominates the landscape. Hamann and Wang (2006) predict that the northern portion of the current SBS zone in central BC will experience a shift in climatic conditions to more closely resemble the ICH zone by 2025. If this change in climate and the associated foliar disease spread further east to the SBS zone, lodgepole pine forests could be severely impacted. Our assessment of the foliar compliment of trees in 25 sites of the Illingworth provenance trial (Illingworth 1978) suggest that the climate change predictions of Hamann and Wang (2006) may have serious consequences for lodgepole pine in BC.

References


Panel: Special Papers

Blakey Lockman, Moderator

Program____________________

Using the New Excel-based Stand Visualization Add-In Software to generate images depicting forest health issues. Lori Trummer (Presenter), Alaska Region, Forest Health Protection, Anchorage, AK; Paul Hennon, Alaska Region, Forest Health Protection, Juneau, AK; James McCarter, University of Washington, Seattle, WA; Robert McLaughney, Pacific Northwest Research Station, Seattle, WA.

A new Phytophthora causes “Mal del cipres” in the Patagonian Andes of Argentina. Alina Greslebin, Centro de Investigación y Extensión Forestal Andino Patagónico—CIEFAP - Esquel, Chubut, Argentina; and Everett Hansen (Presenter), Oregon State University, Corvallis, OR.

Growth impacts of western hemlock dwarf mistletoe on western hemlock in an old-growth Douglas-fir western hemlock forest. David Shaw (Presenter), Manuela Huso, and Howard Bruner, Oregon State University, Corvallis, OR.

Spread and Development of Phytophthora ramorum in a California Christmas Tree Farm. Gary Chastagner (Presenter), Kathy Riley, and Norm Dart, Washington State University, Puyallup, WA.

Molecular Techniques: What Can They Tell Us About Dothistroma Needle Blight? Rosie Bradshaw, Massey University, Palmerston North, New Zealand.
Using the New Excel-Based Stand Visualization Add-In Software to Generate Images Depicting Forest Health Issues

Lori Trummer, Paul Hennon, James McCarter, Robert McGaughey

Abstract—This paper describes a new user-friendly software program, the Stand Visualization Add-in for Excel, developed as an interface to the Stand Visualization System (SVS). The Add-In tool streamlines the process of creating forest images because it works directly from Excel using a standardized worksheet and simple menu commands. Also, the Add-In supports new treeform definitions to depict dwarf mistletoe infections in live trees and a range of mortality structures including uprooted, broken, and standing dead trees. Instructions are provided for downloading and utilizing features of the Add-In tool. Example images generated by the Add-In tool with data from Alaskan forests are used to illustrate the ability to communicate forest health scenarios and management treatments scenarios using SVS visualizations.

Introduction

Communicating forest health issues can be a challenge. The Stand Visualization System (SVS) is a powerful forest image software system, but is limited by its ability to easily depict dead and diseased trees. For those not well versed in the program, SVS can be cumbersome for the user to manually input plot or stand data. Also, it can be a challenge to generate images with features that require multiple tree components, such as dwarf mistletoe, broken trees, broken tops, dead tops, and root wads. The Stand Visualization Add-in for Excel was developed to support those features in Excel with simple data entry and behind the scenes post-processing.

Getting Started

The Stand Visualization Add-In software can be downloaded from:

http://silvae.cfr.washington.edu/standviz-addin/

The Add-In software provides an interface to SVS for creating stand level visualization from tree information in Excel worksheets. The Add-In will also generate bitmaps and web pages of images, and provide tools for managing SVS treeforms. Additionally, users can generate and display visualizations using EnVision. The ability to easily generate images with the Add-In software provides the user with a powerful communication tool for displaying the effects of forest pathogens, dead tree dynamics and decline syndromes at the stand level. This project was funded through a grant from the USDA Forest Service Special Technology Development Program.
To use the program, data must be entered in the proper format into an Excel worksheet. Insert the standardized worksheet header by selecting the **Stand Visualization/Tools.../Insert Worksheet Header** menu command. Next, select **Stand Visualization/Help.../Worksheet Quick Reference** and use it as a guide for the definitions and codes to be used with the worksheet header. This handy reference also indicates which columns are required and identifies the range and type of data to input.
Once data is input, a visualization can be generated with SVS from the Excel worksheet by selecting the Stand Visualization/SVS.../Generate SVS Pictures... menu command (Figure 2). Visualizations can also be generated with EnVision by selecting Stand Visualization/EnVision.../Generate EnVision Pictures... An advantage of creating images in EnVision is that it depicts more realistic trees because this program renders trees with dense crowns and secondary branches. The dense crowns, however, tend to mask some features such as dwarf mistletoe brooms. When generating images with mistletoe infected trees, SVS may be preferred over EnVision. While the EnVision software can create images at both the stand- and landscape-scale, the Add-In tool will only create images at the stand level in this program. The Add-In tool was not designed to support image creation with terrain models.

The spreadsheet and example visualization in Figure 2 demonstrates many of the features supported by the Add-In tool. It includes standing live trees, standing dying trees, broken live trees, down live trees, down dead trees, broken dead trees, and stumps. The Add-In software also supports twelve condition classes: four live tree classes (Dominant, Codominant, Intermediate, and Suppressed), three dying tree classes (1-3), and 5 dead tree classes (1-5).

A comprehensive user-friendly help file, accessed at Stand Visualization/Help.../Help Contents, is an invaluable reference for using the Add-In tool. This help file describes how to use the software, provides example visualizations, describes the format used in the worksheet, and provides additional technical information about the Add-In. It is well worth thoroughly reading before creating visualizations.

Getting the Most Out of the Add-In Features

The following “recipes” provide instructions for generating trees with root wads, bole breakage, and dwarf mistletoe. In the “recipes”, the Excel worksheet column titles are underlined with the column letter in parentheses, followed by advice for input into the column. These instructions presume that data has been inputted into all the required columns of the worksheet for each tree entry.

Root Wad:
Live/dead (F): “l” or “live” if tree crown on the ground is green; “d” or “dead” plus a dead tree condition class (1-5) if tree crown on ground is dead.

Status (G): “d” or “down”.
Condition Class (H): leave blank if tree crown on ground is green; specify a dead tree condition class (1-5) if tree crown is dead.
Bearing (N): specify 0-360 for direction tree is oriented; if left blank, a random bearing is auto-selected.
Root Wad (Q): radius of the root wad in feet; if left blank, root wads will not be drawn.

Broken Tree:
Live/dead (F): “l” or “live” if the tree crown on the ground is green; “d” or “dead” if tree crown on ground is dead.
Status (G): “b” or “broken”.
Condition Class (H): leave blank if tree crown on ground is green; specify condition class (1-5) if tree crown is dead and deteriorated.
Broken (Ht in ft) (L): specify the height of break if known; default is 2 ft.
Broken Offset (M): specify the distance the broken portion is from the tree base; if left blank the broken bole of the tree and the stump remain connected.
Bearing (N): specify 0-360 for direction tree is oriented; if left blank, a random bearing is auto-selected.

Dwarf Mistletoe:
Live/dead (F): “l” or “live” for live trees; mistletoe brooms are not currently drawn on dead trees.
Status (G): “s” or “standing”.
DMR (O): input a three digit mistletoe rating. This is a modified Hawksworth rating with a coding of 0, 1, or 2 for each crown third, rated from bottom to top. A rating of 2+1+0 is input as 210; a rating of 0+1+2 is input as 012 or 12. The default broom density is to draw 5 brooms for a crown third rating of 1 and 10 brooms for a rating of 2. Broom density per crown third and size can be adjusted through the Configuration dialog (see below for adjusting the Configuration settings).

The following tips provide instructions for troubleshooting and adjusting parameters.

The Stand Visualization Add-In default parameters can be adjusted through the Configuration dialog. With this dialog users are able to control many features including viewpoint locations for SVS and EnVision, select image capture resolutions, change dwarf mistletoe broom defaults, and add range poles to a visualization. The Configuration file can be accessed through Stand Visualization/Tools.../Configure StandViz-AddIn. Instructions for adjusting the Configuration file defaults and definitions of the codes...
used can be found by clicking the Help button from the Configuration dialog. Dwarf mistletoe broom density, size, and other defaults can be adjusted through six mistletoe boxes (DMCODE, DmskipTop, DmDen1, DmDen2, DMDia, DMHt). To add range poles to a visualization, input the desired height of the poles in feet in the Range Pole box. If left blank, range poles will not be drawn on images.

If a generated SVS or EnVision image depicts trees with red stems and crowns, the inputted species codes are not supported by the Treeform file that works behind to the scenes to generate the image. The PLANTS TreeForm File (PLANTS-SvAddin.trf) is the default file for the Stand Visualization Add-In for Excel. This file contains 224 species from across the United States. A listing of the species codes supported by the PLANTS Treeform file is located at Stand Visualization/Help…/Help Contents/Getting Results/Working with Treeform files, then click on the link to the PLANTS-SvAddin.trf. Cross-check the inputted codes with the ones listed to ensure the correct codes were used.

The Stand Visualization Add-In supports a second TreeForm file, AK-FHP-SvAddin.trf. This Treeform file is a subset of the PLANTS file and contains tree species from Alaska. A listing of the species codes supported by the AK-FHP Treeform file is located at Stand Visualization/Help…/Help Contents/Getting Results/Working with Treeform files, then click on the link to the AK-FHP-SvAddin.trf. This file, customized for Alaska staff, contains adjustments to Alaskan trees including 5 classes of yellow-cedar snags, 5 white spruce deterioration classes, and numerous live and dead tree changes to other conifer and hardwood tree forms.

To create images with the enhanced AK-FHP Treeform file, users must designate this file. To change from the default PLANTS Treeform file to the AK-FHP Treeform file, click on the Configuration worksheet tab located next to the worksheet tabs of the Excel workbook. In column (B), labeled TreeformFile, of the Configuration worksheet, specify either the PLANTS-SvAddin.trf or the AK-FHP-SvAddin.trf. The Configuration worksheet appears after one SVS or EnVision image has been generated in an Excel workbook.

Communicating with SVS Images_____________________

The Add-In tool can generate SVS pictures to either bitmaps or web pages, at high or low resolution. The images can be used for a variety of purposes including publications, poster presentations, and web pages.

Stand and plot data from Alaskan forests were used to test and adjust the Add-In tool. During beta testing, we made substantial changes to the Alaska Treeform file (AK-FHP-SvAddin.trf) to more realistically depict live trees, snags, and downed trees for nearly all the conifer and hardwood tree forms. As a result, we were able to create images that relay simple forest health and management scenarios through depiction of yellow-cedar decline, deterioration of bark beetle killed trees, tree mortality patterns in old-growth forests before and after selection harvest, and hemlock dwarf mistletoe spread and intensification.

The following images developed from Alaska forest datasets are examples of communicating simple forest health and management scenarios. The images are displayed here in grey scales, but typically appear in colors in SVS and Envision. Color allows for distinguishing tree species and many other subtle details.
Figure 3—SVS and Envision now have the capability of depicting dwarf mistletoe infections in each crown third to represent a range of DMR values. This image depicts a plot in a western-hemlock dominated forest in Alaska that regenerated following a storm in the 1880s. Hemlock dwarf mistletoe spread to the newly regenerating trees, now over 100 years old, from several residual trees that survived the original storm event. Dwarf mistletoe infections, depicted as gray brooms, are much more distinguishable in the typical color images produced by SVS than in this grayscale image. The dense crowns drawn by Envision tend to mask dwarf mistletoe infections, even when color is used.

Figure 4—SVS display of yellow-cedar decline in Alaska using plot data in Excel to illustrate the concentration and size of snags, and succession to the smaller, surviving western and mountain hemlock trees.
Figure 5—Patterns of tree death in an old-growth hemlock-dominated forest before and after selection harvest. Top: before harvest with natural tree death represented by uprooted trees, trees with broken boles, and snags. Middle: condition directly following selection harvest with stumps and some trees damaged by logging. Bottom: subsequent windthrow (uprooting) of three of the 5 larger residual trees.
A New *Phytophthora* Causes “Mal del ciprés” in the Patagonian Andes of Argentina

Everett Hansen and Alina Greslebin

**Abstract**—A survey of *Phytophthora* spp. in *Austrocedrus chilensis* (Ciprés de la cordillera) forest was conducted in order to determine their possible involvement in the “mal del ciprés” decline. Five *Phytophthora* species were recovered from streams and soils in declining stands. *P. syringae* was the most common species isolated from soil and/or streams; *P. gonapodyides* was isolated from streams only, and *P. cambivora*, *P. chlamydo* and *P. taxon raspberry* were isolated rarely. None were recovered from declining trees and none were pathogenic to *Austrocedrus*. *A. chilensis* is a homothallic species characterized by semipapillate sporangia, oogonia with amphigynous antheridia, and very slow growth. ITS DNA analysis indicates that its closest relative is *antheridia*, and very slow growth. ITS DNA analysis

*Phytophthora austrocedrae*, a new species, was isolated from necrotic lesions of stem and roots of *Austrocedrus chilensis*. It is a homothallic species characterized by semipapillate sporangia, oogonia with amphigynous antheridia, and very slow growth. ITS DNA analysis indicates that its closest relative is *Phytophthora syringae*, another species frequently isolated from soil and streams in *A. chilensis* forests. *P. austrocedrae* is pathogenic to *Austrocedrus*, and is the cause of Mal del Ciprés. This paper summarizes a series of investigations already published or in press.

**Introduction**

Patagonia lies nearly as far south of the equator as we are north. It is mostly dry shrub steppe to the Atlantic coast, but the western edge, at the base of the Andes, is more interesting for foresters and forest pathologists. This is a land of ice and snow and lakes and more interesting for foresters and forest pathologists. It forms pure stands as well as mixed stands with *Nothofagus* spp. and, among the few conifers inhabiting southern Argentina, it has the largest distribution, covering ca. 160,000 ha. *A. chilensis* is economically valuable because of the high quality of its wood and it also has a great tourist and scenic relevance. The species is protected as a native tree, and harvest is regulated by the State. Throughout its distribution area, *A. chilensis* suffers mortality termed “mal del ciprés” or “cypress wither” (Filip & Rosso, 1999), caused by unknown factors.

**“Mal de ciprés”**

The “mal del ciprés” decline was noted about 50 years ago, associated with a forest tree nursery testing exotic trees for growth under Argentine conditions in Isla Victoria, Neuquén Province (Varsavsky et al., 1975). Interest and concern have increased due as the affected area expanded. Mortality is now found throughout the range of the tree. It is impossible to carry out appropriate silvicultural management of affected stands since the appearance and evolution of the disease cannot be predicted. As native forest dies, it is often replaced with exotic introduced conifers, or converted to grazing land.

Different studies have been carried out to determine the etiology of this disease. Rosso et al. (1994) showed that the mortality is scattered in patches, suggesting a contagious process caused by a pathogen in the roots. Barroetaveña & Rajchenberg (1996) studied the wood rots associated with the mal del ciprés but concluded that the decay fungi were secondary. Cali (1996) made dendrochronological studies that showed: a) the relationship between radial growth decrease and disease progression; and b) that the phenomenon is sometimes initiated many years before external symptoms can be detected.

Declining stands are usually associated with poorly drained sites where low slopes, high moisture at the end of the dry season, and impervious layers close to

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the soil surface might act as predisposing factors (La Manna & Rajchenberg, 2004).

The evidence gathered so far indicates that the disease has its origin in the root system, where the death of the tissue comes prior to the defoliation of the crown (Hennon & Rajchenberg, 2000). A decay of the roots is observed, followed in some cases by the development of brown rots in the sapwood. The origin of the disease in the roots, in addition to its association with poorly drained sites suggests a possible role for Phytophthora species in the disease (Filip & Rosso 1999, Hansen 2000, Hennon & Rajchenberg 2000, La Manna 2004, Rajchenberg et al. 1998). For a detailed description of the disease and background see Greslebin et al. (2005).

Recent work at CIEFAP has focused on the possible role of Phytophthora species in mal del ciprés, and has resulted in the description of a new species, _P. austrocedrae_, and the conclusion that it is the causal agent. This is the culmination of a series of visits and exchanges facilitated by the USDA Forest Service and CIEFAP.

In order to investigate the possible involvement of _Phytophthora_ species in the origin and development of the “mal del ciprés” a survey of _Phytophthora_ inhabitants of soil and streams of _Austrocedrus chilensis_ forests and symptomatic _Austrocedrus_ trees themselves, was conducted. Phytophthora species in soil and streams associated with mal del ciprés revealed the presence of some species common in forests all over the world (_P. gonapodyides_ and undescribed taxon Pgchlamydo), and some unexpected species, rarely reported in forests (_P. syringae_) or rarely recorded before. Five _Phytophthora_ species were detected in declining _A. chilensis_ forests in all, but none of them showed a clear relationship with the decline (Greslebin et al. 2005). These species did not cause disease on _Austrocedrus_ seedlings in inoculation tests.

Initial isolations from tree boles yielded no pathogens, but ELISA and DNA extraction from the necrotic tissues and PCR with _Phytophthora_-specific primers showed the presence of a _Phytophthora_ species (Sutton, unpublished data). Recent work focused on necrotic inner bark lesions at the root collar and lower stem. This symptom was rarely reported in previous studies, but was frequently found on affected trees that appeared to be in active decline. These lesions originated in the roots and looked very similar to those caused by other pathogenic _Phytophthora_ species, such as _P. lateralis_ on _Chamaecyparis lawsoniana_ in the western United States (Hansen 2000). Still, initial isolation attempts from the _Austrocedrus_ lesions were negative. After incubation at cooler temperatures than previously used, a new _Phytophthora_ species was finally isolated from the lesions.

This new species, now named _P. austrocedrae_, is characterized by the combination of a very slow growth rate and low (17.5°C) optimal temperature, explaining the difficulty of isolation. It is unique morphologically, with semipapillate, non-caducous and non-proliferating sporangia, and is homothallic, forming oogonia with amphigynous antheridia. A BLAST search of the GenBank ITS DNA sequence database identified _P. syringae_ as the closest relative. _P. austrocedrae_ is in clade 8 of the Cooke et al. (2000) molecular phylogeny of the genus.

Pathogenicity tests continue, but initial inoculations of mature forest trees yielded large and expanding phloem lesions, similar to those observed on naturally infected trees.

**Discussion**

_Phyltophthora austrocedrae_ is associated with the mortality of _Austrocedrus chilensis_ known as “mal del ciprés”. Evidence gathered to date suggests that it is the primary cause of the disease. _P. austrocedrae_ was detected in the advancing zone of necrotic lesions in the inner bark of roots, root collar and stem of symptomatic trees in all surveyed areas affected by “mal del ciprés” (Figure 1), and it is pathogenic when inoculated in stems and boles of mature trees.

Other agents also kill _Austrocedrus_, and an important task is to distinguish damage caused by insects, drought, fire, and unknown agents from mal del ciprés, caused by _P. austrocedrae_. The symptomology of “mal del ciprés” is unique. Disease originates in the roots and death of roots and collar tissues precede crown defoliation. Strips of necrotic inner bark with well-defined margins, extending upward from dead roots are characteristic, and affected trees are often marked by resin flow on the external bark. At least in some situations, trees die more rapidly than previously assumed, and crown chlorosis is a good early symptom. Trees of all ages can be affected. At two sites where _Phytophthora austrocedrae_ was not detected in dying _A. chilensis_, symptomology was different (i.e. dead or dying crowns with healthy tissues at root collar).

When viewed in this light, Mal del Ciprés has many characteristics of other exotic, invasive Phytophthora
diseases such as Port-Orford-cedar root disease and Jarrah dieback. Observations in Argentina and inference from related diseases suggest that this pathogen is spread primarily through human activities, especially on vehicles associated with salvage logging of diseased stands, and on the feet of cattle grazing in Austrocedrus woodlands. Historically, planting infested nursery stock was probably important. Much work remains to be done, including population genetic analyses to test the likelihood the pathogen is of recent, exotic origin. We know nothing yet about the behavior of the pathogen in the soil and during initial root infection, and have only guesses about its spread across the landscape. Given the slow and fastidious growth of *P. austrocedrae* in culture, the research will be challenging.

Acknowledgements

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References


**Figure 1**—Areas where *Phytophthora austrocedrae* was detected to date.
Growth Impacts of Western Hemlock Dwarf Mistletoe on Western Hemlock in an Old-Growth Douglas-fir Western Hemlock Forest

David Shaw, Manuela Huso, and Howard Bruner

Introduction

We have been working in an old-growth Douglas-fir/western hemlock forest in the south Cascades of Washington state where hemlock dwarf mistletoe occurs in spatially discrete infection centers (Shaw et al. 2005, Swanson et al. 2006). Infected trees averaged larger than uninfected trees while within the infected trees, heavily infected trees averaged larger than moderately and lightly infected trees. Another study into the effect of dwarf mistletoe on hydraulic architecture, water, and carbon relations of western hemlock showed reduced water use at the whole tree scale, reduced leaf level nitrogen, water use efficiency, and photosynthetic capacity, resulting in estimated reduction of carbon accumulation of 60% at the whole tree scale (Meinzer et al. 2004). However, this level of growth impacts seems contradictory to the observation that the most heavily infected trees are the largest western hemlock trees in the stand.

Interest in the impacts of dwarf mistletoes in natural stands has increased with renewed emphasis on ecosystem management and green tree retention forestry because the emphasis has changed from eradicating dwarf mistletoes to managing dwarf mistletoes. Research into dwarf mistletoes in old-growth forests in particular is generally lacking, and with an increasing emphasis on management of old-growth forests, information on effects of dwarf mistletoe is becoming more important (Hawksworth and Weins 1996).

The objective of this study was to investigate whether reduction in growth of western hemlock trees associated with infection by western hemlock dwarf mistletoe occurs in an old-growth forest which has a unique structure when compared to young stands. We plan to submit this information for a journal article, so our intention here is to inform WIFDWC members about this study and include a figure that will not be included in the journal submission.

Methods

The study site is located in the T.T. Munger Research Natural Area, Wind River Experimental Forest, Gifford Pinchot National Forest, Washington State, an approximately 500 year-old, 478-ha, Douglas-fir/western hemlock forest established in 1934 to exemplify old-growth forests in a natural condition. In 1947 a series of 9 mortality transects were established that consisted of 105, 0.4 ha (1 acre) adjacent plots where all trees ≥ 45.7 cm (18 inches) diameter at breast height (dbh) were surveyed for mortality every 5-7 years (Franklin and DeBell 1988). In 1991 the borders of these mortality strips were hardened, and all trees ≥ 45.7 cm dbh were tagged and measured for dbh. The plots were resurveyed in 1997 and 2004 for diameter growth. We estimated the dwarf mistletoe rating (DMR) for 1,661 western hemlock trees > 45.7cm dbh in 97 of the original 0.4 ha (1 acre) plots in 1997 and the dbh growth record from 1991 to 2004 was used to determine growth rate (13 years).

The original sampling design which includes adjacent 0.4 ha plots in a series of transects can lead to high spatial correlation. The qualities of the soil, available light, aspect, and stand structure all have potential influence on how much a tree will grow. These qualities tend to be similar (have little variation) between places that are physically close to one another and have more variation as the places become physically more separated. Since these qualities affect tree growth, the result that we see is spatially autocorrelated growth responses. We took advantage of this spatial autocorrelation by blocking on the 0.4 ha plot. This blocking acknowledges correlation of growth among the trees within a block but assumes independence of growth of trees from two different blocks, whether contiguous or not. Within each block were trees with DMR ratings ranging from 0 to 6, with 0 indicating no western hemlock dwarf mistletoe observed and 6 indicating that western hemlock dwarf...
mistletoe occurred throughout the tree’s canopy. For this analysis DMR ratings were further grouped into 4 categories, uninfected (DMR 0), low severity (DMR 1&2), moderate severity (DMR 3&4), and high severity (DMR 5&6). Only those plots for which there was at least 1 tree in each of the 4 DMR categories was used as a block. There were 27 plots that met this criterion. In these plots there was a minimum of one tree in each DMR category, and there were sometimes as many as 20, but most often two to four. Each tree within a block was regarded as a sampling unit of the DMR treatment within the block.

Two response variables were analyzed, basal area growth and diameter growth between 1991 and 2004. Initial DBH of the trees in 1991 was found to not be related to DMR category and was included as a covariate in both analyses. Each response variable and the covariate were averaged across trees within each DMR category in each block resulting in a balanced design with 4 treatments (DMR categories) and 27 replicates (blocks or 0.4 ha plots). These data were analyzed using Analysis of Covariance of a randomized block design with PROC MIXED in the SAS/STAT software, Version 9.1 of the SAS System for Windows (SAS Inst. Inc, 2002). Residuals analysis indicated that assumptions of normality and homogeneous variance among treatments were met for both responses and no transformations of the data were made.

Results

Percent difference from growth of uninfected trees for DBH growth (Figure 1) was Low (DMR 1, 2); +5%, Moderate (DMR 3, 4); +11%, and Heavy (DMR 5, 6); -27% while for basal area growth it was Low (DMR 1, 2); +5%, Moderate (DMR 3, 4); +7%, and Heavy (DMR 5, 6) -27%. Only the Heavy DMR infection category was significantly different from the uninfected category.

Figure 1—Mean diameter growth (and 95% confidence limits) of trees in the four DMR categories.
Conclusion

This study of larger diameter western hemlock trees in an old-growth forest supports most other studies that have investigated growth effects of dwarf mistletoes; growth effects do not occur until trees are heavily infected, usually DMR 5 and 6.

Literature Cited


Since 2005, the spread and development of *Phytophthora ramorum* has been monitored in a 23-acre U-cut Christmas tree farm near Los Gatos, CA. Conifers being grown at this site include Douglas-fir (*Pseudotsuga menziesii*), grand fir (*Abies grandis*), giant sequoia (*Sequoiadendron giganteum*), Scots pine (*Pinus sylvestris*), white fir (*A. concolor*), and California red fir (*A. magnifica*). Some known *P. ramorum* hosts in the infected forest adjacent to the edge of the Christmas tree farm include: California bay laurel (*Umbellularia californica*), madrone (*Arbutus menziesii*), big leaf maple (*Acer macrophyllum*), false Solomon seal (*Maianthemum racemosum*), toyon (*Heteromeles arbutifolia*), coast redwood (*Sequoia sempervirens*), and tanoak (*Lithocarpus densiflorus*). After mapping the perimeter of the farm to identify areas where Ramorum blight was evident, 500 trees in the largest area with a past history of infection were mapped, tagged and measured for height. A series of transects were established from the edge of the forest into the Christmas trees in this area to monitor the spread of *P. ramorum* from infected plants (predominantly bay) along the edge of the forest into the Christmas tree plantation. The length of these transects ranged from 17 to 27 meters. Container-grown Douglas-fir and grand fir seedlings that had just broken bud and small rhododendron plants were also placed along three transects at approximately 0, 3.5, 8, and 13 meters from the forest edge. Periodically during the spring and summer, the level of infection and extent of shoot dieback has been assessed on the tagged trees and containerized seedlings. In both 2005 and 2006, new shoot infections on the Christmas trees only developed in the spring and initial dieback symptoms were limited to newly expanded shoot tips. Environmental conditions during spring 2005 were much more favorable to initial shoot tip infections than in 2006. In particular, along the transects where grand fir were underneath the canopy of infected bay, virtually all of the new shoots were infected shortly after bud break in 2005. The progression of dieback on infected shoots of Douglas-fir and grand fir in 2005 progressed for about 4 weeks after the initial appearance of symptoms, typically spreading about two inches into the previous year’s growth. The extent of dieback did not increase between early summer and mid-November. Infection rates and disease severity were also much higher among the seedlings that were placed along the interface of the forest and Christmas tree farm in 2005. On May 19, 2005, 81.7 and 94.3% of the Douglas-fir and grand fir seedlings, respectively, that had been exposed since April 21 had been infected. The percentage of each seedling that was killed as the result of shoot dieback averaged 52.8% for the Douglas-fir and 81.2% for the grand fir. In 2006, infection of conifer seedlings and rhododendrons only occurred during exposure periods when precipitation occurred. The infection of Douglas-fir and grand fir trees and seedlings along the transects indicate that distance from infected plants (predominantly bay) within the forest is an important factor relating to the infection of the Christmas trees. Most of the infected Christmas trees and seedlings occurred within 2 to 4.4 meters of the edge of the forest. Virtually no infection was evident on Christmas trees that were 5 to 8 meters away from the forest edge.
Molecular Techniques: What can they tell us about Dothistroma needle blight?

Rosie Bradshaw

Abstract—Molecular methods can be used as tools to improve our understanding of the Dothistroma needle blight pathogen and the mycotoxin dothistromin. DNA sequence data formed the basis of a species revision that split the pathogen population into two species: Dothistroma septosporum and D. pini. In New Zealand, DNA profiling of D. septosporum showed very limited genetic diversity. These data support the hypothesis that a single clone of the pathogen was introduced and spread around New Zealand by asexual reproduction. Studies of worldwide genetic diversity and mating types are in progress and are expected to shed light on the origins and distribution of these pathogens. Dothistromin toxin is thought to be responsible for the red band coloration characteristic of Dothistroma needle blight. It is closely related to the aflatoxins and the genetics and biochemistry of their biosynthesis appear to be similar. However molecular studies have shown that dothistromin is unusual in being produced at a very early stage of growth, suggesting a role in the early stages of needle infection.

Introduction_________________

Several presentations at the WIFDWC meeting profiled Dothistroma needle blight. In the superb field trips around the Smithers region we saw the current epidemic in British Columbia. There are many aspects of this disease to study and a diversity of tools and methods to use. In this presentation I will give a brief overview of the application of molecular tools to Dothistroma research. Examples from current projects will be discussed along with previously published work. The first section profiles some research that aims to improve our understanding of the pathogen, whilst the second section focuses on developments in knowledge of the dothistromin toxin and its role in the disease process.

The Dothistroma Pathogen_____

What have we got?

The taxonomy of the Dothistroma needle blight pathogen, like many other fungi, has had a tortuous history with many name changes (reviewed in Bradshaw 2004). Comparisons of DNA sequences between fungi are rapidly gaining acceptance as important taxonomic tools and it was on this basis that Barnes and others (2004) recently split the Dothistroma pathogen population into two species. The species showed 9.7% divergence over 1.5 kb of nucleotide sequence taken from sections of three different regions of the genome. The newly re-named Dothistroma septosporum has a global distribution and is the species with which most studies have been carried out to date. The newly classified Dothistroma pini is currently believed to have a more limited distribution, found predominantly in North-Central U.S.A. although Mike and Brenda Wingfield’s group in South Africa are currently investigating other possible sightings of this species.

In New Zealand, the diversity of the Dothistroma pathogen is very low. DNA profiles of herbarium isolates isolated in the 1960s were compared with those of isolates collected more recently from around New Zealand (Hirst and others 1999). All of the profiles were identical, suggesting that only one clone of the pathogen has been present in the country since its proposed introduction prior to its discovery in 1964. This work also implied that there was only one incursion of the pathogen and that no subsequent incursions occurred, or became established. Furthermore this provided an explanation why the sexual stage has never been seen in New Zealand. The fungus is likely to be heterothallic, requiring two mating types, yet only one mating type would be present in a clonal population. The presence of a single mating type in New Zealand has recently been confirmed using molecular methods (unpublished results).

Where is it from?

There are various speculations in the literature about the origin of Dothistroma needle blight. For example the pathogens are thought to be endemic to Central America and Nepal (Ivory 1994). The availability of

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DNA sequence data (Barnes and others 2004) and DNA profiling methods (Hirst and others 1999; Ganley and Bradshaw 2001) for Dothistroma pathogens mean that it is now possible to delve more deeply and make predictions about their origins and global movements. The Wingfield lab is leading the way in this work. The same principles that supported the “Out of Africa” hypothesis for the origin of humans (i.e. that a high DNA sequence diversity is seen in populations in the centre of origin), can be applied to Dothistroma. It is already apparent that there are regions of high and low genetic diversity for *D. septosporum*, such as the low genetic diversity seen in New Zealand. It will be interesting to see if isolates genetically identical to the New Zealand isolates are present in other countries, which may implicate those countries as a possible source of the incursion into New Zealand that occurred some decades ago.

**How is it changing?**

DNA sequencing and profiling methods can be used to determine if a pathogen is changing over time and, in some cases, can indicate the nature of the change. It is now possible to recover DNA from dried herbarium specimens (Bearchell and others 2005), providing an opportunity to compare archived specimens with current isolates. As well as comparing general patterns of genetic change it is possible to focus on specific genes, such as toxin biosynthetic genes (see next section) that may affect the virulence of the isolates. These studies, currently in progress, have the potential to define whether genetic changes have contributed to the current epidemic in BC.

In order to understand the potential for further change in a pathogen it is important to understand its reproductive cycle. The limited genetic variability and single mating type in New Zealand suggests an exclusively asexual mode of reproduction and limited capacity for genetic change. In contrast *D. septosporum* isolates in Canada appear hypervariable and two mating types are present (Dale and others, this proceedings), suggesting that sexual recombination is frequent and therefore providing an opportunity for rapid genetic change.

**The Dothistromin Toxin**

**How is it made?**

Dothistromin toxin is a red-brown coloured polyketide compound with broad toxicity to many types of cells (Stoessl and others 1990). It is remarkably similar in structure to versicolorin B, a precursor of the potent mycotoxin, aflatoxin. On the basis of this similarity we identified genes required for dothistromin biosynthesis in *D. septosporum* (reviewed by Bradshaw and Zhang 2006). Genetic and biochemical studies suggest that dothistromin is made in a very similar way to aflatoxin. There are many genes with matching functions (orthologs) and common biochemical intermediates that accumulate when pathways are blocked at specific places (Bradshaw and others 2002). From an evolutionary point of view it is interesting to note that whilst the aflatoxin biosynthetic genes are neatly arranged together in a cluster the dothistromin genes are in a fragmented and dispersed arrangement (unpublished results).

**When is it made?**

Dothistromin is made in culture as well as *in planta*. The red-band lesions often seen in Dothistroma needle blight are thought to be due to accumulation of dothistromin and indeed dothistromin has been isolated from these (Bassett and others 1970). However dothistromin is known to be degraded quickly in planta (Franich and others 1986) so at least some of the coloration seen may be due to a plant defence response. We have studied the regulation of dothistromin biosynthesis in culture and found that dothistromin is produced mainly in the early stages of exponential growth. This is in contrast to most secondary metabolites, such as aflatoxin, that are produced mainly in late exponential or stationary phase (Skory and others 1993). Molecular tools verified the early onset of dothistromin biosynthesis by showing that dothistromin genes are also switched on at an early stage of growth. We are now determining whether early dothistromin biosynthesis is also seen *in planta*. Since dothistromin is generally regarded to have a role in needle blight disease it is tempting to speculate that this unusual pattern of regulation is an adaptation for this role.

**What is its role in disease?**

Is dothistromin required for the development of Dothistroma needle blight symptoms? Currently we cannot answer this question. However we have made dothistromin-deficient mutants by targeted replacement of selected dothistromin biosynthetic genes (Bradshaw and others 2002; Bradshaw and others 2006) and the pathogenicity of these mutants is currently under investigation. It is interesting that the red-band symptoms of Dothistroma needle blight commonly seen in southern hemisphere plantations are less often seen in the Canadian epidemic, although
many Canadian isolates that produce high levels of the toxin in culture have been isolated from the needles (Dale and others, this proceedings).

An early onset of dothistromin production suggests the toxin may be required for invasion of the pine needle, perhaps by directly killing plant cells or by triggering a defence response that causes tissue damage. The pathogen has a necrotrophic stage and completes its life-cycle on dead pine tissue (Gadgil 1967). Alternatively dothistromin may have a role in competition against other micro-organisms on the needle surface. We are currently testing these hypotheses. Molecular tools are useful here too: by joining the regulatory regions of dothistromin genes to a green fluorescent protein (gfp) reporter gene we can visualize when and where toxin biosynthesis is taking place.

Conclusion and Future Prospects

Following the WIFDWC meeting a special meeting of Dothistroma researchers was held. It is clear that there is still much we need to find out about the pathogen and the toxin and that molecular tools will have a part to play in assembling some of this knowledge, as outlined in this paper. However there are also many other aspects of the pathogen, the host, the environment, and interactions between each of these, that need to be studied. For example a deeper understanding of the epidemiology and phenology of Dothistroma needle blight is required. Molecular tools will augument traditional methods to enhance our overall understanding of this devastating disease and to develop new approaches to reduce its impact throughout the world.

References


Dothistroma Needle Blight in the Czech Republic. Miroslava Bednarova, Dagmar Palovcikova, Irena Bodejckova, Libor Jankovsky, Michal Tomsovsny, Department of Forest Protection and Game Management, Faculty of Forestry and Wood Technology, Brno, Czech Republic.

Population Structure of Armillaria spp. in a Temperate Old-Growth Rainforest, Olympic Peninsula, Washington. Nicholas J. Brazee and Robert L. Edmonds, College of Forest Resources at the University of Washington, Seattle, WA.


International Tree Failure Database (ITFD): A program to improve the ability to identify and manage hazardous trees. Judy Adams, Forest Health Technology Enterprise Team, USDA Forest Service, Fort Collins, CO.

Drainage Patterns and Other Site Factors: Impacts on the Movement of Magnesium Chloride Based Dust Suppression Products from Gravel Roads. Betsy Goodrich and William Jacobi Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO.

Estimated Economic Losses Associated with the Destruction of Plants due to Phytophthora ramorum Quarantine Efforts in Washington State. Norman L. Dart and Gary A. Chastagner, Washington State University Research and Extension Station, Puyallup, WA.

The Effects of Thinning and Borax Application on Heterobasidion annosum Infected Stands on the Olympia Peninsula. Daniel Omdal, Amy Ramsey and Melanie Kallas Ricklefs, Washington Department of Natural Resources, Olympia, WA.

Where Has all the Root Rot Gone? Ground Truthing Bear and Root Disease Damage in Western Washington. Daniel Omdal, Jeff Moore, Melanie Kallas Ricklefs, and Amy Ramsey, Washington Department of Natural Resources, Olympia, WA.

Phytophthora ramorum Survey and Monitoring in Western Washington. Daniel Omdal, Amy Ramsey, and Melanie Kallas Ricklefs, Washington Department of Natural Resources, Olympia, WA.


Screening for Genetic Resistance in Western Hemlock (Tsuga heterophylla) Provenances for Management of Dwarf Mistletoe (Arceuthobium tsugense). Charlie Cartwright, Cowichan Lake Research Station, BC Ministry of Forests, Mesachie Lake, BC. Simon Shamoun, Pacific Forestry Centre, Canadian Forest Service, Victoria, BC. Sue Askew and Lea Rietman, Forest Science Department, Faculty of Forestry, University of British Columbia, Vancouver, BC.

Pruning to Manage White Pine Blister Rust in the Central Rocky Mountains. Amanda Crump and William R. Jacobi, Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO. Kelly S. Burns and Brian Howell, Forest Health Management, USDA Forest Service, Rocky Mountain Region, Lakewood Service Center, Lakewood, CO.
Dothistroma Needle Blight in the Czech Republic
Miroslava Bednarova, Dagmar Palovcikova, Irena Bodejckova, Libor Jankovsky, Michal Tomsovy

Abstract—Dothistroma needle blight was first observed in the Czech Republic in 1999 on an imported Pinus nigra Arnold. In 2000, it was again found on open-grown trees. During the last few years, it has become an important pathogen of pines in the Czech Republic where it has been detected in more than 60 localities, especially in Moravia and Silesia and eastern Bohemia. It has been found on 18 hosts including 5 species of spruce. Austrian pine (Pinus nigra Arnold) is mostly attacked; disease was also found on native species – Pinus sylvestris L. DNA sequencing confirmed that Dothistroma needle blight is caused by Dothistroma septospora (Dorog.) Morelet. Growth rate studies confirmed that optimal temperature for mycelial growth is between 15 – 20 °C.

Acknowledgements—This project was made with support of grants GACR 526/03/H036 and MSMT 6215648902.
Population Structure of *Armillaria* spp. in a Temperate Old-Growth Rainforest, Olympic Peninsula, Washington

Nicholas J. Brazee and Robert L. Edmonds

**Abstract**—*Armillaria* spp. are common forest pathogens worldwide causing root disease and mortality, particularly in managed forests. However, very little information exists about *Armillaria* in undisturbed, old-growth forests. The primary goal of this study was to determine the *Armillaria* species present in the old-growth temperate rain forest in the Hoh River Valley. Specific study objectives were to determine: (1) which *Armillaria* species were present, (2) if they were the same inside and outside of canopy gaps and in the Sitka spruce, western hemlock, and Pacific silver fir forest zones, and (3) genotypic diversity and size of individual genets of *Armillaria*. The 520 ha study area was located in Olympic National Park centering on the West Twin Creek watershed located 48 km southeast of Forks, WA. A ground-based survey using three trail transects was used to find ten canopy gaps. *Armillaria* was very abundant and appears to contribute in making trees susceptible to windthrow during storms. Only two *Armillaria* species were present, *A. sinapina* and *A. nabsnona*; 91% of the isolates were *A. sinapina*. Eight of the ten gaps had *A. nabsnona*, however. No isolates of *A. ostoyae*, considered to be the most pathogenic species in western coniferous forests, were found. Populations of *A. sinapina* and *A. nabsnona* were highly variable and genets were generally small indicating that spore dispersal was an important means of establishment. However, isolates of two genotypes were separated by at least 1600 m indicating that large *Armillaria* genets are not restricted to dry environments. Western hemlock was the dominant tree species as well as the dominant host for *Armillaria* totaling 87 of the 100 trees sampled.
A Proteomics Approach to Investigate Proteins Differentially Expressed by Douglas-fir Seedlings Infected by the Root Rot Fungus *Phellinus sulphurascens*

M.A. Islam, R.N. Sturrock, and A.K.M. Ekramoddoullah

**Abstract**—We carried out a comparative proteomic study to help us explore molecular mechanisms underlying the susceptibility of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) to Laminated Root Rot (LRR), a disease caused by *Phellinus sulphurascens* Pilat. Two dimensional gel electrophoresis was conducted on proteins extracted from roots of laboratory grown, young Douglas-fir (DF) seedlings inoculated with *P. sulphurascens* and from control, uninoculated DF seedlings. A total of 1303 spots were detected from 7 days post inoculated (dpi) and control root samples. Among these spots, a total of 277 were found to be differentially expressed. Out of these differentially expressed spots, 74 upregulated or induced and 85 downregulated spots were statistically significant (0.05%). To identify proteins, a total 70 selected spots were excised and analyzed by liquid chromatography-tandem mass spectrometry (LC-MS/MS) analysis. Results indicate that the major proteins differentially expressed in *P. sulphurascens*-infected DF seedlings include those in the following functional groups: disease/defence (16%), metabolism (11%), secondary metabolism (11%), signal transduction (10%), transcription factors (10%) and energy (9%). A number of additional proteins involved cell structure (7%), protein synthesis (6%) and protein destination and storage (3%) were also identified. Our results demonstrate the feasibility of using a proteomics approach and laboratory grown seedlings to elucidate molecular interactions of the DF-LRR pathosystem.


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R.N. Sturrock and A.K.M. Ekramoddoullah are Research Scientists in the Canadian Forest Service, Natural Resources Canada, Pacific Forestry Centre, 506 West Burnside Road, Victoria, BC, Canada V8Z 1M5.
International Tree Failure Database (ITFD): A program to improve the ability to identify and manage hazardous trees

Judy Adams

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.


Judy Adams is a Modeling Program Manager, Forest Health Technology Enterprise Team, USDA Forest Service, 2150A Centre Avenue, Fort Collins, CO, 80526.
Drainage Patterns and Other Site Factors: Impacts on the Movement of Magnesium Chloride Based Dust Suppression Products from Gravel Roads

Betsy A. Goodrich and Dr. William R. Jacobi

Abstract—Magnesium chloride (MgCl₂) products are applied to gravel roads during summer months for dust suppression and road stabilization. Site factors such as slope, topography, drainage patterns, elevation and precipitation may influence the potential runoff from road edges, influencing spatial distribution and amount of salt in roadside soils and vegetation. Drainage vegetation health plots were established to follow potential movement of water and ions from gravel roads into roadside ecosystems. Plots were variable in length dependent upon the presence or absence of tree crown damage (similar to known symptoms of salinity damage) in the drainage area. Analysis to determine specific causal factors of crown damage is underway. We hypothesized that a higher potential for surface water flow into drainages (longer culvert ditch lengths and steeper slopes) would result in further movement of water and salt ions from roads into the roadside systems. Analysis to determine whether potential surface water flow values correlate with amount and spatial distribution of salt ions in soils and plant tissue is underway. In drainage plots along treated roads, soils ranged in chloride amount and distribution but overall had high amounts of chloride. Mean soil chloride values in roadsides along treated roads were significantly higher than untreated roadside areas and may be contributing to tree damage seen in drainage plots along roads treated with dust suppression products.
Estimated Economic Losses Associated with the Destruction of Plants Due to Phytophthora ramorum Quarantine Efforts in Washington State

Norman L. Dart and Gary A. Chastagner

The quarantined plant pathogen Phytophthora ramorum was first detected in a Washington state nursery in the summer of 2003 during a trace forward survey conducted by the Washington State Department of Agriculture (WSDA). Infected plants were detected at a total of two nurseries in 2003 and WSDA nursery inspections detected P. ramorum in twenty-five nurseries in Washington during 2004 (4). During late 2004, the United States Department of Agriculture Animal Plant Health Inspection Service (USDA-APHIS) issued an Emergency Federal Order requiring all nurseries that sell host plant materials in Washington, Oregon and California that ship plants interstate enter a compliance agreement and be inspected for P. ramorum starting January 10, 2005 (2). In 2005 sixteen nurseries tested positive during certification, recertification or post eradication surveys (4). Nine of these were repeat nurseries from 2004.

When P. ramorum is detected in a Washington nursery, WSDA implements the USDA-APHIS mandated Confirmed Nursery Protocol for P. ramorum (1). As part of this protocol an Emergency Action Notification Form (EAN) is issued to all positive nurseries, notifying the nursery management of the required action(s), and the number and species of plants that are subject to the action(s). Depending on the specific circumstances, an EAN will require that plants are destroyed or held for 90 days for additional monitoring.

The economic impact that P. ramorum has had on Washington state nurseries is unknown. Although it is difficult to obtain reliable information on losses associated with the disruption of sales and some USDA-APHIS required mitigation actions, information on the number, size, and species of containerized nursery plants destroyed in Washington during 2004 and 2005 is included on EAN documentation. Using EANs and information provided by WSDA, our objective was to estimate the losses experienced by Washington state nurseries due to plant destruction relating to P. ramorum mitigation efforts during 2004 and 2005.

Copies of EANs issued in Washington in 2004 and 2005 were obtained from USDA-APHIS. Each nursery was assigned an alpha-numeric code (WA-1 through WA-32) to allow reference to specific nurseries while protecting their identity. The number, species, variety and size of plants destroyed were entered into spreadsheets and sorted by year, nursery code, and species. We calculated the total and mean number of plants destroyed per nursery, as well as the number of plants that were destroyed by species over the two-year period.

The retail dollar value of the plants that were destroyed was estimated using prices quoted by a Washington retail nursery based on size, species, and variety. This information was used to estimate the total value of the destroyed plants at all nurseries during 2004 and 2005 and the mean loss per nursery.

EAN forms indicated that containerized nursery plants were destroyed at thirty-two different retail nursery sites in Washington in an effort to eradicate P. ramorum. Plants were destroyed at twenty-two sites in 2004, and fifteen sites in 2005. Five nurseries destroyed plants two consecutive years due to repeat detections by WSDA. Based on WSDA information and EAN documentation, 17,266 containerized nursery plants were destroyed in Washington State between 2004 and 2005. Of these plants, 12000 were destroyed in 2004 and 5,266 were destroyed in 2005. The mean number of plants destroyed per nursery was 545 in 2004, 341 in 2005, and 540 over the two-year period.
The most commonly destroyed genera of containerized nursery stock in 2004 and 2005 included Rhododendron (89%), Calluna (4%), and Camellia (4%). The total retail value of plants destroyed over the two-year period was estimated at $423,043. The total retail value of plants destroyed in 2004 and 2005 were estimated at $246,144, and $176,899, respectively. The mean loss per nursery was estimated at $11,188 in 2004, $11,798 in 2005, and at $13,220 per nursery over the two year period. Five of the thirty-two nurseries that were issued EANs requiring plant destruction accounted for 94% of the total estimated value of plants destroyed in 2004 and 2005. These included WA-1 (30%), WA-6 (53%), WA-8 (2%), WA-15 (2%), and WA-20 (7%).

We have not attempted to estimate other costs to nurseries associated with implementing the USDA-APHIS confirmed nursery protocol such as labor, fees for burning or burial of plants in a landfill, potential soil and/or water mitigation treatments, as well as the lost opportunity cost associated with placing plants on a minimum 90 day hold for further monitoring. The owners of a nursery (WA-14) that had to destroy 109 plants between 2004 and 2005, accounting for roughly 1% of the total retail value of the losses experienced in Washington report that in addition to the value of the destroyed plants, they spent $30,000 for labor, disposal fees, and mitigation measures at their nursery (3). This suggests that the economic impacts of P. ramorum on Washington state nurseries are much greater than just the value of the plants that are destroyed.

References___________________


The Effects of Thinning and Borax Application on *Heterobasidion annosum* Infected Stands on the Olympia Peninsula

Dan Omdal, Amy Ramsey and Melanie Kallas Ricklefs

**Abstract**—*Heterobasidion annosum* is a common root and butt rotting pathogen on hemlocks in the Pacific Northwest. Forest management has increased the incidence of this fungus because *H. annosum* spreads via spores germinating on freshly cut stumps and stem wounds, as well as through root graphs between stumps and live trees. The objective of this study was to determine the effectiveness of borax stump treatment in preventing *H. annosum* spread following density management, or thinning, on the western side of the Olympic Peninsula in Washington State. Two hundred and sixty-four stumps were sampled twice over a period of two years. A chainsaw was used to cut a one inch horizontal piece, or a cookie, from the top of each stump in 2004, immediately following forest thinning, and again in the winter of 2005/2006. The cookie was then wrapped in newspaper and incubated in a plastic bag for ten days. The cookies were then examined under 30X magnification for the presence of *Spiniger meineckellum* (A. Olson) Stalpers, the anamorph of *H. annosum*. 82.3% of the stumps contained *H. annosum* during the first year of sampling. The application of borax prevented the spread and infection of *H. annosum* on 75% of the 17.7% (40) stumps without *H. annosum* during the first year of sampling.
Where Has all the Root Rot Gone? Ground Truthing Bear and Root Disease Damage in Western Washington

Dan Omdal, Jeff Moore, Melanie Kallas Ricklefs, and Amy Ramsey

Abstract—In 2002, approximately 1.8 million acres in western Washington contained trees killed or defoliated by insects and/or diseases. Bear damage, identified on more than 100,000 acres, was the most widespread west-side disturbance agent noted in the 2002 aerial survey. Damage caused by root disease, which when occurring in young Douglas-fir plantations and viewed from the air is virtually indistinguishable from bear damage, was noted on only 781 acres. Our objective was intended to validate bear damage and to determine the frequency with which root disease was found within these polygons. The first ten dead trees at 103 polygons identified by the 2004 aerial survey data to have bear damage were examined for root disease. One thousand two hundred and twenty-three recently dead trees were examined. The bole was examined for animal damage and root systems were exposed using a Pulaski. Bear damage was the primary mortality agent (i.e. associated with more trees than any other agent) on 54% of the polygons. Root disease was the primary mortality agent on 44% of the polygons. Root disease occurred as *Phellinus weirii*, *Heterobasidion annosum*, or *Armillaria* spp. *Phellinus weirii* was observed on 56% of the dead trees.


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Abstract—Phytophthora ramorum is the causal agent of sudden oak death (SOD), ramorum leaf blight, and ramorum dieback. Western Washington is at high risk for ramorum leaf blight/shoot dieback due to the presence of known P. ramorum hosts in the natural environment, suitable climatic conditions (extended periods of moist weather and mild temperatures), and the presence of nurseries receiving positively identified P. ramorum host stock. While Washington’s native oak species (Oregon White Oak) is not threatened by P. ramorum, Pacific madrone, huckleberry, rhododendron, and Douglas-fir, among many others, are susceptible hosts. Between January and October 2006, six nursery perimeter, twenty-four general forest, and eleven aquatic sites were surveyed and monitored. All 343 foliage samples were screened for P. ramorum and all but eight samples tested negative for P. ramorum. The eight positive P. ramorum samples were the first detections of P. ramorum outside of a nursery in Washington State and were from an aquatic sampling site (stream) that ran through a nursery previously identified as containing positive P. ramorum plant stock.
White Pine Blister Rust in Juvenile Western White Pine on State Lands in Washington

Dan Omdal, Amy Ramsey and Melanie Kallas Ricklefs

Abstract—Western white pine (Pinus monticola Dougl.) was once an integral part of the forest ecosystems of Washington. Around 1910, Cronartium ribicola J.C. Fisch., the causal organism of white pine blister rust (WPBR), was introduced into western North America from Europe, causing widespread mortality throughout the range of five-needle pines. In the last two decades the USDA Forest Service and the University of Idaho have established breeding programs to genetically enhance western white pine (WWP) for resistance to WPBR. During this time, the Washington Department of Natural Resources (DNR) has been steadily increasing the outplanting of WWP seedlings, including those genetically enhanced (F2 progeny) on state lands. A 2002 survey of WWP saplings and mature trees in Washington revealed infection levels of up to 100% in some geographic regions, but the percentage on genetically enhanced white pine was unknown. High infection levels in WWP saplings suggest that mortality due to WPBR may be underestimated by excluding juvenile (~ four years in age) white pine from studies. Between 2002 and 2005, 22 permanent plots were established across Washington to assess the development of WPBR in young plantations of F2 progeny WWP. Twelve plots were established in 2002/2003 and 10 plots were established in 2005. WPBR infection levels ranged from 0% to 93% across the 22 permanent plots. Less than 0.01% of the WWP were killed by stem girdling cankers. Two out of twelve plots established in 2002/2003 had WPBR infection rates of 0%. Eight out of ten plots established in 2005 had WPBR infection rates of 0%. The greatest number of cankers on one tree was 38 and these were cankers that were located within six inches of the main stem. The highest regional WPBR infection rates were in the Northwest region of the Washington, which includes areas west of the Cascade crest and north of Seattle to Canada. The 22 plots will continue to be monitored and efforts are currently underway to establish six new F3 WWP progeny resistant trials.
Screening for Genetic Resistance in Western Hemlock (Tsuga heterophylla) Provenances for Management of Dwarf Mistletoe (Arceuthobium tsugense)

Charlie Cartwright, Simon Shamoun, Sue Askew and Lea Rietman

Abstract—The screening for dwarf mistletoe resistance (Arceuthobium tsugense) in western hemlock (Tsuga heterophylla) provenances may provide an alternative method to control Arceuthobium tsugense (western hemlock dwarf mistletoe). Recently a variable retention harvest system has been adopted in BC, which maintains the structural complexity and biological legacies that can increase the wildlife habitat and maintain biodiversity. Variable retention practices can lead to an increase in hemlock dwarf mistletoe infection, resulting in a loss of tree growth and vigour, reduction in wood quality, and tree death. Five open-pollinated families from ten provenances from Oregon to BC were inoculated with dwarf mistletoe seed. Eight potted trees, grown in a greenhouse, were used for each family. Each tree was inoculated with 8 dwarf mistletoe seeds. Three years after inoculation the trees were assessed for height, root collar, apical dominance and dwarf mistletoe infection. Growth of the mistletoe was not correlated with the growth of the host plant. Analysis of variance revealed significant variation in growth characteristics by provenance at age 4, but no trends in susceptibility to infection were detected. A Duncan’s multiple range test detected that the Nitnat provenance showed a significantly lower infection than the other 9 provenances. Although most provenances were not significantly different for dwarf mistletoe infection, there was a wide variation in dwarf mistletoe resistance between families within a provenance. Family distinctions for dwarf mistletoe infection signify that resistant families can be selected within a provenance for future hemlock breeding programs for both increased growth rate and dwarf mistletoe resistance. Ongoing work includes monitoring transplanted surviving trees from this provenance trial at a field site located at Jordan River located on Vancouver Island, BC.

Future work will include dwarf mistletoe screening of 48 seed orchard families selected for growth traits (12 replications/family) inoculated with 8 dwarf mistletoe seeds/plant.


Charlie Cartwright is with the Cowichan Lake Research Station, BC Ministry of Forests, Mesachie Lake, BC.

Simon Shamoun is with the Pacific Forestry Centre, Canadian Forest Service, Victoria, BC.

Sue Askew and Lea Rietman are with the Forest Science Department, Faculty of Forestry, University of British Columbia, Vancouver, BC.
Pruning to Manage White Pine Blister Rust in the Central Rocky Mountains

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

Abstract—White pine blister rust, caused by the invasive fungus Cronartium ribicola, infects and often kills limber (Pinus flexilis) and Rocky Mountain bristlecone (P. aristata) pines. Techniques for managing the disease by removing cankers on stem and branch tissue have been successful in British Columbia, Canada and in the north central United States. This study tested the efficacy of both preventative (crown lifting) and pathological pruning (removal of cankers) in prevention and treatment of white pine blister rust on high value trees in recreation areas in Wyoming and Colorado. We found limber and bristlecone pines can be pruned to lift crowns and remove cankers up to 17 feet from the ground in 30 minutes or less with crews of 2 to 3 people. The long term results of the preventative and pathological pruning treatments will be available when the trees are reevaluated in 3 to 5 years.
Photo Contest
Ellen Goheen, Coordinator

Overview

WIFDWC’s first ever photo contest was held during the poster session on Wednesday evening, October 4, 2006. Participants submitted nine photos in the category of SIGNS, ten photos in the category of SYMPTOMS and eleven photos in the category of FOREST PATHOLOGISTS. A team of judges evaluated all of the photos and WIFDWC attendees were given the opportunity to vote for the “People’s Choice” in each category. First place winners for each category are featured on the back cover of these proceedings.

Judges’ Award Winning Photos

SIGNS (Page 32)

First Place
Subject: Armillaria ostoyae fan
Photographer: Alan Kanaskie

Second Place
Subject: Pucciniastrum goeppertianum on evergreen huckleberry
Photographer: Jeff Stone

Third Place
Subject: Polyporus squamosus
Photographer: Angel Saavedra

SYMPTOMS (Page 34)

First Place
Subject: Cristulariella depraedens on bigleaf maple
Photographer: Jeff Stone

Second Place
Subject: Dothistroma septosporum on lodgepole pine
Photographer: Dave Weaver

Third Place
Subject: Phytophthora cambivora canker
Photographer: Angel Saavedra

FOREST PATHOLOGISTS (Page 36)

First Place
Subject: Lew Roth passing pathology wisdom to the next generation
Photographer: Blakey Lockman

Second Place
Subject: Everett Hansen in meditation
Photographer: Mike McWilliams

Third Place
Subject: Borys Tkacz contemplating “Danger: tree men at work”
Photographer: Everett Hansen

People’s Choice Awards

SIGNS
Subject: Armillaria ostoyae fan
Photographer: Alan Kanaskie

SYMPTOMS
Subject: Cristulariella depraedens on bigleaf maple
Photographer: Jeff Stone

FOREST PATHOLOGISTS—A TIE!!

Subject: Lew Roth passing pathology wisdom to the next generation
Photographer: Blakey Lockman

Subject: “The Young Lionesses”: Susan Frankel and Ellen Michaels Goheen
Photographer: Don Goheen
Discussion

Verbal Reports

Paul Hennon—Literature review on hemlock dwarf mistletoe should be out in a couple of months.

Marcus Jackson—Completed 15-year remeasurement of Jane Taylor's Larch DM plots with Brennan Ferguson.

Brian Geils—Half complete in remeasuring Hawksworth's 1960's silvicultural plots last summer and hope to finish measurements next summer. Intermediate results were reported in WIFDWC proceedings. The thinning study incorporated different ages of lodgepole pine regeneration, different levels of dwarf mistletoe severity, and thinned vs. non-thinned.

Sue Askew and Charlie Cartright—Looking at hemlock resistant to dwarf mistletoe. Height, root collar, and dwarf mistletoe pressure were measured at Jordan River and lab inoculations were completed at Vancouver Island.

Funding for Dwarf Mistletoe Treatments

Bill Jacobi asked is if money was available for treatment. Jim Worrall mentioned that pathology money in the West dried up and was replaced with western bark beetle money and believed that no more than 10% of these monies it to be used for anything other than bark beetles. There could be some money for white pine blister rust and some money could come for fire suppression on Indian Reservations.

Bill Jacobi asked if there is still interest on the District level. Meeting participants generally responded that there was some tree girdling work on-going. Placerville has no money for genetics and resistance work and is not actively pursuing it. Dave Shaw questioned if the Healthy Forest Initiative was hampering suppression activity due to bark beetles and no money for dwarf mistletoe in Region 6. There was no definitive response.

Dwarf Mistletoe and Modeling Fire Risk

Bill Jacobi asked if anyone had ideas on how to get incidence, severity, frequency and distribution of dwarf mistletoe for modeling fire risk. Somebody mentioned that there was an STDP (Special Technology Development Program) proposal looking at dwarf mistletoe and fire in Region 6. Jim Worrall mentioned that there was a project using a grid system in place on the Uncompaghre Plateau. Bob Mathiasen is working on modeling fire behavior and dwarf mistletoe.

The dwarf mistletoe maps that are available for the West are very coarse (derived from FIA data, etc.). Risk map is very coarse and is not applicable at the drainage scale. Paul Hennon mentioned that thousands of stand data points were put on the map but were not geo-referenced and it would be more useful if they were geo-referenced. Region 6 has had no money for stand exams in years, but do have walk through exams. Plumas National Forest has drawers full of data, but too much stuff to make a map. Tahoe Forest started to work on this issue, but very little dwarf mistletoe was left after a big fire. Terry Shaw mentioned that dwarf mistletoe does not have a systematic distribution. Modelers use a 30 meter resolution that works well for trees, but does it work well for pests? Bill Jacobi mentioned CVU (common vegetation unit) and noted that knowledge is gone for stand exams. He asked how we get insects and other pests in this.

Discussion on New Taxonomy

Dave Shaw—Nickendt merged a bunch of species to one, worked on DNA. Hawksworth's system works well for land managers.

Suggestion made for a panel next year to discuss the nomenclature of dwarf mistletoe.

Submitted Reports

IX. Surveys

Marcus Jackson, USDA Forest Service Northern Region, and Brennan Ferguson, Ferguson Forest Pathology Consulting, Inc. Permanent plots were established in 1991 in order to evaluate spread and intensification of western larch dwarf mistletoe (*Arceuthobium laricis*) following removal of an infected overstory and (or) precommercial thinning. A report on the twelve-year remeasurements was completed in 2006 (Jackson et al. 2006). The positive effects of overstory removal and thinning on crop tree growth, and the negative effects of increased dwarf mistletoe spread and intensification within the thinning treatments, were statistically significant. The fifteen-year remeasurements were completed during the summer of 2006; a report on these results will be available by spring 2007.


Baker, Fred, Hansen, Mark, Shaw, John, Shelstad, Dixon and Mielke, Manfred. Field surveys are completed for a study comparing the incidence and severity of dwarf mistletoe determined by FIA in stands with FIA plots. All stands within a circle of a 0.5 mile radius containing an FIA plot were surveyed with transect lines 2-300 yards apart. These results were used in an ArcView simulation model to determine current area of infestation, and to project infestation for 20 years. We surveyed clusters of stands where the FIA plot has dwarf mistletoe or brooms, and the cluster around the nearest FIA plot without dwarf mistletoe. We are currently linking FIA data with field data for the comparison.

IX. Modeling

Don Robinson and Brian Geils published their work on modeling dwarf mistletoe—Modeling dwarf mistletoe at three scales: life history, ballistics, and contagion. This will (has) appear(ed) in Ecological Modeling (2006), doi:10.10.16/jecolmodel.2006.06.007.

IX. Miscellaneous

I had a call from a woman in New Mexico requesting information about the use of extracts of dwarf mistletoe on/in condoms and suppositories as a measure to fight sexually transmitted diseases. If anyone else has heard of this, please tell the rest of the story!  Submitted by (not dreamed up by. . ) Fred Baker
Root Disease Committee Meeting
October 3, 2006, Smithers, BC

Kathy Lewis, Acting Chair

Kathy Lewis substituted for Brennan Ferguson. There were no formal presentations at this year’s Committee meeting which was attended by about 30 people. Round-the-room reports were given by individuals who wished to do so. Further discussion arose on several topics, including initiating efforts to make pest models robust to effects of climate change, efficacy and economics of stumping as a root disease treatment, and the reliability of root disease data as collected on FIA plots.

Michelle Cleary—My PhD thesis entitled 'Host responses in Douglas-fir, western hemlock and western red cedar to infection by Armillaria ostoyae and Armillaria sinapina' is completed; I will defend in January 2007. Based on results showing effective resistance in western red cedar against Armillaria ostoyae, I have implemented operational trials that look at the effectiveness of stumping versus mixed-conifer plantations, including western red cedar. One site with five (1 ha) plots was done last year (2005/06) and another site with six (1 ha) plots was done this year (2006/07).

As of August 2006 I am working for the B.C. Ministry of Forests and Range as the Regional Forest Pathologist for the Southern Interior Region, based out of Kamloops. I am revisiting and remeasuring several stumping trials next year, including two 20-year old Phellinus trials and a 25-year old Armillaria stumping trial inherited from Duncan Morrison. Duncan currently has 35-year data from the Skimikin stumping trial (Phellinus & Armillaria root diseases) and is writing up results for that study.

Mike Cruickshank—We are working with Bill Wagner (CFS) on the economics of stumping as a root disease treatment and how losses affect long-term forestry economics. I was not aware that I had jumped into the climate change area, but I certainly have information that would affect this, especially long-term effects. I had been planning to include this as part of the impact manuscripts that I will write in the next few years. I am going to help Michelle Cleary and Rona Sturrock with impacts of Phellinus if the work gets funded. I would like to also work on impacts of Armillaria in spruce and also control methods and bioenergy depending on what we can get funding for.

I am still trying to get pathologists here in Canada to work together on a larger project (probably incidence and impacts) and convince people that we can benefit immensely by doing so (especially in the funding and policy area). This will be difficult to do as folks would rather work on their own projects but we will see if anyone can agree on this.

Greg Filip—Helen Maffei, Kris Chadwick, and I are analyzing data from a 15-year old study located south of Crater Lake National Park. It’s a site severely infected by Armillaria root disease where we are testing the effects of four silvicultural treatments (clearcutting, commercial thinning, group-selection harvesting, and shelterwood harvesting) on white and Shasta fir mortality 10-years post-treatment. We plan on using the site for a field trip for the IUFRO Root & Butt Rot meeting in August, 2007.

Mike McWilliams—Each year the Insect and Disease section (Oregon Department of Forestry) coordinates pre-harvest root disease surveys on state land in northwest Oregon at the request of Districts. Units are 100% surveyed for root disease and disease centers are marked on maps and then digitized to a GIS. Phellinus weirii is the most common pathogen detected. The Districts then use the disease information in formulating their site-specific prescriptions.

Richard Reich—Collaborating on a proposal to use LiDAR and digital photography on a 1,000 ha portion of the Aleza Lake Research Forest (east of Prince George). The main objectives are to estimate timber volume and carbon stocks. We will also estimate canopy structure including understory forest regeneration (tree heights, crown diameter and species composition). We want to determine the feasibility of using LiDAR as opposed to aerial photography (digital or conventional) for estimating understory attributes in MPB killed stands. The need is for a cost effective and accurate method of screening millions of hectares of MPB killed forest to identify stands containing understory worth retaining for mid-term timber supply. We will know by March 13, 2007 whether the proposal is approved. The LiDAR and photography were flown in 2006, and most field data has already been collected.
Continuing work on a project initiated in 1991 to map the relatively discrete distribution of *A. ostoyae* in the Robson Valley (the northernmost limit of its distribution in B.C.) and confirm the viability of using large-scale aerial photography in plantations to detect and stratify disease centres. Next year, in cooperation with Michelle Cleary, will be heading up a study looking at the population structure of *A. ostoyae* and *A. sinapina* in that area. This will become part of a thesis project for two students at UBC.

**Dave Schultz**—The Californians Against Toxic Substances (CATS) have been appealing many timber sales on the National Forests during the past year. Many of the appeal letters were extremely similar, or identical. The Regional Pesticide Use Coordinator, David Bakke, eventually assembled the most frequently asked questions and the Forest Health Staff provided answers. This information was used in a Q&A format to make a white paper that was made available to the National Forests. As it became less cumbersome for the National Forests to respond to the appeals to the use of borax, the number of appeals appears to have tapered off. Several National Forests attempted to solve the appeals to the use of borax by dropping the use of the registered pesticide. This simply generated more appeals because the original environmental document indicated it as important to use borax to prevent the initiation of additional annosus root disease centers. The words of the Government's own specialists were used to document that if the timber sales went forward without borax, there would be environmental degradation.

**Dave Shaw**—One of several co-authors, along with first author Bob Edmonds, of NSF grant application titled "Role of pathogens as disturbance agents in old-growth coastal temperate rainforests"; application was not funded.

**Terry Shaw**—WWETAC (USFS - Western Wildland Environmental Threat Assessment Center) is considering hosting a workshop that will get pest folks, including some modelers, together with climate change folks. Of all the pest models the root disease model is likely the most difficult one to consider dealing with the various climate change interactions, so the initial effort may center more on some of the bark beetles and defoliators.

There needs to be a meeting between the FIA and FHP folks regarding the situation with detecting and reporting root disease on FIA plots.

**Rona Sturrock**—As a comment to the group's discussion of the efficacy of stumping, Rona mentioned that a technology transfer note she published in 2000 (see below) demonstrated that stumping does reduce root disease inoculum, especially where and when it is done correctly (i.e. - infected stump roots not left hinged in the ground nor large root pieces left in the ground). However, many potential users still ask questions about the cost-benefit of stumping as a root disease treatment. This economic perspective of stumping has not been adequately addressed. Fortunately, Mike Cruickshank has recently begun work on this subject with a forester/economist at the Pacific Forestry Centre.


In response to a question from Everett Hansen about progress made on a project/manuscript Rona talked about at the 2005 Root Disease Committee meeting (a new method for differentiating *P. weirii* (now *P. sulphurascens*) homokaryons from heterokaryons), Rona said that the manuscript is still in the works. This is mostly due to delays in writing caused when Rona's colleague, and the paper's lead author, Dr. Young W. Lim moved in May 2006 from a term position at UBC to a full-time position at the Seoul National University in South Korea. The paper should be completed and published in 2007.

**Alex Woods**—I have re-measured my Nichyeskwa Creek Tomentosus root disease stumping trial for the past 10-years (see below). I have annually measured height and diameter and found that the stumped sights have significantly greater height (11%) and diameter (20%) than the control units. There is slightly less infection in the stumped areas but the differences are not significant yet. (Contact Alex for detailed information and/or a poster on this work.)

Background: Initiated in 1995, the Nichyeskwa Creek Tomentosus root disease stumping trial is now in its 10th year of data collection. The trial site is located approximately 100 km north-east of Smithers, BC in the Bulkley Forest District (1260 26' N, 550 44' E ), at an elevation of 800-850 m. Tomentosus root disease caused by *Inonotus tomentosus* is the most destructive root disease in the Northern Interior Forest Region in British Columbia. Unlike the southern interior of BC where Armillaria and Laminated root disease studies have been on going since the late 1960's (e.g. - the Skimikin trial), there has been relatively little research
conducted on the control of Tomentosus. The efficacy of stump removal for the control of Tomentosus root disease is not well known. Similarly, the time required for subsurface Tomentosus inoculum to deteriorate to a non-infectious state in a fallow treatment has not been determined. The purpose of this trial is to test the
efficacy and efficiency of stumping in areas infected with Tomentosus root disease.

Jim Worrall—Working to distinguish the biological species of *H. annosum* infecting various conifer hosts in different geographical locations.
Blister Rust "Happy Hour" Committee Meeting

October 3, 2006, Smithers, BC

Holly Kearns, Chair

The Rust Committee “Happy Hour” meeting was held on October 3, 2006. This meeting was well attended thanks in part to the beer and wine tickets distributed by our hosts in Smithers. The reports on recent, current, and pending activities were as follows.

**Blakey Lockman**—The Whitebark Limber Pine Information System (WLIS) is available on-line (http://www.fs.fed.us/r1-r4/spf/hp/prog/programs2.html). This database contains information on the geographic extent and condition of whitebark and limber pines.

**Bill Woodruff**—The second year of California’s (USDA Forest Service R5) survey of high elevation five-needled pines has been completed – information will be forthcoming. At the July IUFRO Rusts of Forest Trees conference, there was some discussion on resistance to western gall rust in ponderosa pine and concerns over importing the pathogen to New Zealand.

**Ellen Goheen**—The August 2006 conference Whitebark Pine: A Pacific Coast Perspective was a huge success with 85 attendees and 25 contributed papers. The proceedings will soon be available on-line (http://www.fs.fed.us/r6/nr/fid/wbpine/index.shtml). There has been some talk of a Pacific Coast chapter of the Whiterack Pine Ecosystem Foundation (WPEF). In surveys of Oregon whitebark pines, they found that rust is present and levels of mountain pine beetles are lower than those found in the Rocky Mountains. The Dorena Genetic Resource Center will soon be advertising for a pathology/silviculture SCEP position.

**Bill Jacobi**—Reported on the following Colorado activities:

Rocky Mt Research Station: Anna Schoettle is continuing to monitor the infection rate from inoculations at Dorena OR, of bristlecone pine collected in various locations in Colorado. Cones were collected from infected and non-infected limber pine to determine if there are any differences in major resistance gene occurrence.

A management guide for white pine blister rust on bristlecone and limber pine is in the final stages of production by Forest Health Management (FHM, USDA Forest Service R2).

Kelly Burns and Jim Blodgett (USDA Forest Service R2) with Marcus Jackson (USDA Forest Service R1) have an evaluation monitoring project in which long-term ecological monitoring plots were established by FHM in limber pine stands lightly or heavily impacted by white pine blister rust in Colorado and Southern WY in the summer 2006. Plots will be established in northern WY and Montana in 2007.

Rocky Mt Station, CSU, and two local Districts helped collect cones of limber pine from phenotypic resistant trees on Roosevelt and Medicine Bow NFs. Earlier than expected cone development and insect damage restricted collecting to no more than about 40 trees. Future collections will continue to add to this modest start. The two districts made general collections of cones for future restoration work. Seed will be processed at the Bessey Nursery.

White pine blister rust (WPBR) was found south of Rocky Mountain National Park by Jim Hoffman, Marcus Jackson, and Blakey Lockman in March, 2006.

Colorado State University has an on-going project on pathological pruning of limber pine at two locations.

**John Schwandt**—Completed year long Washington Office detail as whitebark pine coordinator. Summary publication with a range-wide assessment of whitebark pine health and restoration strategies is now available. An earmark for whitebark pine restoration in Montana, Idaho, and Wyoming is on the US congressional budget; if it passes, $800,000 funding will be available for restoration projects in 2007.

A seed planting trial of whitebark pine near Baker City, Oregon found that a warm stratification period prior to fall sowing resulted in greatly enhanced germinating the following spring (38% vs 0-10% for
other treatments). We will now be interested in seeing if the early germinants survive the summer and if additional germination occurs next year.

Work continues in establishing permanent plots in western white pine plantations to see if pruning provides as much benefit in stock with improved resistance (F2) as we have seen with unimproved stock. A new pruning handbook has just come out that was developed as a PNW extension publication that is aimed at showing forest owners how to prune to reduce blister rust impacts and how to decide if pruning would be worthwhile for them (available free from me).

We are continuing to annually monitor canker growth on over 100 cankers on F2 stock and hope to do some preliminary data analysis on the first 4-5 years of growth this winter. Amy Eckert (OSU graduate student) continues to analyze bole cankers on F2 stock and has observed different canker growth rates.

Permanent plots in about 20 F1 and F2 plantations on FS and Idaho Department of Lands were remeasured in cooperation with Brennan Ferguson to determine infection levels and mortality rates over the past 5-10 years. Data analysis and write-ups are scheduled for this winter.

**Don Goheen**—Surveys are being conducted in western white and sugar pine plantations and natural stands as a result of increased interest in mountain pine beetle and WPBR.

**Dave Russell**—Working on transition management plans to move 2nd generation sugar pine to the BLM's Tyrrel Seed Orchard in cooperation with Dorena Tree Improvement Center as a result of management decisions not to do 2nd generation at the BLM's Charles A Sprague Seed Orchard.

**Alex Woods**—Interested in management of western white pine - to prune or not to prune resistant Idaho stock? In some cases in the past there has been a mixed message from BC Ministry of Forests regarding western white pine management. Forest licensees that pay for the rust resistant Idaho seed are not required to prune these trees to improve their chances of survival even more. In the opinion of BC Ministry of Forest pathologists if the effort is being made to reforest with western white pine, a noble cause, then every advantage the trees could have over the disease should be afforded them. Alex also announced that Sybille Haeussler of the Bulkley Valley Research Centre has put together a research proposal for restoration of whitebark pine along a natural gas pipeline that runs through whitebark pine habitat. This could represent a novel approach to securing funding for forest ecology/pathology research.

**Ron Diprose**—Has been working on the fourth year of controlled breeding of western white pine; controlled full-sib crosses are currently being out-planted. Issues with western white pine bough collections for the floral industry wiping out study areas.

**Amy Ramsey**—Washington State Department of Natural Resources has been monitoring 22 permanent plots in F2 western white pine stock for three years. This year, six new plots were established in F3 stock from Dorena and 36 families will be out-planted this winter.

**Paul Zambino and Bryce Richardson (not in attendance, presented by Holly Kearns)**—Conducted inoculation tests on both whole plants and detached leaves of *Pedicularis racemosa* using aeciospores and urediniospores of the white pine blister rust fungus (*Cronartium ribicola*) collected from locations across North America. They are analyzing patterns of successful infections representing Western, Eastern, and Midwestern populations of the fungus. *Pedicularis racemosa* is very common in whitebark pine stands and may be important to the epidemiology of WPBR. The ability of the fungus from areas outside of the geographical range of whitebark pine to infect this non-*Ribes* alternate host will have important implications for understanding 1) origins / diversity of North American populations of the rust fungus, and 2) the fungus' reported global-scale patterns of specialization to different alternate hosts.

**Rich Hunt (not in attendance, presented by Holly Kearns)**—Could not attend the meeting as he was out re-checking all the Spring spot counted western white pine seedlings to confirm that they are still spotted. He is also establishing a field trial. The trial is based on the hypothesis that stock derived from the Moscow arboretum is resistant because resistance increases with age and elevation. (Unfortunately, samples are small, particularly at the low elevation site). A positive outcome would parallel his published data on selected BC parents (2005, Can. J. Plant Pathol.).

**Jim Hoffman (not in attendance, presented by Holly Kearns)**—Established ten permanent plots in whitebark pine in southern Idaho and northern Nevada. For the most part, WPBR incidence was less than 20%. Southern Idaho may be a unique area to view low
WPBR incidence levels in whitebark pine, so Jim invites anybody interested to “come on down.” The story is very different for limber pines in southern Idaho, which are currently being killed by the disease.
Hazard Tree Committee Meeting  
October 4, 2006, Smithers, BC  

Pete Angwin, Chair

Jim Worrall (USDA Forest Service, Rocky Mountain Region) provided details of upcoming 5th Hazard Tree Workshop, scheduled for 2007, June 11-4 at the Homestead Resort near Salt Lake City. A poster and handout gave highlights of the tentative agenda and of the venue.

Judy Adams (USDA Forest Service, Forest Health Technology Enterprise Team) gave a brief overview of the International Tree Failure Database (ITFD) for those who were unfamiliar with the project. Approximately 4900 reports are now in ITFD (4000 transferred from the California Tree Failure Report Program and 900 new submissions). About 1500 individuals have received ITFD training since the system was online. The ITFD is a production web site with few changes taking place until 2010. Marketing is presently our area of concentration with articles being written by committee members, and our poster displayed at conferences along with the recently completed pamphlet.

Lori Trummer (USDA Forest Service, Alaska Region) reported on increased hazard tree awareness and activity in the Alaska Region due in part to a 2005 hazard tree fatality in the backcountry of the Chugach National Forest. A training program was developed and presented to employees, as well as Parks and Recreation staff from the Municipalities of Anchorage and Palmer, Denali National Park, and at the statewide Alaska Parks and Recreation conference. Development of a leaflet, “Safe Backcountry Travel in Alaska – Hazard Trees” is nearing completion; Lori presented a draft. The Regional Hazard Tree web pages are also being converted to a book format.

Greg Filip (USDA Forest Service, Pacific Northwest Region) presented information on the newly developed roadside danger-tree policy and workshops to support them. Fast tree growth, lack of timber haulers keeping roadsides clear, fires, accidents, and complaints have contributed to the issue. Development of a Regional Directive has involved Regional Engineering, OR and WA safety personnel, and the Regional Leadership Team. It includes training based on a Field Guide for Danger Tree Identification and Response. The training is a joint venture between Forest Products, Forest Health Protection, Engineering and Wildlife. Details and the Field Guide can be found at http://www.fs.fed.us/r6/nr/fp/FPWebPage/FP70104A/FP70104A.htm
Previous Minutes

2005 Business Meeting Minutes were circulated to all attendees. Ellen Goheen moved to approve the minutes and Bob Edmonds seconded the motion. The 2005 minutes were accepted unanimously by voice vote.

Acknowledgements

Chair showed appreciation to the 2006 Organizing Committee and Holly Kearns for accepting the Interim Program Chair for the 2007 meeting. Kathy Lewis noted that a greater proportion of early registrations would reduce stress for the Local Arrangements Chair. Alex Woods thanked the following people—

Karen McKeown for lining up the caterers and for making preliminary contacts with 'Ksan, where the banquet was held.
Laurel Mould, 'Ksan Administrator, for allowing us to have our banquet there and for lining up the banquet caterers and the Gitsan Dancers.
Sadie and Victor Mowat and family, for catering the traditional feast.
Stefan Zeglen and Dorothy Beneke, for taking care of registration and for moral support.
Erin Havard for helping with everything from audio/visual support to bar-tending, to poster designing, to trail clearing etc etc.
And he thanked Jane, his wife, and Kate and Hamish, his kids, for putting up with him through it all.

Alex said he wasn't able to thank everyone, but these folks really made it work.

Treasurer's Report

John Schwandt reported that in spite of uncertain budgets in the US which resulted in postponing the meeting into October, we had 74 registrants including 61 regular members, and 13 students; 35 each from Canada and the US and 4 from other countries. The following is a summary of transactions for the WIFDWC account from 12/1/2005 to 12/31/2006.
### WIFDWC and Hazard Tree Committee Accounts (US$)

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<thead>
<tr>
<th>TRANSACTION</th>
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<th>Expenses</th>
<th>Balance</th>
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<tr>
<td><strong>2006 WIFDWC meeting – Smithers, BC</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total registration</td>
<td>18,384.35</td>
<td></td>
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<tr>
<td>Meeting room rental, breaks</td>
<td>3,263.52</td>
<td></td>
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<tr>
<td>Hotel costs: rooms, breaks, banquet, lunches</td>
<td>2,951.43</td>
<td></td>
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</tr>
<tr>
<td>2 Field trips transportation &amp; lunches</td>
<td>6,893.41</td>
<td></td>
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<tr>
<td>Outside speaker expenses</td>
<td>1,000.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ice Cream social</td>
<td>547.32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospitality, Supplies, and awards</td>
<td>1,209.52</td>
<td></td>
<td></td>
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<tr>
<td>Mtg Room Advance (paid back to acct)</td>
<td>1,000.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Balance from 06 meeting = $1,519.15)</td>
<td></td>
<td></td>
<td>13,154.13</td>
</tr>
</tbody>
</table>

**2005 Meeting Account Activity**

| 2005 Printing costs | 1,460.00 | 11,694.13 |
| 2005 Expenses | 442.69 |         |
| 2005 Speaker expense | 500.00 | 10,751.44 |

**Other Account Activity**

| 2007 meeting reservation | 1,000.00 | 9,751.44 |
| Bank Interest/dividends | 105.84 | 9,857.28 |

**WIFDWC Balance as of 12/31/2006**

| 05 proceedings printing/mailing estimate | 2,500.00 | $9,857.28 |

**Hazard Tree Balance – last report**

| 3/27/06 -07 Hazard tree meeting setup (SLC) | 150.00 | 1,544.54 |
| 7/19/2005--07 Hazard tree conf. mtg. reservation | 1,200.00 |         |

**Hazard Tree Balance as of 12/31/06**

| $344.54 |         |

**Total Bank Balance as of 12/31/06 (WIFDWC + Hazard Tree)**

| $10,201.82 |         |

John noted that very little interest is currently earned on the account. For those that need it for travel, our Federal Tax Id. number is: #91-1267879
Old Business________________

Chair noted that the letter to the Chief of the USDA Forest Service addressing the reduction in pathology researchers yielded no known impact.

The surcharge, as mandated during last year's business meeting, was implemented for this meeting to cover the debt on WIFDWC Proceedings CDs. All people who had purchased a CD received a $10 reduction in their registration costs. The surcharge should continue for another 2 years as described in the 2005 business meeting minutes.

During the 2005 business meeting, Brian Geils, Bill Jacobi, and John Guyon were assigned to a committee to research if republication in the WIFDWC proceedings would inhibit subsequent publication in peer-reviewed journals. This question arose due to concerns about a motion that had been tabled regarding placement of the proceeding on the WIFDWC website. Since none of the 3 committee members were present at the meeting, and none present could comment on the progress, the subject was tabled again.

A motion was passed during the 2005 business meeting to assign Marcus Jackson to simplifying the format for the 2006 proceedings. An updated version of author guidelines was provided in the registration packet and a simplified version of the guidelines was provided at the business meeting. Marcus will send simplified guidelines to panel moderators and to Judy Adams for placement on the WIFDWC website.

Nominations_________________

The Railroad Committee, led by Ellen Goheen, nominated Stefan Zeglen as the 2007 WIFDWC Chair and Mike McWilliams as secretary. Don Goheen seconded the nominations. Both were accepted by voice vote.

Future Meetings________________

2007 Meeting

Next year's WIFDWC will be held at the Radisson Poco Diablo Resort on October 15th-19th in Sedona, Arizona. The Chair read the following note from the local arrangements committee (Mary Lou Fairweather, Bob Mathiasen, and Brian Geils) who were all unable to attend the Smithers conference.

Located at about 4,500 feet elevation in central Arizona, Sedona is a World class destination spot due to its red rocks, blue skies and perfect October climate. There are local brew pubs, great eateries, and lots of shops. Sedona also features positive vortices spread around town, nice places to hike to if you are in need of a jolt of positive energy. Field trips will include riding through the birthplace of C. Hart Merriam’s life zone concept, on the San Francisco Peaks near Flagstaff.

2008 Meeting

Blakey Lockman said that several locations are currently under consideration for the 2008 meeting in Montana. One option is the Bozeman/Livingston in south-central/western Montana to showcase some of the whitebark pine cooperative programs there. Another option is the Flathead/Kalispel area where root diseases, dwarf mistletoes and deterioration of fire-killed trees could be showcased. A wide variety of suggestions were given to the Montana contingent with the general consensus that the hosts would select the specific location. There was discussion of holding the meeting in August, but it will be held in early/mid September 2008 with consideration to related professional meetings.

2009 Joint Meeting

Alex Woods moved we do not hold a joint entomology/pathology meeting in 2009 because those meetings are often dominated by entomologists and their issues. Ellen Goheen seconded the motion. After much discussion, the motion passed by a hand vote of 17 to 4. There will be no joint meeting in 2009.

Location of 2009 Meeting

Several possibilities were discussed. John Schwandt and Don Goheen stated that Greg Filip had mentioned that Wenatchee, WA was a possibility if nobody else was interested, but thought Region 2 would be a good location. Judy Adams mentioned that Jim Worrall had mentioned holding it in Region 2, but was apprehensive due to the joint meeting. Stefan Zeglen motioned we hold the meeting in CO in 2009. Bart van der Kamp and Everett Hansen asked to amend the motion to have the Chair speak with Colorado representatives to see if they would accept holding the meeting there in 2009. Don Goheen seconded the motion. The motion was passed as amended.
New Business

Dave Shaw asked for people’s thoughts regarding competition between graduate student paper presentations. After much discussion, no motion was put forward. Walt Thies asked that leadership deal with graduate student assistance. The Chair noted that it can be different for each conference, but agreed to form a committee consisting of Blakey Lockman, John Schwandt, Bob Edmonds, and Holly Kearns to explore graduate student assistance options.

Stefan Zeglen noted that the parameters for the outstanding achievement award need revision. He looked back at the history and found it was originally skewed towards one kind of nominee and against another type and otherwise vague, creating difficulties for the 2006 Achievement Award Committee. Stefan asked for a reaffirmation of the purpose of the award. Ellen Goheen stated that it is to “recognize outstanding achievement in forest pathology.” Several members noted that the award should consider the full spectrum of forest pathology and it did not have to be awarded every year. Richard Reich is now the Canadian representative on the Outstanding Achievement Award Committee, joining Dave Shaw and Brennan Ferguson.

Blakey Lockman has been trying to maintain the updates for the WIFDWC list-server in recent years. She recently learned the updates she had forwarded to the server provider had not been installed. Blakey’s been maintaining the list of e-mails on her own computer from where she can send out a mailing, but nobody else can. Judy Adams offered to provide a new home for the list-server. Blakey said that she would confirm that the current server manager has stopped maintaining it and let Judy know. Concerns were raised about the future of the FORPATH list-serve, given the news about the WIFDWC list-server. Nobody at the meeting knew if the FORPATH list-serve had been recently updated. The Chair stated there is an advantage to maintaining the two lists together, but if the WIFDWC list-serve isn’t being maintained it will be moved to Judy.

John Schwandt has been investigating the designation of WIFDWC as a non-profit 501-3C. Non-profit status would allow WIFDWC to accept donations and honoraria and may provide tax benefits to attendees. He also mentioned he was planning to look into the costs of accepting credit cards by WIFDWC. Several members suggested John consider credit card payments a higher priority than the non-profit designation.

John Schwandt mentioned a Utah State University proposal to print the WIFDWC proceedings. He noted it may be easier for the secretary to work with a standard printer/binder each year. John Guyon has been working with USU this year and it appears they will print publications and produce a .pdf file that could be placed on the WIFDWC website for a reasonable cost. The group agreed it would be best to see the product(s) produced by USU this year and place the subject on next year’s business meeting agenda for consideration.

The attending members recognized the fine contributions of the 2006 WIFDWC Chair.

Everett Hansen moved to adjourn the meeting and Ellen Goheen seconded the motion. The motion passed.
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>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1953</td>
<td>Victoria, BC</td>
<td>R. Foster</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>1954</td>
<td>Berkeley, CA</td>
<td>W. Wagener</td>
<td>P. Lightle</td>
<td></td>
<td></td>
</tr>
<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>
</tr>
<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>
</tr>
<tr>
<td>5</td>
<td>1957</td>
<td>Salem, OR</td>
<td>G. Thomas</td>
<td>T. Childs</td>
<td>R. Gilbertson</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>
</tr>
<tr>
<td>7</td>
<td>1959</td>
<td>Pullman, WA</td>
<td>H. Offord</td>
<td>R. Foster</td>
<td>C. Shaw</td>
<td></td>
</tr>
<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>
</tr>
<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. McMin</td>
</tr>
<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>
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<tr>
<td>12</td>
<td>1964</td>
<td>Berkeley, CA</td>
<td>K. Shea</td>
<td>R. Scharf</td>
<td>C. Leaphart</td>
<td>H. Offord</td>
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<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>
</tr>
<tr>
<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>
</tr>
<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>
</tr>
<tr>
<td>16</td>
<td>1968</td>
<td>Couer D'Alene, ID</td>
<td>S. Andrews</td>
<td>R. McMin</td>
<td>J. Stewart</td>
<td>C. Leaphart</td>
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<tr>
<td>18</td>
<td>1970</td>
<td>Harrison Hot Springs, BC</td>
<td>R. Scharpf</td>
<td>H. Toko</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>
</tr>
<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>
</tr>
<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>
</tr>
<tr>
<td>22</td>
<td>1974</td>
<td>Monterey, CA</td>
<td>R. Bega</td>
<td>D. Hocking</td>
<td>J. Parmeter</td>
<td></td>
</tr>
<tr>
<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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<tr>
<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>
</tr>
<tr>
<td>26</td>
<td>1978</td>
<td>Tucson, AZ</td>
<td>R. Smith</td>
<td>D. Drummond</td>
<td>L. Weir</td>
<td>J. Hadfield</td>
</tr>
<tr>
<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>
</tr>
<tr>
<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>
</tr>
<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. Bloomlo</td>
<td>W. Jacobi</td>
<td>E. Hansen</td>
<td>F. Cobb</td>
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<tr>
<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. Schwanadt</td>
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<tr>
<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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<tr>
<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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<tr>
<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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<tr>
<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>
</tr>
<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. Kanakie</td>
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</tbody>
</table>

Bylaws were amended in 1989 to split the office of Secretary-Treasurer.
Meetings and Officers, 1990—2006

<table>
<thead>
<tr>
<th>Annual</th>
<th>Year</th>
<th>Location</th>
<th>Chairperson</th>
<th>Secretary</th>
<th>Treasurer</th>
<th>Program Chair</th>
<th>Local Arrangements</th>
<th>Historian</th>
<th>Web Coordinator</th>
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</thead>
<tbody>
<tr>
<td>38</td>
<td>1990</td>
<td>Redding, CA</td>
<td>R. Hunt</td>
<td>J. Hoffman</td>
<td>K. Russell</td>
<td>M. Marosy</td>
<td>G. DeNitto</td>
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<tr>
<td>40</td>
<td>1992</td>
<td>Durango, CO</td>
<td>D. Morrison</td>
<td>S. Frankel</td>
<td>K. Russell</td>
<td>C. G. Shaw III</td>
<td>P. Angwin</td>
<td></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>M. Schultz</td>
<td>D. Conklin</td>
<td>T. Rogers</td>
<td></td>
</tr>
<tr>
<td>43</td>
<td>1995</td>
<td>Whitefish, MT</td>
<td>S. Frankel</td>
<td>R. Mathiasen</td>
<td>K. Russell</td>
<td>R. Mathiasen</td>
<td>J. Taylor</td>
<td>J. Schwanld</td>
<td></td>
</tr>
<tr>
<td>44</td>
<td>1996</td>
<td>Hood River, OR</td>
<td>J. Kliejunas</td>
<td>J. Beatty</td>
<td>J. Schwanld</td>
<td>S. Campbell</td>
<td>J. Beatty</td>
<td>K. Russell</td>
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<tr>
<td>45</td>
<td>1997</td>
<td>Prince George, BC</td>
<td>W. Thies</td>
<td>R. Sturrock</td>
<td>J. Schwanld</td>
<td>K. Lewis</td>
<td>R. Reich</td>
<td>K. Lewis</td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>2000</td>
<td>Waikoloa, HI</td>
<td>W. Jacobi</td>
<td>P. Angwin</td>
<td>J. Schwanld</td>
<td>S. Hague</td>
<td>J. Beatty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>54</td>
<td>2006</td>
<td>Smithers, BC</td>
<td>K. Lewis</td>
<td>M. Jackson</td>
<td>J. Schwanld</td>
<td>B. Lockman</td>
<td>A. Woods</td>
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</table>

Bylaws passed in 1998 WIFDWC Business Meeting identify officers as chairperson and secretary elected at annual business meeting and treasurer and historian, elected every five years.
### Standing Committees and Chairs, 1994—2006

<table>
<thead>
<tr>
<th>Committee</th>
<th>Chairperson</th>
<th>Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hazard Trees</td>
<td>J. Pronos</td>
<td>1994-2005</td>
</tr>
<tr>
<td></td>
<td>P. Angwin</td>
<td>2006</td>
</tr>
<tr>
<td>Dwarf Mistletoe</td>
<td>R. Mathiasen</td>
<td>1994—2000</td>
</tr>
<tr>
<td></td>
<td>F. Baker</td>
<td>2004—2006</td>
</tr>
<tr>
<td>Root Disease</td>
<td>G. Filip</td>
<td>1994—1995</td>
</tr>
<tr>
<td></td>
<td>E. Michaels Goheen</td>
<td>1996—2005</td>
</tr>
<tr>
<td></td>
<td>B. Ferguson</td>
<td>2006</td>
</tr>
<tr>
<td>Rust</td>
<td>J. Schwandt</td>
<td>1994, 2005</td>
</tr>
<tr>
<td></td>
<td>R. Hunt</td>
<td>1995-2004</td>
</tr>
<tr>
<td></td>
<td>H. Kearns</td>
<td>2006</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>

*Disease Control was disbanded and Nursery Pathology established in 2002.*
Active members are those who have attended a meeting in the past five years. The most recent and up to four previous meeting years are noted below member's name.

Judy A. Adams
USDA Forest Service - FHTET
2150 Centre Ave., Bldg A
Fort Collins, CO 80526 USA
(970) 295-5846
jadams04@fs.fed.us

Mike Albers
(2004)
1201 East Hwy. 2
Grand Rapids, MN 55744 USA
Mike.albers@dnr.state.mn.us

Stew Alcock
(2006)
P.O. Box 2110
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(250) 788-7904
alcock@intpac.ca

Pete Angwin
3644 Avetech Parkway
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pangwin@fs.fed.us

John Anhold
Southwest Forest Science Complex
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Flagstaff, AZ 86001 USA
(928) 556-2073
janhold@fs.fed.us

Sue Askew
UBC/CFS
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Victoria, BC V9C 3B7 CANADA
(250) 474-5499
saskew@pfc.forestry.ca

Fred Baker
Dept of FRWS, Utah State University
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Logan, UT 84322-5230 USA
(435) 797-2550
fred.baker@usu.edu

Karen Bartlett
(2003)
School of Occupational and Environmental Hygiene
2206 East Mall
Vancouver, BC V6T 1Z3 CANADA
(604) 822-6019
kbartlet@interchange.ubc.ca

Alan Baxter
(2006)
P.I.R. Division
P.O. Box 3130
Smithers, BC CANADA
(250) 847-2656

Russell Beam
(2006, 2005)
Dept. of BSPM
885 Kline Drive
Lakewood, CO 80215 USA
(303) 249-2051
russell.beam@colostate.edu
Jerome Beatty  
USDA FS  
3160 N.E. 3rd Street  
Prineville, OR 97754 USA  
(541) 231-8942  
jbeatty@fs.fed.us

Miroslava "Mirka" Bednarova  
(2006)  
Department of Forest Protection and Game Mngt.  
Zemedelska 1, 613 00  
Brno, CZ Czech Republic  
svezi.mirka@email.cz

Frank Betlejewski  
(2003)  
2606 Old Stage Road  
Central Point, OR 97502 USA  
(541) 858-6127  
fbetlejewski@fs.fed.us

James Blodgett  
Forest Health Management  
1730 S.ame Rd  
Rapid City, SD 57702 USA  
605-394-6191  
jblodgett@fs.fed.us

Rosie Bradshaw  
(2006)  
Institute of Molecular Biosciences  
Turitea Campus, Private Bag 11222  
Palmerston North, NZ  
646 350 5515  
r.e.bradshaw@massey.ac.nz

Clive Brasier  
(2003, 2000)  
Forest Research Agency  
Alice Holt Lodge  
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Honorary Life Members

According to the bylaws passed at the 1998 WIFDWC business meeting [and amended at the 1999 and 2000 business meetings], honorary life membership is automatically awarded to those members of WIFDWC who have attended at least five previous meetings of WIFDWC, and have retired from active forest pathology endeavors. Newly retired members who meet these criteria should notify the current WIFDWC Chairperson of their status. Other members who have retired but do not meet the attendance criteria, or other outstanding contributors to the field of Forest Pathology may request, or be proposed for, Honorary Life Membership by members present at an annual business meeting.

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## WIFDWC Honorary Life Members

### Complete List (D = Deceased)

<table>
<thead>
<tr>
<th>Year</th>
<th>Members</th>
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<tbody>
<tr>
<td>1956</td>
<td>Don Buckland (D)</td>
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<tr>
<td>1959</td>
<td>Norm Engelhart (D) John Hunt (D)</td>
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<td>1960</td>
<td>Hans Hansen (D) Albert Slipp (D) Charles Waters (D)</td>
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<td>1965</td>
<td>Lowell Farmer Harold Offord (D) Wilhelm Solheim (D) Willis Wagener (D) Ernest Wright (D)</td>
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<td>1966</td>
<td>Jesse Bedwell (D) Warren Benedict (D) Lake Gill (D) John Gynn (D) Homer Hartman (D) James Kimmy (D) James Mielke (D) Virgil Moss (D)</td>
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<td>1967</td>
<td>John Bier (D) Paul Keener (D)</td>
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<td>Toby Childs (D) Ross Davidson (D) John Hansbrough (D) Clarence Quick (D)</td>
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<td>Richard &quot;Dick&quot; Bingham David Etheridge Ray Foster (D) Don Leaphart (D) Jack Roff</td>
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<td>George Harvey (D) Alex Molnar Nagy Oshima (D) Phil Thomas (D) Bratislav Zak</td>
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<td>Lewis Roth</td>
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<td>Clarence Gordon (D) Lee Paine (D)</td>
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<td>Oscar Dooling (D) Jerry Riffle James Trappe John Woo (D)</td>
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<td>John Hopkins</td>
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<td>Art Parker (D)</td>
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<td>William Bloomberg (D) Richard B. Smith Roy Whitney</td>
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<td>Frank Hawkworth (D) Otis Maloy John Parmeter Robert Scharpf Stuart Whitney Ed Wicker</td>
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<td>Roy Blomstrom (D) Charles Driver Bob Harvey Vidar Nordin</td>
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<td>Dave French (D) Ray Hoff Tom Nicholls E. Mike Sharon Richard S. Smith</td>
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<td>James Ginns Kenelm Russell Jack Sutherland</td>
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<td>Tom McGrath Pritam Singh James Stewart Allen Van Sickle</td>
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<td>Alan Harvey</td>
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<td>James Byler David Johnson</td>
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<td>Robert Gilbertson</td>
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<td>2004</td>
<td>Duncan Morrison John Muir Geral McDonald Rich Hunt Mike Schomaker Clive Brasier</td>
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In Memoriam

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1946-2007

Dave grew up and went to school in upstate New York, receiving a B.S. in 1968 from the State University of New York in Syracuse. Following a brief tour with the Agricultural Research Service, he enlisted in the Army in 1969 and served in Vietnam, earning a Bronze Star and several other medals. Returning to SUNY Syracuse after his military service, he was granted a Ph.D. in forest entomology in 1976.

Dave started his permanent Forest Service career as a staff entomologist in the Region 5 Regional Office in San Francisco. When Forest Health Program service areas were created, Dave came to the Shasta-Trinity National Forest in 1989, spending the rest of his career providing technical assistance to landowners and resource managers to promote the health, productivity and diversity of forests across all ownership boundaries in northern California.

Dave participated in many WIFDWC meetings throughout his career. His friends and colleagues came to appreciate him as a master of dry wit, an encyclopedia of knowledge of the West, and an artist of frugality. He was one of a kind.