59th Annual
WIFDWC
2011
Leavenworth, WA

Proceedings of the 59th Annual Western International Forest Disease Work Conference

October 10-14, 2011
Leavenworth, Washington

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CHAIR'S OPENING REMARKS

Pete Angwin[1]

On behalf of the WIFDWC Organizing Committee, welcome to the 59th annual Western International Forest Disease Work Conference, here in beautiful Leavenworth, Washington! I think you'll find that we have a fantastic week of freewheeling discussions, field trips and all of the other things that make WIFDWC so special in store, so I invite you to dive right in.

At this point, I'd like to acknowledge the rest of the Organizing Committee. These people have put in a lot of work to make this all happen:

Local Arrangements: Greg Filip, Angel Saavedra, Amy Ramsey-Kroll and Dan Omdal.
Program Chair: Alan Kanaskie, with support from Bob Edmonds.
Secretary: Stefan Zeglen.
Web Master: Judy Adams.
Treasurers: Holly Kearns and John Schwandt.

And also thanks to Jim Hadfield, who helped organize yesterday's Lake Chelan boat tour.

On a personal note, I've got to say that this particular WIFDWC is particularly special to me, as it marks 30 years since my first WIFDWC, in Vernon, BC in 1981. Without getting overly sentimental, I'll just say that it's been a heckuva ride, and through WIFDWC, I've met a lot of people, who at various times in my career, have been great mentors, valuable colleagues, and most important, have been lifelong friends. For me, that has always been one of the best things about WIFDWC!

Pete Angwin, WIFDWC 1987- Nanaimo, B.C.

I've noticed from the registration totals that we have about 15 graduate students here. That is great! To you, I want to give a special welcome, and ask that you feel free to pick our brains, ask the tough questions, get involved, and most of all, have fun!

At this time, I'd like to take a moment to recognize the WIFDWC members that we've lost this year:

Dick Parmeter: Professor of Forest Pathology at UC Berkeley.

Dick Bingham: Researcher at the US Forest Service Forest Science Lab in Moscow, Idaho, and was a founding member of WIFDWC.

Keith Shea: Who among other things, was the head of the US Forest Service PNW Research Station in Corvallis, OR and the Associate Deputy Chief of the US Forest Service in Washington DC.

In: Zeglen, S. Comp. 2012. Proceedings of the 59th Annual Western International Forest Disease Work Conference; 2011 October 10-14; Leavenworth, WA.
[1]USDA Forest Service, Forest Health Protection, Northern California Shared Service Area Office, Redding, CA.

Peter Schutt: Professor of Forest Botany at the Ludwig-Maximilians University in Munich, Germany.

So please, a moment of silence for our distinguished colleagues…

Before we move on to introduce our first guest speaker, I want to point up that it's not too early to start thinking about next year's WIFDWC. Thanks to the work of Phil Cannon, 2012 Local Arrangements Chair, the dates and location have now been set. It will be at the Granlibakken Conference Center and Lodge at Lake Tahoe, Monday October 7 to Friday October 12, 1012. I encourage everyone to help with the organization of next year's WIFDWC!

KEYNOTE ADDRESS

Jim Hadfield[1]

I felt honored when Greg Filip asked if I would give the keynote address at this conference. I accepted. After a short time I began to fret over the obligation of having to be profound before this group —what critically significant messages can I pass on, what advice should I offer, what prognostications do I make? Holy Mackerel! What did I get myself into? Then I got to thinking, they probably asked me to be the keynote speaker because I am a local, therefore they would not have to pay me or even give me an expensive gift to induce me to accept their invitation to speak. So I relaxed somewhat and decided I would not need to be quite so profound after all. My major guidance is to stay within my allotted time.

In preparing for this keynote address I looked back at previous proceedings and found keynote talks have not consistently been given. I discovered Walt Thies seems to have given one of the few presentations specifically described as a WIFDWC keynote address. I decided that would be a good template to follow. As an elder Forest Service Forest Health Protection forest pathologist I have spent my career taking information developed by research forest pathologists and attempting to put it into operational practice. So I am following Walt's format with major departures. I have 3 topics to briefly cover, all related in some fashion to forest pathology and WIFDWC.

My history- According to Walt it is obligatory the keynoter provide some personal history, so here goes. I grew up in eastern Connecticut. My father was a forester for the Connecticut State Forestry Department. My mother graduated from the University of Connecticut in 1929, an accomplishment I find rather remarkable for that era. My father also owned and operated a charcoal manufacturing business in which I worked as a high school and college student. I got a BS in forestry from the University of Connecticut in 1965 and a MF in forest pathology from Duke University School of Forestry in 1967. The summer of 1966 I was a seasonal forest pathologist for the Forest Service surveying and supervising a field crew doing Scleroderris canker surveys on National Forests in the Lake States. My full-time appointment as a forest pathologist was 1967 in Amherst, Massachusetts where I covered all the New England states and New York and New Jersey. My first Forest Service vehicle was a 1963 Studebaker station wagon with a V-8 engine and standard transmission. It was a real hot rod. In 1969 the office was relocated to Portsmouth, NH. I got to work with Alex Shigo, Dave Houston, and Phil Wargo during that time. In 1971 I accepted a transfer and promotion to the Pacific Northwest Region in Portland, Oregon. Dave Johnson had arrived about one month before me. Dave Graham was the staff director. Walt Thies joined the staff in 1973. Over the years many forest pathologists joined this staff, including Don and Ellen Goheen, Greg Filip, Craig Schmitt, Paul Hessburg, Bob Harvey, Boris Tkacz, Sally Campbell, Susan Franckel, Alan Kanaskie and my apologies to others I have overlooked. We provided forest pathology advice to all National Forests and other federal and tribal lands in Washington and Oregon from Portland. I took a bit of a break from forest pathology from 1988 to 1993 when I more-or-less became a killer of western spruce budworms and Douglas-fir tussock moths. In 1994 I resumed being a forest pathologist when I moved to Wenatchee, WA where I am thoroughly entrenched.

In: Zeglen, S. Comp. 2012. Proceedings of the 59th Annual Western International Forest Disease Work Conference; 2011 October 10-14; Leavenworth, WA.
[1]Wenatchee Forest Insect & Disease Service Center, Okanogan-Wenatchee National Forest, Wenatchee, WA.

My service computation date for the U.S. Forest Service is August 20, 1966. My USFS forest pathology career spans 45 years, all in Forest Health Protection. I predate NEPA. I started working on *Fomes annosus, Fomes pini, Polyporus schweinitzii, Poria weirii*, and *Armillaria mellea*.

Reflections on Pacific Northwest forest pathology— When I was a forest pathologist in Amherst, MA and Portsmouth, NH I encountered many forest diseases but was frustrated that not much ever was done to manage them. When I moved to Portland, OR in 1971 I found an entirely different world of forest pathology. The trees and most pathogens were different from those I worked on in New England but what was really different was many forest diseases in the Pacific Northwest were recognized as serious economic disturbances and there was a willingness to actively manage them. Forestry and wood products industries were major economic forces. The result was there was a long history of forest pathology research in Oregon, Washington, Idaho, California, and British Columbia. Forest diseases have been investigated intensively for almost 100 years in the Pacific Northwest. There is a relatively rich base of technical information about many forest diseases in the Pacific Northwest. People I consider giants of forest pathology worked in the Pacific Northwest. James Weir, John Boyce, John Bier, Ray Foster, Toby Childs, Jim Kimmey, Lew Roth, and Bill Bloomberg to name a few. Forest pathologists know a lot about Pacific Northwest forest diseases. The Pacific Northwest, including British Columbia, is the greatest location on earth to be a forest pathologist.

The decline of forest pathology research in the USFS was described by Walt. Since his retirement there is only one person in the Pacific Northwest Research Station with the title of Forest Pathologist. I wish this was not the case. It is of interest to me that at least 34 employees of the PNW Research Station have the job title of Ecologist, including three who formerly had the job title of forest pathologist. I suspect when I got to Portland in 1971 the PNW Station probably had no one with the job title Ecologist. In contrast, the number of forest pathologists in the USFS Forest Health Protection program has expanded or has not declined in the last 10 years. The Pacific Northwest Region at the time I prepared this presentation had 9 forest pathologists. I believe the British Columbia Forest Service had no forest pathologists in 1971 and now has six. Kudos to our northern neighbors.

Forest pathology is alive, and I believe well, in the area covered by the Wenatchee Forest insect & Disease Service Center. There is a veritable cornucopia of diseased trees. We experience a steady demand for advice on forest diseases. Forest restoration is a major emphasis program on the Okanogan-Wenatchee National Forest and forest disease and insect assessments are key components of restoration activities. Indian Nation forest lands served by our service center are actively managed to prevent and reduce losses attributable to forest diseases. Forest health is a major public issue in the Pacific Northwest. Forest pathologists in the Pacific Northwest have a large amount of information available for many diseases, but there are still significant gaps in our knowledge and it has become increasingly difficult to get research underway to fill the voids. I doubt the USFS is going to be adding research positions with the job title forest pathologist and I am concerned about American universities maintaining forest pathologist faculty positions. I anticipate the number of forest pathologists in the FHP and State forestry organizations in the Pacific Northwest is going to be stable.

WIFDWC- This is a very important confederation, to borrow a term from Walt's keynote. In my opinion, it is important to the science and profession of forest pathology that WIFDWC continue. It is important for western North American forestry because forest diseases are major disturbance agents. WIFDWC members/ participants have been responsible for much of the forest disease knowledge base in western North America. The format and traditions of WIFDWC provide unparalleled opportunities to debate, challenge, interact, commiserate, and absorb from peers. The conferences foster exchange of information and opinions and provide opportunities for valuable face-to-face conversations with fellow forest pathologists. I have been to national American Phytopathological Society meetings and found the rigid meeting formats did not allow for the free-wheeling discussions that are commonplace and expected at WIFDWC. I contend that WIFDWC has served western North American

forestry well by providing a cadre of very knowledgeable, professional forest pathologists.

The Internet is a fantastic tool for forest pathologists. I am impressed at the amount of tree disease information one can get from Google and Wikipedia searches. I use the Internet all the time to find and get papers and reports, many 60 to 75 years old. The internet cannot come close to the value and experiences obtained from sitting down at WIFDWC with a fellow forest pathologist you have never met before and drinking beer or wine while discussing some obscure pathogen that only you and they care a whoot about. WIFDWC provides that opportunity. Take that opportunity at this conference. Let us make sure WIFDWC continues.

WIFDWC proceedings provide a wealth of information basically not available in publications such as the Canadian Journal of Forest Research, Forest Science, and Phytopathology because of the stringent statistical analyses and review requirements. There is a lot of value in the gray literature. I urge you to consider giving presentations on studies perhaps with less than rigorous experimental designs at these conferences and writing a paper for the proceedings if you are confident in your observations. The proceedings provide opportunities for opinion expression and reminiscence not available in most other forest pathology literature. Some of the most informative papers I have read were from a panel discussion of the use of antibiotics for control of white pine blister rust at the 1964 12th WIFDWC conference in Berkeley, CA. Please continue to produce conference proceedings.

I have a sporadic history of attending WIFDWC but I am an ardent supporter of the conference.

Almost every forest pathologist in North America is a government employee. My hat's off to the few who are in business for themselves. There is a significant cost to attend WIFDWC meetings, with most expenses being ultimately paid for with taxpayer dollars. As a supervisor of other forest pathologists, I have tried to be supportive of subordinates attending the WIFDWC conferences because they are rich opportunities for forest pathologists to improve themselves. But as a supervisor I also have a budgetary responsibility. I have been somewhat hesitant to send more than one pathologist to a WIFDWC conference due to costs and would rather send a subordinate to the conference rather than me attending and them not going. I encourage western North American forest pathologists to be involved in WIFDWC and attend conferences, even if you cannot go to everyone. WIFDWC offers the best opportunity for you to maintain and improve your professional skills.

We all know the United States federal government and many states are in serious financial trouble. Attendance at conferences is being very closely scrutinized and limits on travel are being imposed. My advice is to rate attendance to WIFDWC as high priority professional development and sell your supervisor on the value of participating. We need to make sure the taxpayers are getting a good value for their taxes when they pay for us to have WIFDWC. I look at the agenda for this conference and feel the taxpayers are getting are getting a great value.

Suggestions for attendees—First, for the retirees at this conference, Safeway has a good supply of Metamucil and talk to me after my presentation for where to go for wine tasting and good meals. My advice to students and folks with degrees who are seeking jobs as forest pathologists is to visit and talk with employed forest pathologists as they could be hiring in the future. Hang in there because there are going to be openings as us elders move on in one manner or other. The most common question posed to foresters is probably "What is wrong with my tree"? Who better to respond to that question than forest pathologists? Obviously, you need to conduct yourself with a high degree of decorum. For the currently gainfully employed forest pathologists, maintain, renew and expand your contacts with other forest pathologists at this conference.

Walt, in his 2007 keynote address had five points of advice. I have five.

1. Support WIFDWC, it is important to the success of forest pathology in western North America.

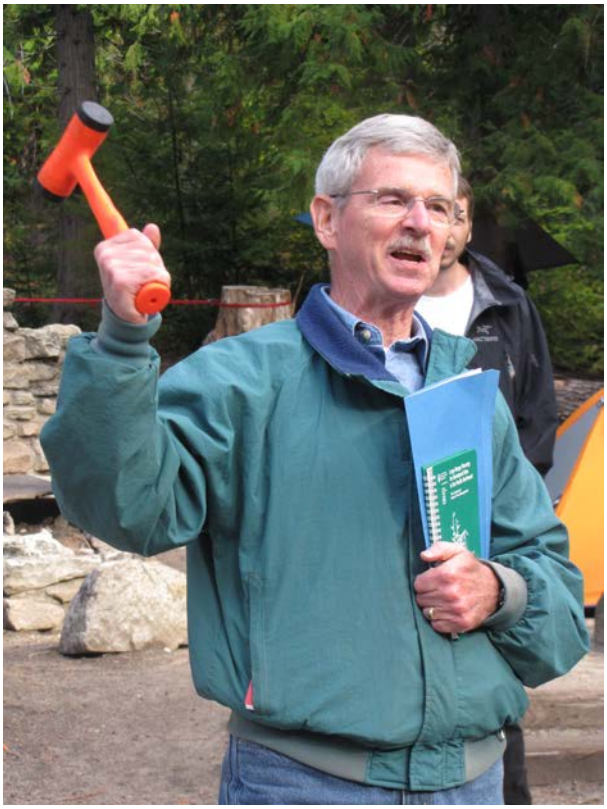
2. Give the taxpayers who are paying your salaries and expenses great value by doing your job to the best of your ability. We are in the business of providing advice and information on forest and tree diseases. In most instances this advice is provided to foresters. I have always felt my job as a forest pathologist is so much simpler and easier to do than the jobs of foresters I advise. I always try to ask myself is the information I am providing to this person(s) of real value to them? If not, what do I need to do to make it so? Provide the best information and advice possible to foresters, tree managers, natural resource managers, and anyone who asks. Tell people what they need to know about forest and tree diseases. Be thorough, be accurate. Know how to say "I do not know, but I will try to find out." And be sure to follow-up. Be true to the blend of science and art we call forest pathology.

3. Study the old reports, especially reports prepared by forest pathologists who actually worked out in the forests from the early 1900's through the 1970's before computers were used to simulate forests and forest disease effects. Look at historical reports-they are chock-full of valuable information.

4. Have fun in your job. Take time to smell the wildflowers, look at the mountains and wildlife, admire the huge trees some of us are privileged to work with, take pictures of mushrooms, breath in the fresh air, find humor in your work. We have some of the best jobs on the planet. We work in places where others pay lots of money to visit.

5. Marry well.

Thank you. Please have an informative and productive 59th Western International Forest Disease Work Conference.



2010 OUTSTANDING ACHIEVEMENT AWARD RECIPIENT PRESENTATION - PAUL HENNON

Forest Pathology at an Alaskan Outpost

I'm deeply honored and somewhat shocked to be recognized for this award. As a USFS employee, I'm accustomed to awards. Where else do you get an award for just showing up (we receive pins for every five years of employment)?

But *this* award is different, and I was stunned when Ellen Goheen called me a year ago with this news. Working in Alaska, I have not had very direct interactions with many of you, and we don't often collaborate on many projects. I assumed no one was paying much attention to my forest pathology exploits in Alaska. More importantly, I know there are other worthy forest pathologists in line for this award. I won't mention names, but I can think of certain WIFDWC members with more varied experiences and knowledge in forest pathology than I have: one with an extreme duality of skills in science and administration, one with rich experiences from industry, one with unbridled enthusiasm for forest pathology (Hail Beavers!), and many others who have tirelessly devoted their careers to applied forest pathology. One such applied pathologist has performed these duties for some 46 years with the passion of someone right out of graduate school. My main thought about this award is that because every forest pathologist at WIFDWC has a unique job with specific responsibilities and opportunities, comparing achievements among us is problematic.

It's been a year since Ellen called me about his award. I waited all year for some inspiration that would lead me to say something profound here today. But there was no epiphany … lightning did not strike. Today, I'll fall back and talk about the only topic that I'm qualified to discuss, forest pathology in Alaska.

Before I mention a few forest pathology issues, I'd like to provide a brief history of forest pathology in Alaska with a short list of pathologists who have worked there. In the early days, no pathologist was stationed in Alaska. Visitors, several of whom are mentioned here, generated knowledge. James Kimmey worked seasonally in coastal Alaska in the 1950s to produce an outstanding report of cull (stem decay fungi), still used today. Tommy Hinds visited Tom Laurent in interior Alaska and together they surveyed aspen diseases. Forty years later, these survey results provide our current understanding of aspen disease agents. Frank Hawksworth visited southeast Alaska where he made some important observations on hemlock dwarf mistletoe.

The first permanent forest pathologist in Alaska was Tom Laurent. He worked out of Juneau, initially with forest inventory and then Forest Pest Management. Tom participated in another regional cull study published in the 1970s, documented many tree diseases, and conducted a study to determine sulfur effects on trees around two pulp mills. Tom retired in 1984 and lives in Douglas, Alaska, where he continues to play practical jokes on unwitting friends. Terry Shaw arrived in Juneau in the 1970s as the first PNW research pathologist in the state. Terry was charged with evaluating the potential for diseases to impact productivity in the young growth stands that developed naturally after clear cut harvest of old-growth western hemlock and Sitka spruce. As such, Terry studied hemlock dwarf mistletoe spread, spruce mycorrhizae, root diseases, shoot blights, and stem fluting. Terry also initiated work on decline of yellow-cedar. As to the young growth stands, the overall general conclusion from the array of studies Terry conducted was that they were disgustingly healthy. He left Alaska for Colorado, but returned to Alaska several years later to lead efforts in incorporating science in forest planning.

I was the next to arrive in the early 1980s, originally as a seasonal biological technician, then as an OSU student studying cedar, and finally to work with Forest Health Protection and PNW. Keith Reynolds was a research forest pathologist in Anchorage for several years before moving to Oregon. His vacated position became a more applied job, filled by Lori Trummer. Lori had a productive career in Forest Health Protection with projects on tomentosus root disease (assisted by Kathy Lewis), birch stem decays, *Phytophthora* on alder, alder canker, and hazard trees. Lori retired several years ago and today lives in Gustavus, Alaska.

There are currently three forest pathologists in Alaska, all with the USFS. Lori Winton is in Anchorage; Robin Mulvey and I are both stationed in Juneau. There is no permanent university forest pathology program in Alaska. The State of Alaska has an entomology position, but has never supported a forest pathology position or program, even though the state is wealthy. Thus, there is no backup or redundancy for the three of us. We still rely on visiting forest pathologists to fill technical gaps. With similar ecosystems and close proximity, we have natural alliances with British Columbia and the Yukon. I have worked with Stefan Zeglen, Rona Sturrock, Alex Woods, and others in Canada. More collaboration between Alaska and Canada is needed, but the border between the two countries creates an administrative and travel barrier that we need to overcome.

The forests of coastal Alaska are unique in that they almost never experience fire. Without a frequent source of disturbance, stands and trees in these true temperate rain forests can reach old age. Harvests and the resulting young-growth forests are common in some locations, but there are enormous areas of pristine, old forest where natural processes, including tree diseases, are on full display. The coastal rain forests have been described as being compositionally simple but structurally complex. The structural complexity is in part due to these forests oozing with disease agents attacking old trees. This creates a wonderful setting for an ecological forest pathologist, or, for that matter, almost any forest scientist other than a fire specialist.

Once in Alaska in the early 1980s, my intuition was to focus my time on forest disease issues that were ecologically and economically important. There are other useful strategies, such as an attempt to catalog pathogenic species for a baseline of native pathogens to contrast with new introductions. My ecological-economic approach led me to investigate hemlock dwarf mistletoe, stem decays (heart rots), and a forest decline. Below are a few thoughts on each topic.

Outside of the Dwarf Mistletoe Committee, dwarf mistletoes don't seem to get the same respect among western forest pathologists as the fungal diseases. However, whole careers were devoted to the one dwarf mistletoe species that occurs on hemlock found along the Pacific Coast up into Alaska. Pathologists in British Columbia worked out elaborate spread models, and when coupled with tree impact information, give us a surprisingly complete view of disease behavior of hemlock dwarf mistletoe. Terry Shaw supplemented this with elegant research that contrasted vertical intensification of the parasite with height growth of trees as a mechanism by which the disease dies out of productive young managed forests. We now know that hemlock dwarf mistletoe can be manipulated to any desirable disease level by silvicultural practices using different harvest scenarios, including partial harvests of old-growth stands. Lori Trummer's graduate thesis project provided a century-long time perspective of spread and intensification. I wonder if any other forest disease can be managed with similar precision and certainty.

The stem decays, or heart rots, seem to be an archaic forest pathology issue to many. Several pathologists have even asked if they are true tree diseases. Stem decays were important topics in the Pacific Northwest in the early days of Boyce, Bier, Foster, Buckland, Kimmey, and others. But as pathologists turned their attention to issues affecting young-growth managed forests, these diseases of older forests took a back shelf. Perhaps interest will return as the managed forests of the Pacific Northwest age, or if managers attempt to speed stand development toward old-growth characteristics by encouraging the stem decays. Meanwhile, the stem decays have always been important in coastal Alaska where old-growth forests

abound and these diseases create wildlife habitat, alter carbon budgets, contribute to hazard trees, and operate as disturbance agents. In Alaska we have found that tree death through bole collapse, where heart rot weakens the structural integrity of bole wood, is a leading form of gap formation and the perpetuation of the old-growth condition. Without question, the stem decays qualify as pathogens that cause tree disease.

Forest declines are a collection of often-misunderstood diseases. I define them as being widespread, occurring in multiple years, and having either a complex or unresolved etiology. This last feature creates wiggle room, allowing forest pathologists to place difficult disease situations in a bin (the forest declines) while they are being researched. How do we solve them? In the case of our investigation on the cause of yellow-cedar decline, we made good progress with a team of pathologists, but close collaboration with other scientists in the fields of soils, hydrology, ecophysiology, dendrochronology, climatology, and spatial ecology was needed to eventually solve the problem. Meaningful policy and management guidance was only possible once the cause was determined. I have proposed that more frequent widespread tree mortalities will appear on landscapes as forests are stressed by a rapidly changing climate. I believe that there is no substitute for pathologists (and entomologists) to investigate every one of these emerging forest problems. Evaluations by pathologists are necessary to distinguish largely biotic- from abiotic-caused mortalities, and to place any alleged role of climate into proper perspective. No one else is qualified to make these determinations. Also, it is important for forest pathology to receive credit for solutions, where they occur, and in some cases forest pathology can "own" these issues (e.g., pole blight, sudden aspen death, and yellow-cedar decline) because of our initial and substantial involvement.

As a fairly long-term member of WIFDWC, I want to make a few observations about the health of our forest pathology institutions. One of the benefits of taking over Tom Laurent's forest pathology position in Juneau was that I inherited all of his WIFDWC proceedings dating back to 1961. Great reading! The 1982 proceedings caught my eye: Everett Hansen led a panel on the future of forest pathology. Earl Nelson discussed the future of forest pathology research but could not predict the almost total eventual collapse of PNW pathology research. Roy Whitney and Rich Hunt gave an entertaining narrative on Canada. I was particularly interested in Bob Loomis' gloomy prediction of the future of forest pest management (later to be called forest health protection or FHP) in the US.

How did their predictions fare? Forest pathology research tanked, at least in the PNW Station. Positions were lost through funding reductions, and some research pathologists transitioned to other disciplines, as broader ecosystem research was favored. One could argue that ecological research in the forests of the Pacific Northwest will be incomplete without the input of forest pathology. I would make that case for coastal Alaska. Certainly with climate change stresses on forests, arising forest problems will need engagement from research pathology.

I have already mentioned that Alaska provides no training for forest pathology graduate students; therefore, I'm particularly interested in the fate of university forest pathology programs elsewhere in western US and Canada. I don't have first-hand knowledge of their health, but I fear they are diminishing. Will retiring professors be replaced to maintain these programs? Even if so, who among us is broad and deep enough to succeed them? We probably take our current cohort of outstanding professors for granted. They have trained a group of fine graduate students and working young forest pathologists (e.g., Lori Winton and Robin Mulvey in Alaska). Our near-term future is bright, but long-term forest pathology is not sustainable without strong university programs producing students.

Finally, I'd like to offer a few words about forest health protection, FHP. The future of FHP is important because this is where a good part of today's US forest pathology jobs and funding reside, contrary to Loomis' pessimistic prediction. I was fortunate to work alongside FHP entomologist Andy Eglitis in my early Juneau years. Andy is a role model for technical assistance, what I consider to be the

bread and butter for regional FHP programs. How else is a graduate student trained in research to learn such things? The Alaska FHP program was supervised by a series of entomologists out of Anchorage at that time. They had some appreciation for pathology and left me alone to develop a forest pathology program aimed at issues in coastal Alaska. The orientation of the Alaska FHP program began to shift in 1992, nearly 20 years ago, when a succession of leaders came from a background in forest management rather than moving up from within FHP. Serving national mandates, upward reporting, and promoting the program took precedence over providing technical assistance to solve forest health problems for local clients and partners. I hope an FHP entomologist or pathologist will eventually move into a leadership position to restore the original emphasis of FHP in Alaska.

I'll close by saying how lucky I am to have found this field and WIFDWC. My supportive wife and family allowed me to pursue my life's passion, forest pathology. I had more good fortune from the training and experiences in Everett Hansen's program at OSU. And, my luck continued as I moved to Alaska to work with Terry Shaw, Andy Eglitis, Tom Laurent, and now new partners in one of the world's great forests.

2011 OUTSTANDING ACHIEVEMENT AWARD RECIPIENTS ELLEN GOHEEN AND SUSAN FRANKEL

Pete Angwin, Harry Kope, and Bill Jacobi
2011 Outstanding Achievement Award Committee

The Outstanding Achievement Award is the highest honor that WIFDWC can give to one of its members. According to the WIFDWC bylaws, the award recognizes "an individual that has, in the opinion of the membership, contributed significantly to the field of forest pathology in western North America. This year, the selection was very easy. In response to the call for nominations, several phone calls; letters and emails were received regarding Ellen Goheen and Susan Frankel. In addition, everyone said that it was impossible to recognize the achievements of one without recognizing the achievements of the other. The Committee agreed, and decided that the most appropriate thing to do would be to give the award to both.

While Ellen and Susan's most notable contributions have been in response to the introduction of *Phytophthora ramorum* in western North America, both have a long history of significant accomplishment in field of forest pathology. The nomination letters that were received point up several aspects of this accomplishment:

"Ellen Goheen and Susan Frankel share the glory of the biggest disease management program the west has seen since the heyday of blister rust. They have very different operating styles but they share traits of success: In their own ways they are effective and dedicated facilitators and team builders. They work hard, and exert leadership to get the job done. They creatively take advantage of their positions to provide both emotional and material support for outreach and research activities. They truly represent Outstanding Achievement in the pathological west." *Everett Hansen, May 25, 2011*

"…More recently, both Susan and Ellen have played major roles in the Forest Service program to understand and try to control *Phytophthora ramorum*. Through leading by example and taking the time to care, they have both encouraged and mentored many younger pathologists, thus further enriching our profession. It's time their contributions receive this appropriate recognition." *Terry Shaw, May 31, 2011*

"…These two Forest Service pathologists consistently have demonstrated effective leadership in forest pathology in the western U.S. Their critical roles and their exemplary performance in the management of sudden oak death in Oregon and California highlight their contribution to our professional community. Both deserve recognition, but their joint impact suggests that they be recognized together. It will not be unnoticed that if selected for the award they would be the first women to receive the honor; it would be difficult to put one before the other in that respect." *Alan Kanaskie, June 10, 2011*

"…The key thread that runs through this joint nomination for Ellen and Susan is their ability to pull people together. I have been consistently been impressed with their continued leadership abilities in the face of a high profile and hot button environmental issue. They have certainly given the field to forest pathology a good name and they are certainly deserving of this award." *Dave Rizzo, June 13, 2011*

"In the long run, I think Ellen's biggest contribution to forest pathology and forest health will be her desire to educate people and help them put that education to work on the ground. And she is very good at it. I am always amazed when we do training sessions at how well she connects with audiences from a wide range of backgrounds, generates enthusiasm for forest pathology, and relates the information to their needs. She provides training not only for federal agencies but also teaches classes regularly for the local Master Gardeners and the OSU Extension Tree Schools. I can directly credit Ellen's enthusiasm for my interest in pursuing forest pathology." *Katy Mallams, May 13, 2011.*

It is worth noting that not only are Ellen and Susan the first women to receive the WIFDWC Outstanding Achievement award, they are also among the first to receive both the WIFDWC Outstanding Achievement Award and the old WIFWDC Social Achievement Award (SAA to Ellen in 1989 and to Susan in 1995). Although the Social Achievement Award recognized a very different (and some have said, dubious) kind of distinction, Bob Gilbertson is the only other WIFDWC member to receive both awards (SAA in 1983 sand OAA in 2001).

PANEL: MONITORING ON THE MARGINS

MONITORING ON THE MARGINS PROGRAM PURPOSE AND DESCRIPTION

Eric Smith[1] and Borys Tkacz[2]

Monitoring on the Margins (MoM) is a Forest Health Monitoring (FHM) initiative for an integrated, enhanced monitoring program for critical ecosystems in areas threatened by insects, disease, and climate change. Rob Mangold, Director of Forest Health Protection, charged the Forest Health Monitoring Management Team to develop a template adaptable to any species across the country and a program initiative for monitoring at risk tree species. Data collected under this template will tier to the "National Roadmap for Responding to Climate Change" (http://www.fs.fed.us/climate change/pdf/roadmap.pdf), which proposes targeted monitoring based on vulnerabilities.

The MoM program initiative was developed as a template for any species of concern, using high elevation, five needle pines (FNP) as the initial focus for a pilot of the program initiative. These pines include whitebark, foxtail, and bristlecone, and, where in high elevation conditions, limber and southwestern white pine. For the last century, white pine blister rust (WPBR) has spread from its introduction site in the Pacific Northwest, initially devastating western white pine and sugar pine. However, only in the last 20 years has the spread and impact of WPBR on high elevation, FNP caused wide concern for the viability of the species. Recent mountain pine beetle epidemics in these pines in the interior West have expanded the concern. Long term climate warming projections also raise concerns that the habitats of these pine will greatly decrease. Warmer recent weather has also been implicated in the mountain pine beetle epidemics.

In: Zeglen, S. Comp. 2012. Proceedings of the 59th Annual Western International Forest Disease Work Conference; 2011 October 10-14; Leavenworth, WA.
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A core team[a] including FHM Coordinators from the west and an extended team[b] including Forest Health Protection (FHP) specialists from the western regions, developed the MoM program initiative following the approach outlined in "A Model for Monitoring and Conserving Forest Trees Threatened by Climate Change and Invasive Species", (Smith and others 2009). This approach lays out an eight-step process as follows:

1- Creation of a resource distribution map of trees and ecosystems (document the resource).

2- Creation of a multi-threat disturbance map (document current and imminent threats).

3- Combine the resource and threats maps to determine resource conditions at risk (margins).

4- Map existing spatial genetic knowledge for species of interest.

5- Perform regional level assessments to determine high priority monitoring conditions.

6- Determine existing plot coverage and need for (additional) monitoring (what and how).

7- Install, maintain, and analyze monitoring results.

[a]Core Team: Rob Cruz (R1/4), Jeri Lyn Harris (R2), John Anhold (R3), Lisa Fischer (R5), Bruce Hostetler (retired) and Alison Nelson (R6), Dale Starkey (R8), Jim Steinman (NA), Eric Smith (WO), and Borys Tkacz (WO).

[b]Extended Team: Blakey Lockman (R1), Holly Kearns (R1), Kelly Burns (R2), Jim Hoffman (R4),), John Guyon (R4), Matthew Bokach (R5), Sheri Smith (R5), and Kristen Chadwick (R6).

8- Use threat analysis and ongoing monitoring results to direct genetic conservation management activities.

Steps 1 through 6 involve information gathering and analyses of current conditions and existing monitoring, step 7 includes implementation of an enhanced monitoring system, and step 8 is the use of the monitoring information for management and conservation activities. This proposal is divided into two phases; phase I addresses steps 1 through 6 and phase II addresses step 7. Phase I tasks addressed in this proposal for high elevation FNP:

A. Create a GIS framework and an associated relational database to contain and display the information and support the analyses of steps 1 through 6 of the process.

B. Collect and review information needed for analyses, and use it to populate the GIS and relational databases.

C. Analyze data and other pre-existing studies to determine the "margins" for the populations being considered, and their priorities for monitoring attention.

D. Assess current monitoring efforts, consideration of alternatives for additional monitoring, and creation of a monitoring plan showing how new and existing sources will be integrated and analyzed.

PHASE I

Program Task A: Creation of an Arc-GIS framework and an associated relational database to contain and display information on the species of concern, using high elevation FNP as a pilot group of species.

Status: In progress; Task Lead: Eric Smith, FHTET

There is a need for an authoritative database for the resource, threat, and monitoring information on high elevation FNP. The concept of this database is a seamless extension or link to existing FHM databases and information systems (e.g. Aerial Detection Survey (ADS), National Insect and Disease Risk Map (NIDRM), Whitebark and Limber pine Information System (WLIS)) and to be an extension of other FS corporate databases, where the combined data and information about resource threats and monitoring can be stored, displayed, and analyzed. This MoM database will provide an integrated linkage to a standard set of data sources, and enhance consistency with information being used in other Forest Health Protection (FHP projects and reports.

The database approach will focus on storing only plot or polygon location data and a minimum of additional fields (such as data source, tree species, and measurement year) and primarily rely on previously developed systems (primary data sources when available, or compilations such as WLIS) to organize and maintain the actual full data sets. This database will have parallels with the NIDRM datasets. Data development may need special modeling efforts in NIDRM because of the relative rarity of high elevation FNP in many areas. These species are on the margins for inventory by the Forest Inventory and Analysis (FIA) program.

Program Task B: Collect, survey and synthesize existing sources of data and information on the species of concern, using high elevation FNP in this pilot. Identify spatial information (databases) and evaluate data collection methods (metadata) for inclusion into a geospatial portal/database.

Status: In progress; Task Lead: Matthew Bokach, R5

Steps to determine the currency, survey intensity, and distribution of tree and disturbance information across the populations of high elevation FNP include "surveying" existing databases and specialists within the FS and in partner agencies and organizations across the west. Phone and email surveys as well as investigation of the literature (Task C) will be employed to identify data sources for incorporation into Task A. While many known data sets are available such as FIA and WLIS, lesser known datasets such as CAIDA and independent plot data and information are important to identify to create the most complete synthesis and feed into a database for further query and analysis.

Program Task C: Produce a systematic review of the literature regarding high-elevation FNP/WPBR management-oriented plans, research/monitoring papers and proposals concerning their geographic scope, managerial scope, stated information needs,

and analyses. This will aid in identifying information gaps and the kind of information found to be useful in developing these plans. Use existing plans and reports and analysis of data sources to prioritize areas of greatest interest and concern for high elevation FNP populations.

Status: In progress; Task Lead: Kristen Chadwick, R6

A great deal of information has already been gathered and published for some of the high-elevation FNP species across the West in various formats or publications and by various entities. There is a need for a systematic review of the information available in management-oriented documents for high-elevation FNP species across western North America. This would include reports and scientific papers that would be useful in developing a management plan such as management guides, species status reports, genetic conservation papers, etc. There are substantial numbers of documents regarding management and status of whitebark pine, but few documents dealing with other high-elevation FNP species including limber pine, southwestern white pine, foxtail pine, Rocky Mountain bristlecone pine, and Great Basin bristlecone pine.

This synthesis will look at the geographic areas covered, sources of information used, types of analyses that were implemented, and management recommendations. The individual performing this task will identify data sources used in the documents. The data source can be many and varied. For example, in the whitebark pine restoration strategy document for the Pacific Northwest Region (Aubry et al. 2008), the data sets used included: FIA plots, CVS plots, area ecology plots, a North Cascades grizzly bear habitat study, Pacific Northwest Albicaulis Project survey and reconnaissance data points, reconnaissance and survey points from other sources, all whitebark pine cone collection sites in the Region, and verified sites from other reliable sources. The individual working on Task C will need to collaborate closely with knowledgeable people from each of the Forest Service Regions, numerous external partners, and with the person(s) doing Task B, who will be drilling down into the data sources to get more specific geographic locations, parameter data, etc.

Program Task D: (1) Identify margins where current information gaps occur, particularly:

(i) Places where significant disturbances have occurred without adequate subsequent plot sampling or other monitoring. Work from Tasks B and C will be used to identify these gaps west wide by Regional specialists.

(ii) Places where genetically distinct populations may have received little sampling or study. Tasks B and C will form the basis for this evaluation however Regional seed bank maps and related data sources will also be reviewed.

(iii) Places where climate change (or weather variation) or intensification of WPBR is predicted to produce increased disturbance (MPB, WPBR, fire). Forest Service approved climate change and global circulation models (Hadley, CSIRO and CGCM) will be used for these scenarios to determine information gaps.

(2) Produce and evaluate a common set of monitoring techniques which might be employed to fill in the existing gaps in different areas and to efficiently provide adequate monitoring in priority areas into the future. Techniques appropriate across the high elevation FNP range will vary, depending on past and current monitoring efforts, existing knowledge concerning high elevation FNP distribution and abundance, and the likelihood of disturbance.

Some techniques to consider might be:

- Intensified (in time or space) FIA (type) plots.
- Application of remote sensing techniques.
- Establish priority areas for ADS coverage.
- Special HIGH-5 PINE transects

Status: Planned for 2012; Task Lead: TBA

PHASE II

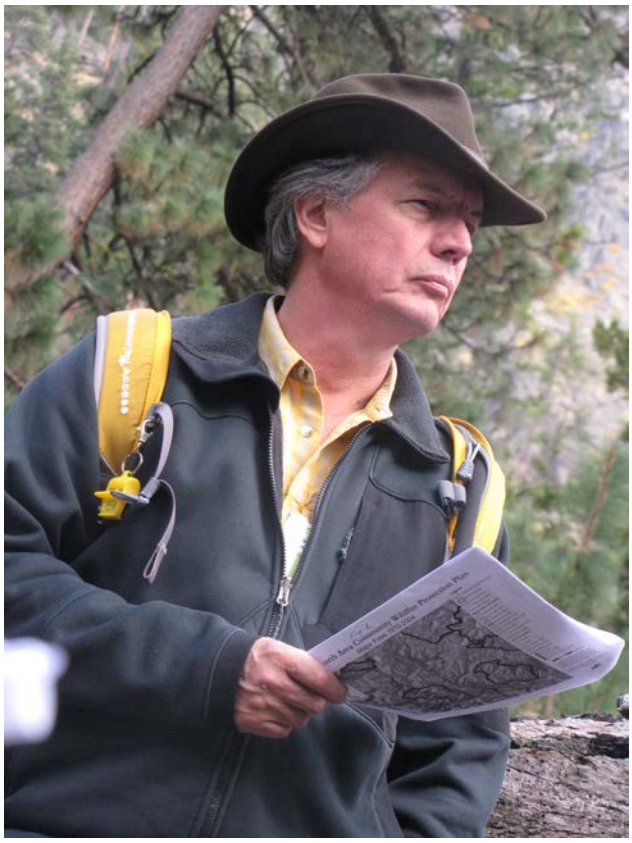
Program Task E: Implement a monitoring plan which includes appropriate supplemental data collection and analysis.

Status: Planned for 2012 and beyond; Task Lead: TBA
Using the information gathered in Phase I (tasks A through D), the Core MoM Team, along with specialists knowledgeable about white pine ecosystems, will design a MoM pilot project to implement techniques for monitoring. The pilot project will evaluate the utility of a corporate database, whether it is a web portal or some other database format, and implement standard and consistent data collection protocols west-wide. West-wide core protocols will be developed with regional add-ons if needed. In each FHM Region, a few areas will be identified that have been determined to be on the "margin" to gather monitoring data. Ground-based data and information will be collected from permanent sample plots or transects as well as information using one or more remote sensing techniques. This will allow for the evaluation between techniques and identify which techniques are better suited for specific parameters. In addition, costs of each sampling technique will be evaluated. Plans and/or protocols developed and tested will also be evaluated for their portability, applicability, and adaptability for monitoring other host species on the margins.

REFERENCES

Aubry C., Goheen D., Shoal R., Ohlson T., Lorenz T., Bower A., Mehmel C., Sniezko R.A. 2008. Whitebark pine restoration strategy for the Pacific Northwest 2009-2013: executive summary. USDA Forest Service, Pacific Northwest Region, Portland, OR. Accessed 2/29/12 at http://www.fs.fed.us/r6/genetics/publications/detail/pub802.

Smith, E., Mangold, R., Tkacz, B., Sapio, F. 2009. A model for monitoring and conserving forest trees threatened by climate change and invasive species. XIII World Forestry Congress. Buenos Aires, Argentina. 18-23 October 2009.



WHITEBARK AND LIMBER PINE INFORMATION SYSTEM AND ASSESSING HIGH ELEVATION FIVE-NEEDLE PINES

Blakey Lockman[1] and Kristen Chadwick[2]

The first task for this project involves collating existing data, specifically plot level data for these high elevation 5-needle pines. We need to display what we know before we can figure out what we don't know. The need to gather and collate data on high elevation 5-needle pines was actually identified and started in 2003, with the birth of WLIS- the whitebark and limber pines information system. WLIS is now being expanded and updated and is evolving into a new generation WLIS- a new name has yet been chosen.

The original WLIS was initiated in 2003; it was officially released in 2006 (Lockman and DeNitto 2010). WLIS housed plot level data for whitebark and limber pines only. The current WLIS has an interface system for inputting plot level data with a self-validating system which ensures that out of range values are not entered into the underlying Access database. It has an interactive GIS mapping application and also a query builder for querying the data contained in WLIS. WLIS was installed on, and downloadable from, Region One Forest Health Protection's website. Although it has proved to be an extremely useful tool, it was very difficult to update.

The new generation WLIS is being updated and will be internet accessible and easily updated by the user. It will also be expanded to include not just whitebark and limber pines, but also both bristlecone pines, foxtail pine and southwestern white pine.

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This effort to update and expand WLIS was initiated in 2010, and was quickly included under the umbrella of the Monitoring on the Margins (MoM) effort. Plot data from the additional species has been collated and formatted during 2011 by John Popp of US Forest Service, Rocky Mountain Research Station, Fort Collins, Colorado. The actual structure for the new generation WLIS will be completed in 2012 by US Forest Service, Forest Health Technology Enterprise Team (FHTET) out of Fort Collins, Colorado. Building the new structure for WLIS is being funded through US Forest Service Special Technology Development Program funding.

Although the structure for the new WLIS is not complete, the plot locations have been mapped by Matt Bokach, US Forest Service, Forest Health Protection, Region 5, through other GIS applications for this presentation, as well as for the completion of the next steps in the MoM pilot project.

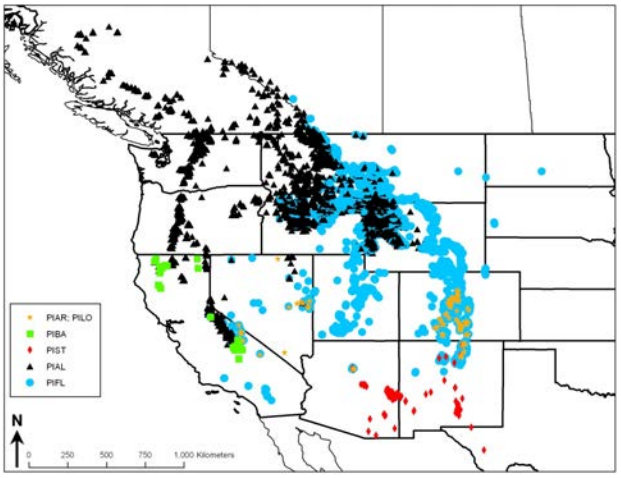

Figure 1: Plot locations of all high elevation five-needled pines contained within the new WLIS.

A request for data for the new WLIS was sent out this past winter. Any data not shared by the end of December 2011, will have to be entered by the plot stewards into the new WLIS when it becomes

available online. Matthew Bokach has also been creating layers from various raster datasets from around the west. This effort has been coordinated through FHTET. Examples of these raster datasets include seed zones, various management areas, and Research Natural Areas.

The MoM effort is now at a point where we can display what we know and determine what we don't know. Kristen Chadwick took on the task of doing a systematic review of the data, review of the literature, and a review of existing strategic plans. The following is a brief synopsis of this review.

The grouping of these six species is quite diverse, ranging from limber pine, which has the widest range of all of the 5-needle pines, to foxtail pine, which is limited to California only.

There are many threats faced by these species, starting with the exotic white pine blister rust. Regarding blister rust, we need to know where it is currently established in these high elevation pines, where it has recently been found, and where is the margin of invasion.

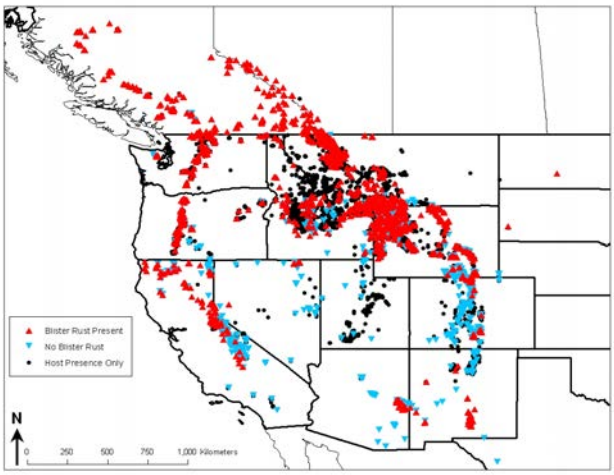

Figure 2: Plots in the new WLIS with known blister rust for all high elevation five-needle pines.

Following is a brief synopsis of what is known regarding white pine blister rust, species by species, and some interesting observations for each. In whitebark pine, the incidence of blister rust varies from high in the northern Rocky Mountains, and lessens as you go south and east. It is higher on western slopes in the Sierras and Cascades, and less on the eastern slopes.

In limber pine, blister rust ranges from being so severe that it threatens viability of some populations in Alberta, but is absent from some plot locations in southern California. It is also present in numerous disjunct populations.

Great Basin bristlecone pine grows on very dry exposed slopes, in colder climates with a range of precipitation levels from 12 inches/year in the White Mountains to 24 inches/year in the Nevada location. No blister rust has been found to date in Great Basin bristlecone pine.

Rocky Mountain Bristlecone pine also exists on dry exposed slopes with extremely variable precipitation, from as little as 16 inches on Pike's Peak to over 59 inches in the San Juan range of Colorado. Blister rust was first reported in RM bristlecone pine in 2002 in Colorado- with a 30% incidence near Mosca pass, and very minimal infection levels in adjacent upland slopes.

There are 2 allopatric subspecies of foxtail pine, with very different climates. Southern foxtail climate tends to be cold in the winter and warm and dry in the summer. The annual precipitation ranges from 20 to 30 inches. Blister rust has not been observed in this population. Northern foxtail climate is influenced by the Pacific Ocean and annual precipitation ranges from 49 to 60 inches. Blister rust commonly occurs in this northern population.

In southwestern white pine, there is a high incidence of blister rust in the Sacramento Mountains in New Mexico, but has not been found in Arizona. There is no known incidence of white pine blister rust across the border in Mexico- but there are Mexican 5-needle pines at risk.

Moving onto another currently large threat to 5-needle pines is mountain pine beetle (MPB; *Dendroctonus ponderosae*). In order to assess the threat from mountain beetle, we need to know: where climate has changed the life cycle of the beetle; the areas where MPB has caused extensive mortality; where MPB outbreaks are occurring outside the historic range of variability; and the areas at risk but not currently experiencing outbreaks.

Although the current MPB outbreak is alarming, there is a history of outbreaks in whitebark pine- mainly documented in the Northern Rockies. Through dendrochronology work, there is evidence that outbreaks occurred in the 18th and 19th century. More recent is a well-documented outbreak which occurred in the 1930's, leaving behind the famous high elevation ghost forests. The 1930's outbreak apparently started in whitebark in some locations, and the presence of MPB in whitebark pine was considered a threat to the existence of lodgepole pine. Attitudes have changed tremendously since then. Even more recent is the outbreak in the Interior northwest in the 1970's and 1980's. The current outbreak is range-wide, but MPB seems to be behaving differently in different locales.

Plot level data is not a good technique for monitoring MPB. The MoM effort is currently using aerial detection surveys to monitor MPB, which is problematic, but is the best data we currently have for monitoring MPB in these high elevation species.

In Montana, MPB is widespread in whitebark pine, and appears to be expanding throughout, as seen in aerial detection maps. As stated before, MPB is widespread, but seems to be behaving very differently in different locales. Very little is known about the behavior of MPB in the Sierra Nevada. Although there is no lack of host species, aerial detection surveys of high elevation pines in the Sierra Nevada show a spotty pattern of MPB. It doesn't seem to be expanding throughout, like in Montana.

Other threats include changes in fire cycle due to fire suppression, which allows for the buildup of species that readily outcompete these high elevation species.

Another threat is climate change and its impacts on the establishment of regeneration and the overall movement of alpine species. The impact of climate change on WPBR risk is unknown- in some areas, climate changes may lead to an increase in the probability of infection. Climate change may be altering the life cycle of MPB. Climate change will likely have an impact on fuel loading, which then leads to changes in fire severity. Climate change may also be impacting other high elevation species, such as the movement of balsam wooley adelgid, which impacts subalpine fir, and thus whitebark pine.

The impacts of climate change are probably way more complex then we realize. The simplistic view is that as temperatures warm up, species will move up in elevation, with alpine species having nowhere to go but "to heaven"- but there is evidence indicating that moving up in elevation is not always the response of these species.

There is evidence that "simple shifts in elevation of treeline and of plant species and communities are incomplete descriptions of subalpine response to climate" (Millar 2006). Connie Millar, USDA Forest Service, Pacific Southwest Research Station, Albany & Lee Vining, California, has looked at species shifts in the Sierra Nevada, and alpine species often respond to warming trends by moving down into protected areas in drainages and ravines- indicating the movement of species in response to climate change is very complex.

Some changes happen so quickly, like with mountain pine beetle, that genetic resources are lost before they can be conserved. Knowing where we need to conserve genetics of these species before they are lost is critical.

Known current locations of seed and cone collections from these high elevation pines- as well as locations of collections for the WO gene conservation effort have been mapped. Not all of these collections are in the long-term storage facility in Fort Collins. Gaps in our efforts towards gene conservation need to be identified and remedied.

Other identified gaps and limitations include using Forest Inventory and Analysis (FIA) plots for monitoring these species. The standard FIA plot system is problematic in that populations may not be adequately sampled or missed altogether within the FIA grid system. Also, FIA data collection is not set up to accurately record the presence of white pine blister rust.

A knowledge gap already mentioned is the lack of documentation regarding mountain pine beetle outbreaks in the Sierra Nevada. We don't know what triggers MPB to attack other hosts in the same area-

such as large lodgepole pine in Yosemite- while not attacking the high elevation 5-needled pines. There is a lack of information on historic MPB occurrence, ecological role of MPB, and the current risk for MPB in the Sierra Nevada. Current risk should be assessed and tied into the National Risk Map.

There is very little information on regeneration for these species. There is a paucity of information on how much regeneration currently exists. Strategies for surveying and monitoring regeneration are needed. There exists a large gap in our knowledge regarding regeneration of these high elevation pines. A similar gap exists in understanding stand development patterns outside of the Greater Yellowstone Ecosystem and whitebark pine systems. There is a real need for information on the disturbance dynamics in foxtail, both bristlecones, southwestern white, and some areas of limber pines. As mentioned earlier, another large gap in our knowledge is the white pine blister rust risk to the 5-needle pines in Mexico and how that might affect the 5-needle pines in the southwest.

Following is a collection of few other items we need to be thinking about as we move forward with developing MoM strategy for these species:

- We don't know if the current restoration activities will be successful, such as planting in high hazard sites. We need to determine where we should be focusing our restoration efforts.
- We need good information regarding the existence of these high elevation species in mixed stands and what role those populations may have in the viability of these species.
- There is very little information on the silvics of these species. Whitebark pine can release after 200 years, but what can be said about the other species?

Kristen has spent time reading and comparing existing Restoration Strategies, but her review of these strategies will be written up at a later date. For now, she is comparing the following key strategies:

- WBP Range wide restoration strategy (US and Canada) (Keane et al. *In Draft*).
- Greater Yellowstone Area Whitebark Pine Strategy May 2011—Interagency.
- USFS Region 6 Strategy for NFS Lands 2009-2013 Aubry et al. 2008.
- White Pines, Blister Rust, and Management in the Southwest (Conklin et al. 2009).
- Schoettle and Sniezko (2007) Other Species (Schwandt et al. 2010).

The next phase of MoM is to develop and initiate a pilot monitoring program in 2012.

Steps in this next phase include:

- develop monitoring techniques and a monitoring plan
- determine where we need to focus our monitoring efforts
- continuously update MPB activity during this outbreak
- work on risk assessments for WPBR and MPB and plan accordingly
- move forward with including climate models in our assessment
- include new data as it becomes available

REFERENCES

Aubry, C., Goheen, D., Shoal, R., Ohlson, T., Lorenz, T. Bower, A., Mehmel, C. Sniexko, R.A. 2008. Whitebark Pine Restoration Strategy for the Pacific Northwest 2009- 2013: exuctive summary. USDA Forest Service, Pacific Northwest Region, Portland, OR. 12 p. Accessed 2/29/12 at http://www.fs.fed.us/r6/genetics/publications/detail/pub802.

Conklin, D.A., Fairweather, M.L., Ryerson, D.E., Geils, B.W., Vogler, D.R. 2009. White pines, blister rust, and management in the Southwest. USDA Forest Service, Southwestern Region, R3-FH-09-01. 16p. Accessed 2/29/12 at http://www.fs.usda.gov/Internet/ FSE_DOCUMENTS/stelprdb5238475.pdf

Greater Yellowstone Coordinating Committee, Whitebark Pine Subcommittee. 2011. Whitebark Pine Strategy for the Greater Yellowstone Area. 41p. Accessed 2/29/12 at: http://fedgycc.org/documents/WBPStrategyFINAL5.31.11.pdf.

Keane, R.E., Mckinney, S., Jenkins, M., Perkins, D. Reinhart, D., Ryan, C., Tomback, D. In Draft. A range-wide restoration strategy for whitebark pine forests.

Lockman, I. B., Denitto, G. 2007. WLIS: The Whitebark-Limber Pine Information System and What It Can Do for You. USDA Forest Service R6-NR-FHP-2007-01. 146 p.

Millar, C. 2006.Complex responses of high-elevation forests in the Sierra Nevada to climate change. Presentation at the Third Annual Climate Change Research Conference, Sept. 13-15, 2006. Sacramento, California. Accessed 2/29/12 at http://www.climatechange.ca.gov/events/2006_conference/presentations/2006-09-15/2006-09-15_MILLAR.PDF

Schoettle, A.W., Sniezko, R.A. 2007. Proactive intervention to sustain high-elevation pine ecosystems threatened by white pine blister rust. J. Forest Research. 12:327-336.

Schwandt, J.W., Lockman, I.B., Kliejunas, J.T., Muir, J.A. 2010. Current health issues and management strategies for white pines in the western United States and Canada. Forest Pathology. 40:226-250.

PANEL: SYNTHESIS OF STUMP REMOVAL TRIALS IN THE NORTHERN HEMISPHERE

IMPACT OF STUMP REMOVAL ON ROOT ROT INCIDENCE AND DIAMETER GROWTH IN *PICEA ABIES*: ASPECTS OF ROOT ROT AND FUNGAL DIVERSITY

Rimvys Vasaitis[1], Natalija Arhipova[1], and Jan Stenlid[1]

Tree stumps are expected to be increasingly used for energy production in Fennoscandia, thus environmental consequences of stump removal from forest land must be assessed. Data from available trials (Vasaitis et al., 2008) demonstrate that stump removal from clear-felled forest areas in most cases results in, a) reduction of root rot in the next forest generation, b) improved seedling establishment, and c) increased tree growth and stand productivity. Observed disturbances caused to a site by de-stumping operations are normally acceptable. It is strongly suggested that possibly many (if achievable, all) rot-containing stumps must be removed during harvesting of stumps. Provided equal availability, the priority should be given for stump removal from root rot-infested forest areas, instead of healthy ones. Several questions must be yet answered under Fennoscandian conditions: a) if and to which extent the conventional stump removal for biofuel on clear-felled sites could reduce the occurrence of *Heterobasidion* and *Armillaria* in the next forest generation, b) what impact is it likely to have on survival of replanted tree seedlings, and c) what consequences will there be for growth and productivity of next forest generation.

Stump removal trials for root rot control in *Picea abies*, investigated in this study were established in 1958 on two *P. abies* clear-cuts severely infected by *Heterobasidion*. One of them is located in Skåne (Ramsåsa) and another in Norrland (Brynge). They were evaluated in 1975/78 and in 1984/85. In first evaluation (after 18-20 years) the frequency of *Heterobasidion* infections to next generation *P.abies* trees in de-stumped sites was 0-0.7%, but in sites from which stumps were not removed it was 8-11% (Stenlid 1987). The aim of this study was to evaluate the persistence of the effect of stump removal on root rot frequency after yet another 25 years (Cleary et al., 2012). Simultaneously, tree diameter growth was also checked. In year 2008, on those sites a total of 589 Norway spruce trees were sampled (106 in Ramsåsa and 483 in Brynge), their diameters were measured and presence of rot recorded by taking increment bore cores and isolating fungi. In de-stumped site at Brynge, *Heterobasidion* incidence was 17.0%, while on sites with remaining stumps (negative control), it was 33.6-37.8%, and the difference was statistically significant (p=0.002). In Ramsåsa, control site in the meantime was destroyed by storm (root rot increases susceptibility to wind damage), thus only stump removal site was available. There, the frequency of *Heterobasidion* infections was 8.3%, thus much lower than it was on control (stumps remaining) sites back in 1985, prior to storm damage (16.7%). In Brynge, mean dbh of trees on de-stumped sites was 15 cm, while on sites with stumps remaining it was 17 cm, thus the decrease in the mean diameter of tree on de-stumped sites was small, although statistically significant (p=0.005). Most of *Heterobasidion* genotypes found in 1985, were also found in 2008. Other fungi, causing decay in spruce in were *Armillaria* sp., *Coniophora puteana, Resinicium bicolor, Fomitopsis pinicola, Stereum sanguinolentum* and *Pholiota squarrosa*, but they seldom occurred (1-4 trees infected). In conclusion, the present study shows that, i) infection frequencies *Heterobasidion* significantly increased with stand age (p=0.001 in Brynge, and p=0.005 in Ramsåsa), but that ii) stump removal significantly reduced the incidence of root rot in Norway spruce of next forest generation.

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A number of works report high fungal diversity in wood of cut stumps. Yet, the crucial questions remain: a) are among those fungi rare and threatened species? b) if yes, are those species dependent on cut stumps for survival and completing the life cycle? (producing sporocarps and releasing spores for further spread in an ecosystem). To investigate this, Penttilä (2004) conducted sporocarp-based study examining a total of 14252 man-made stumps in mature managed (age <120 yrs), over-mature managed (age > 120 yrs) and natural old-growth (age 130 – 200 yrs) spruce stands in Finland. Not a single red-listed (endangered) species of wood-inhabiting fungus has been found on stumps. Threatened species were almost entirely found in old-growth forests and on large-diameter logs in advanced stage of decay. Even among the common fungi there was not any species, for which a man-made stump would be the obligatory substrate for survival and completion of lifecycle, as they are typically occur also on other types of woody substrates. Notably, *Heterobasidion parviporum*, a serious root rot pathogen of living spruce causing high economic losses in managed forests found almost exclusively in cut stumps in managed stands. We conclude, that as cut stump is a specific substrate produced by man during forest management (and in huge numbers over large territories), harvesting of stumps does not constitute any threat to natural diversity of wood-inhabiting fungi.

ACKNOWLEDGEMENT

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REFERENCES

Cleary, M.R., Arhipova, N., Morrison, D.J., Thomsen, I.M., Sturrock, R.N., Vasaitis, R., Gaitnieks, T., Stenlid, J. 2012. Stump removal to control root disease in Canada and Scandinavia: A synthesis of results from long-term trials. Forest Ecology and Management (in press).

Penttilä, R. 2004. The impacts of forestry on polyporous fungi in boreal forests. Doctoral Dissertation. Department of Biological & Environmental Sciences, University of Helsinki.

Stenlid, J. 1987. Controlling and predicting the spread of Heterobasidion annosum from infected stumps and trees of Picea abies. Scandinavian Journal of Forest Research. 2:187-198.

Vasaitis, R., Stenlid, J., Thomsen, I.M., Barklund, P., Dahlberg, A. 2008. Stump removal to control root rot in forest stands: a literature study. Silva Fennica. 42:457-483.

STUMP REMOVAL TO CONTROL ROOT ROT OF *PICEA ABIES* IN DENMARK

Iben M. Thomsen[1] and Rimvys Vasaitis[2]

The aim of this study was to check impact of stump removal on occurrence of root rot in the next generation of *Picea abies*. This is a second of only two available trials of a kind in Scandinavia (Cleary et al., 2012). The trial has been established in 1964 on a clear-cut *Picea abies* stand heavily infected by the root rot at Froslev, Denmark. The site has been divided into 4 parcels (A, B, C & D), of which A and C were treated by stump removal, while in B and D the removal has not been accomplished (control). Area has been replanted by *P. abies* and fir in proportion 3 : 1. From year 1967 to 1990 all parcels were regularly inspected for dead trees, and on those the symptoms of infections by *Heterobasidion* and *Armillaria* were recorded in order to determine cause of mortality. The plantation has been thinned three times (in years 1991, 1997 and 2005), and on each stump presence or absence of rot has been also recorded. During the thinning in 2005 it was observed that 70% of rot in parcel C (de-stumped) originated from its part that was bordering parcel B (control). Following thinning, all stumps were sprayed either with nitrite or urea. Therefore it was ensured that all infections of rot in the trial occur via root contacts in the soil, and not by ”new” primary infections via airborne basidiospores via surfaces of freshly cut thinning stumps. In 1983, the last fir has died out in the plantation, thus it became a pure *P. abies* stand. In 1991, top section has been cut from each stump with root rot symptoms. Cut slices were transported to the laboratory, kept in moist chambers (plastic bags), and subsequently their surfaces were examined microscopically for conidia of *Heterobasidion*. This was done in order to check what proportion of observed rot should be attributed to *Heterobasidion*.

In all parcels, *Heterobasidion* was the cause of mortality for 32% of trees. However, parcels where stump removal has not been accomplished contained three times more trees killed by *Heterobasidion*, although the removal of stumps had no effect on the occurrence of *Armillaria* (Table 1). As one could expect, rot frequency in the plantation was increasing with age, but it was consistently lower on sites from which the stumps had been removed (Table 2). In particular, parcel C throughout all the period had clearly less rot frequencies as compared to parcels where stumps were not removed (Figure 1). Study in 1991, which focused on occurrence in particular of *Heterobasidion*, has revealed presence of the pathogen in 30% of thinning stumps in parcels without removal, while in parcels where stumps had been removed the pathogen was detected in only 7% of stumps (Figure 2).

In agreement with the majority of other related studies (Vasaitis et al., 2008), stump removal had an effect and reduced the occurrence of root rot in the plantation, although the effect was decreasing with stand age. Parcel C is a good example of successful stump removal for controlling the root rot. In first thinning, root rot in the parcel C was very seldom observed, but the frequency of infections sharply increase after subsequent thinnings in all parcels (Figure 1). As during the thinnings amount of stumps and, consequently, dead root systems were produced, one possible explanation could be that spread and secondary infections by *Heterobasidion* proceed more efficiently via dead roots as compared with living roots. Another possible explanation that wounds on remaining trees were inflicted during

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thinning, opening gate for infection by wound rot fungi. The frequency of root rot was extremely high in parcels B & D where the stumps were not removed, but also in treated parcel A since 1997. Almost half of all trees there were containing rot already at the age of 37 years. As root rot frequency was very high in control parcels B & D already in first thinning, this indicates spread of disease from stumps and roots of previous stand to outplanted trees of new generation, and furthermore from tree to tree.

It is clear that stump removal had not the same effect in treated parcels A & C, in particular from the long-term perspective. It is possible that stump removal in parcel A was not thorough enough and many remnants of rotten roots had been left on the site, resulting in failure of root rot control. It is unlikely that root rot frequency was higher in old stand at the parcel A, because significantly higher rot occurrence would have been noted at the time when the experiment was established. Despite lower rot frequency in de-stumped sites it is doubtful that the observed effect could pay off the investment in such a costly operation when aimed solely at root rot reduction. However, as a part of a multipurpose operation, e.g. harvesting stumps for biofuel or cleaning forest areas uprooted by wind, stump removal should be regarded as a plausible measure, at least for keeping root rot infections at possibly lowest level.

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Table 1: Tree mortality in years 1967 – 1990.

Cause of mortality	No. (%) of dead trees in study areas (parcels)		
	stumps removed (A+C)	stumps not-removed (B+D)	all (A+C+B+D)
Heterobasidion	14 (25)	41 (75)	55 (32)
Armillaria	30 (50)	30 (50)	60 (35)
Other	28 (49)	29 (51)	57 (33)
All	72 (42)	100 (58)	172 (100)

Table 2: Rot frequency in trees cut during different thinnings.

Thinning year	Stand age, years	Trees cut, no.	Trees containing rot in study areas (parcels), %	
			stumps removed (A+C)	stumps not-removed (B+D)
1991	31	484	12	37
1997	37	669	31	44
2005	45	339	42	52

REFERENCES

Cleary MR, Arhipova N, Morrison DJ, Thomsen IM, Sturrock RN, Vasaitis R, Gaitnieks T, Stenlid J (2012) Stump removal to control root disease in Canada and Scandinavia: A synthesis of results from long-term trials. Forest Ecology and Management (in press).

Vasaitis R, Stenlid J, Thomsen IM, Barklund P, Dahlberg A (2008) Stump removal to control root rot in forest stands. A literature study. Silva Fennica. 42: 457-483.

STUMPING TO CONTROL ROOT DISEASE IN BRITISH COLUMBIA: WHAT DID WE GET FOR $50,000,000?

Michelle Cleary[1]

Across the southern one-third of British Columbia (BC), Canada, *Armillaria ostoyae* (Romagnesi) Herink occurs in a variety of different ecosystems but it is especially damaging in Interior-Cedar-Hemlock (ICH) biogeoclimatic zone (Cleary et al. 2008). Within this zone, western redcedar (*Thuja plicata* Donn ex D. Don) and western hemlock (*Tsuga heterophylla* (Raf.) Sarg.) comprise the dominant species in the mature overstory (Lloyd et al. 1990). Here, symptom expression occurs in only about one-quarter of the trees that are actually infected belowground (Morrison et al. 2000). Mortality in juvenile stands results in unstocked or understocked openings that can sometimes occupy more than 30% of the stand area still at a relatively early stage of stand development (Morrison and Pellow 1994). Moreover, the fungus causes significant growth loss on trees that sustain non-lethal infections (Cruickshank 2000; Cruickshank et al. 2011). Over a rotation, such losses become quite profound and result in a significant reduction in anticipated volume at harvest.

The incidence and spread of *A. ostoyae* is exacerbated by forest management activitites (Morrison and Mallet 1996). Tree stumps are colonized by *A. ostoyae* residing on roots and the fungus infects neighbouring trees by mycelial spread across root contacts and root grafts and by rhizomorphs. In large stumps, the fungus can remain viable for decades. Robert Hartig (1874) first suggested that removal of infested root systems from soil will reduce future infections by *Armillaria* spp. The underlying principle behind 'stumping' is to remove the food base from which the fungus thrives, reduce the amount of inoculum available to infect newly regenerating trees, and thereby lower the probability of root disease transmission. The size of the food base is important in terms of fungal longevity; large inoculum masses pose a higher risk to trees both early and later in the life of the stand.

Since the early 1990's, stump removal has been a common forestry practice in BC. It is the preferred method of control on infested sites, especially in the ICH zone. Current guidance for Armillaria root disease management in BC is given in Cleary et al. (2008) in the form of a decision key which differentiates between the known distribution of *A. ostoyae* inoculum and the extent of damage on host species within the different ecosystems in the southern interior region. For example, in all but the driest sites in the ICH zone, *A. ostoyae* is almost universally present. In all other biogeoclimatic zones, the fungus has a more patch-wise distribution and therefore more investigation may be required to stratify diseased areas before deciding on treatment options. The main treatment options, as shown in Cleary et al. (2008), include stump removal, planting with alternate species or species mixtures where it's ecologically feasible to do so, or the 'do-nothing' approach in which the volume or species that are expected at rotation will not realized and such reductions must accounted for in timber supply analyses.

As current legislation in BC allows for professional reliance, strict guidance for where and when stumping should take place cannot be enforced. The onus falls on the forest professional to first identify root disease as a problem on a site and then suggest stump removal as an appropriate measure to minimize the long-term impact of forest health in the next rotation stand.

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Investment in stumping operations primarily lies in the hands of the Crown because of the value placed upon the potential impact of Armillaria (and Phellinus) root diseases on rotation production. For a licensee, an adjustment will be determined for that part of the cutting authority area where stumping for root disease control is required. The licensee submits a cost estimate for root disease control to Appraisals showing the treatment area accurately delineated on a map and a cost estimate is calculated based on piece size. The range for stumping costs in the southern interior region is between $600-$1,200 per hectare, averaging around $1,000 per hectare. Since 1991, the cost estimate in Appraisals for the specified ops was just over $53 Million. (K. Chantler, personal communication, July 2010). However there may be some adjustments to that figure depending on what the licensee actually did on the ground. This equates to an area of almost 50,000 hectares that have been treated (stumped) in the southern interior region alone (Figure 1), however the total treated area would be considerably higher if the coast region were included. Figure 2 shows that most stump removal operations occur within the ICH zone and its transitional areas where some of the biggest impacts of root disease are realized.

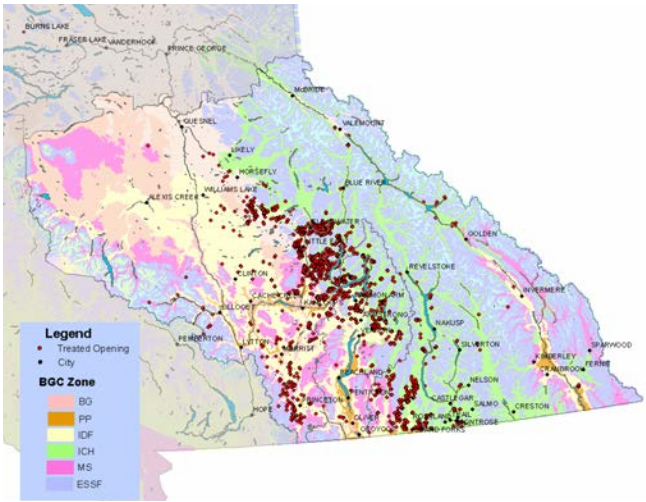

Figure 1: Location of stumping operations in the southern interior forest region of BC since 1991. The cumulative area is approximately 50,000 ha.

There has been much criticism regarding the practice of stump removal with arguments that stumping creates detrimental soil disturbance, in some cases degradation, that it negatively impacts long-term site productivity, and that better results could be obtained by using less aggressive strategies. Operational limitations that have been developed for stumping (Sturrock 2000) among which include: continuous slopes greater than 30%; soil textures susceptible to compaction (e.g. clay, silt) unless otherwise demonstrated that it will not cause soil disturbance limit greater than 5%; where soil depth is shallow over bedrock; soil moisture regimes that are subhygric or wetter; within Riparian Reserve Zones or in wet microsites, fish bearing streams or wetlands; and where the operation may negatively affect reserve trees, reserved areas, or reserved standard units. The non-convincing argument also stems from the fact that even in stumped areas we still see some mortality happening in the next regeneration stand. So what do we know about the long-term effect of stumping on root disease incidence and just how effective is the treatment?

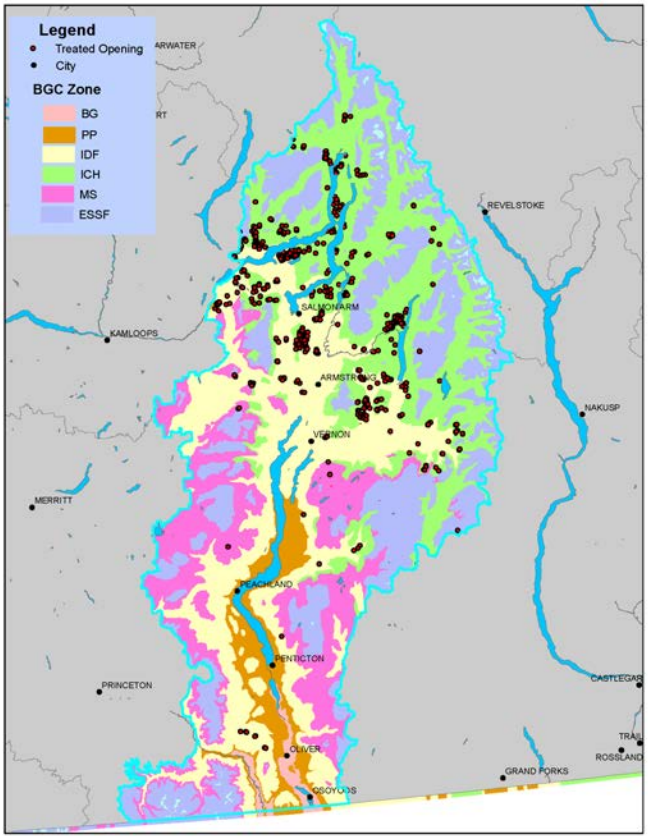

Figure 2: Location of stumping in the Okanagan-Shuswap District in the southern interior forest region of BC showing most operations occurring in the ICH biogeoclimatic zone and its adjacent transitional areas.

The Skimikin trial is the oldest stump removal trial in western North America. It was established in 1968 by Larry Weir to determine the efficacy of inoculum removal for controlling Phellinus root disease (*Phellinus sulphurascens* Pilát (formerly *P. weirii* (Murr). Gilbn.). Secondary objectives were to assess the resistance of several tree species and to test the effect on disease spread of alternating rows of susceptible and less susceptible species (Morrison et al. 1988).

The experiment was located on a site in the moist-warm subzone of the ICH biogeoclimatic zone near Salmon Arm, BC. The previous stand consisted of Douglas-fir and lodgepole pine (*Pinus contorta* Dougl. *et* Loud. var *latifolia* Engelm. *ex* S. Wats.) with minor components of western redcedar and paper birch (*Betula papryifera* Marsh). At harvest, approximately 20% trees were recently killed by root rot and approximately 60-70% of coniferous stumps had stain or decay typical of *P. sulphurascens* (Morrison et al. 1988). Subsequently, *A. ostoyae* was also discovered on site. The experimental design consisted of two blocks (80 m x 160 m) that were clearcut. In the first block, trees were pushed over with a bulldozer and removed from the site with roots still attached. The site was then raked to a depth of about 45 cm with a toothed, land-clearing blade. In the second block trees were felled and skidded to a landing in the conventional manner. A 10-m wide border was cleared and root raked around blocks to prevent the disease from entering the blocks from the adjacent forest. In each block, 32, 20 m x 20 m plots species plots were randomly established comprising those species in the original stand: Douglas-fir, lodgepole pine, western redcedar, and paper birch (3 species plots; alone and in all combination of two species). One of the two remaining plots in each block was planted with western larch (*Larix occidentalis* Nutt.) and Engelmann spruce (*Picea engelmannii* Parry). Seedlings that suffered early planting failure were replaced with similar planting stock in 1969 and 1970. Assessments of tree condition was first conducted in 1973 with the number of living trees in 1973 taken as the starting point. Mortality from *A. ostoyae*, *P. sulphurascens*, and other causes was determined during subsequent assessments in 1977, 1981, 1983, 1987, 1992, 1997, and 2007.

The principle causes of mortality in this trial have been *P. sulphurascens* and *A. ostoyae*, especially the latter. After 20 years, cumulative mortality by *A. ostoyae* in the untreated block ranged between 9-27% for Douglas-fir, lodgepole pine, western larch and Engelmann spruce compared to 1.3-3.0% in the treated (stumped) block (Morrison et al. 1988). After 40 years, cumulative mortality range between 23-38% for the same species in the untreated block compared to 2.8-4.6% in the stumped block (Cleary et al. 2012; Morrison et al. *in preparation*). These cumulative values are 5 (for Douglas-fir) to 14 (for western larch) times greater in the untreated block than in the stumped block. No paper birch or western redcedar were killed in the stumped block and only 3.9% and 6.4% respectively, in the untreated block (Cleary et al. 2012; Morrison et al. *in preparation*). After 40 years, mortality caused by *P. sulphurascens* (restricted to Douglas-fir) was 0.2% in the stumped block and 18% in the untreated block.

Mortality rates varied among species. For example, in Engelmann spruce, there was a steady increase in mortality in the unstumped block, peaking at about 25 years, and then mortality declined. However in recent years, mortality has been steadily increasing again. The first wave of mortality was likely due to trees contacting primary inoculum (stumps from the previous stand), whereas the second wave is now occurring because of tree-to-tree spread. In lodgepole pine, mortality rates in the stumped block were fairly constant during the different assessment periods albeit significantly lower than in the untreated block up until about age 30 and then mortality increased, likely a result of tree-to-tree spread within plots. For western larch, there was very little mortality in the stumped block, and only up until age 20. In the unstumped block, mortality increased and peaked at about age 20 before starting to decline. During the last decade, there's been no mortality in larch which agrees with earlier studies showing increased tolerance to *A. ostoyae* with age (Robinson and Morrison 2001; Robinson et al. 2004). In western redcedar, there was no mortality at all in the stumped block, and in the untreated block, mortality was very low which may be attributable to more effective resistance mechanisms operating in roots that enable better survival of the species in the presence of inoculum

relative to other common conifers (Cleary et al. 2011). In Douglas-fir, mortality caused by *A. ostoyae* in the untreated block increased up until about age 20. Thereafter, mortality has been slowly and steadily declining but still each year some trees are being killed. Mortality in the stumped block was significantly lower than in the untreated block, but after age 20, fewer trees have been killed. Only a couple Douglas-fir trees were killed by *P. sulphurascens* in the stumped block and only within the first 20 years (Morrison et al. 1988). However, in the untreated block, the frequency of mortality has been increasing during each assessment period and at present there is no indication that mortality will slow down (Cleary et al. 2012; Morrison et al. *in preparation*).

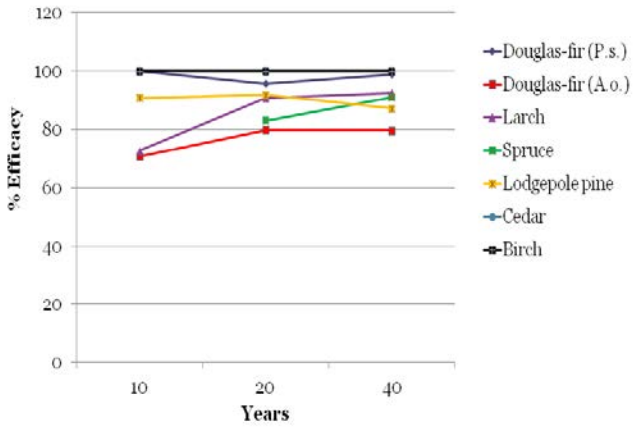

Figure 3: Efficacy of stump removal at Skimkin for the various species.

So what about efficacy of treatment? We see that at minimum, efficacy in terms of reduced mortality caused by *A. ostoyae*, is at 70% at age 10, and for all other species 80% or higher by age 20 (Figure 3). Considering *P. sulphurascens* alone, efficacy was nearly 100%. Over the next several decades one might expect to see an increasing divergence in disease incidence between the treated (stumped) and untreated area (Figure 4). At least in the untreated area, we are still seeing a steady rise in the rate of mortality as a result of more frequent tree-to-tree spread, developed root systems now overlapping across alternate rows in the mixed species plots, and the increasing annual rate of mortality occurring in Douglas-fir caused by *P. sulphurascens*. In the stumped area, the inoculum potential has been greatly reduced and disease levels will likely remain low.

Stump removal is not a sanitation treatment rather its objective is to reduce the amount of inoculum to a level that is somewhat manageable. To assume there will be healthy trees everywhere is an unreasonable expectation considering that stumping does not remove all the roots from the soil. Root raking quite often removes larger diameter roots but also raises roots from lower soil depths to the upper soil levels. Even small, broken roots can serve as viable inoculum for several years and are capable of producing rhizomorphs that can potentially infect and kill regenerating trees (Morrison 1976; Redfern 1970). However, the small volume of residual roots seldom perpetuates root disease losses in the long-term, had the larger stump and root systems not been removed.

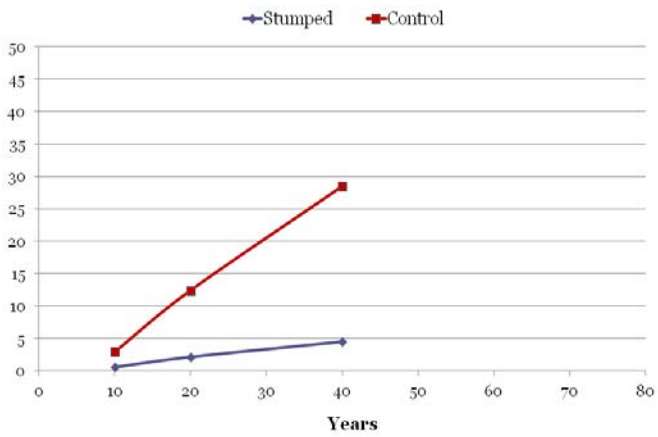

Figure 4: Cumulative mortality of all species in the stumped and control area at Skimikin after 40 years.

Decisions around investments in stumping are based on the value of losses in the absence of control and the effectiveness of the control. One might ask: what is the impact on productivity if stump removal is not done? Take for example the Sugar Lake permanent sample plot which is among the oldest Douglas-fir plantations in the ICH that has been followed through since it was first established. Now approaching the mid-point of rotation, more than 40% of the planted trees at this site are dead, mostly from Armillaria (Morrison 2011). What seems to be happening at this site in terms of species shifts is really quite distinct. Root disease openings are filled in with brush, broadleaved trees, or much younger

western redcedar (Morrsion 2011). These species changes often go unnoticed over time, mainly because of the lack of monitoring done in stands after they have been declared free-growing. Similar trends with respect to changes in the leading species composition have been recorded in other studies (e.g. A. Woods, FREP study of post-free growing stands in the Okanagan TSA, unpublished report). In many cases root disease plays a role in accelerating species succession by removing more susceptible seral species to be replaced by tolerant ones (e.g. western redcedar). Other long-term species trials suggest that where stump removal is not done, high rates of mortality in conifers can be expected, even with more tolerant hosts like western larch (Vyse et al. 2012). Western larch is highly susceptible to being killed by *A. ostoyae* before the age of 20. Its generally rapid growth rate means that roots may contact inoculum in the soil perhaps much sooner than other conifers. In the above examples, there will be lower than expected productivity which means there will be lower than expected harvestable volumes. On sites with high disease incidence, the cost for stump removal is justified through increases in productivity, piece size, and wood quality when a susceptible commercial species (e.g. Douglas-fir) is planted on potentially productive sites (M. Cruickshank, personal communication, October 2010). Where stump removal is impracticable due to soil type, site factors or other reasons, planting species with low susceptibility to killing and/or in higher proportion in mixtures with susceptible hosts may also reduce losses in new plantations (Cleary et al. 2008).

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REFERENCES

Cleary, M.R., Arhipova, N., Morrison, D.J., Thomsen, I.M., Sturrock, R.N., Vasaitis, R., Gaitnieks, T., Stenlid, J. 2012. Stump removal to control root disease in Canada and Scandinavia: A synthesis of results from long-term trials. Forest Ecology and Management [*Submitted*]

Cleary, M.R., van der Kamp, B.J., Morrison, D.J., 2012. Effects of wounding and fungal infection with *Armillaria ostoyae* in roots of Douglas-fir, western hemlock and western redcedar. II. Host response to the pathogen. Forest Pathology. *In press*

Cleary, M., van der Kamp, B., Morrison, D. 2008. British Columbia's southern interior forests: Armillaria root disease stand establishment decision aid. BC Journal of Ecosystems and Management. 9(2):60–65.

Cruickshank, M. 2000. Volume loss of Douglas-fir infected with *Armillaria ostoyae*. Pages 127-129 in T.A. Innes et al. eds. From science to management and back: a science forum for southern interior ecosystems of British Columbia. Southern Interior Forest Extension and Research Partnership, Kamloops, B.C.

Cruickshank, M.G., Morrison, D.J., Lalumière, A. 2011. Site, plot, and individual tree yield reduction of interior Douglas-fir associated with non-lethal infection by Armillaria root disease in southern British Columbia. Forest Ecology and Management. 261:297-307.

Hartig, R. 1874. Important diseases of forest trees. Contributions to Mycology and Phytopathology for Botanists and Foresters, Phytopathological Classics No. 12. American Phytopathological Society. St. Paul, MN. 127 pp.

Lloyd, D., Angrove, K., Hope, G., Thompson, C. 1990. A guide to site identification and interpretation for the Kamloops forest region. Research Branch, B.C. Ministry of Forests, Victoria, Land Manage. Handbook 23.

Morrison, D.J., 1976. Vertical distribution of *Armillaria mellea* rhizomorphs in soil. Transactions of the British Mycology Society. 66(3):393–399.

Morrison, D.J. 2011. Epidemiology of *Armillaria* root disease in Douglas-fir plantations in the cedar-hemlock zone of the southern interior of British Columbia. Forest Patholgy. 41:31-40.

Morrison, D.J., Pellow, K. 1994. Development of Armillaria root disease in a 25-year-old Douglas-fir plantation. Pages 560-571 in M. Johansson and J. Stenlid eds. Proceedings of the Eighth International Conference on Root and Butt Rots, August 9-16, 1993, Wik, Sweden and Haikko, Finland. Swedish University of Agricultural Sciences, Uppsala, Sweden.

Morrison, D. and Mallet, K. 1996. Silvicultural management of Armillaria root disease in western Canadian forests. Canadian Journal of Plant Pathology. 18:194-199.

Morrison, D.J., Wallis, G.W., Weir, L.C. 1988. Control of *Armillaria* and *Phellinus* root diseases: 20-year results from the Skimikin stump removal experiment. Canadian Forest Service, Pacific Forestry Centre, Information Report BC-X-302. 16pp.

Morrison, D.J., Pellow, K.W., Norris, D.J., Nemec, A.F.L. 2000. Visible versus actual incidence of Armillaria root disease in juvenile coniferous stands in the southern interior of British Columbia. Canadian Journal of Forest Research. 30:405-414.

Morrison, D.J., Cruickshank, M.G., Lalumière, A. Root disease control: 40th year results from the Skimkin stumping and species trial. [*In preparation*]

Redfern, D.B. 1970. The ecology of *Armillaria mellea*: rhizomorph growth through soil. Pages 147-149 in T.A. Toussoun and others eds. Root Diseases and Soil-borne Pathogens: Proceedings of the Symposium, July 1968, Imperial College, London. University of California Press, Berkeley. pp. 147.149.

Robinson, R.M., Morrison, D.J. 2001. Lesion formation in the roots of western larch (*Larix occidentalis* Nutt.) and Douglas-fir (*Pseudotsuga menziesii*) in response to infection by *Armillaria ostoyae* (Romagn.) Herink. Forest Pathology. 31:376-386.

Robinson, R.M., Jensen, G.D., Morrison, D.J. 2004. Necrophylactic periderm formation in the roots of western larch and Douglas-fir trees infected with *Armillaria ostoyae*. II. The response to the pathogen. Forest Pathology. 34:119-129.

Sturrock, R. 2000. Management of root diseases by stumping and push-falling. Canadian Forest Service, Victoria, B.C. Technical Transfer Note No. 16.

Vyse, A., Cleary, M., Cameron, I. 2012. Species selection in the Interior-Cedar-Hemlock zone in the southern Interior of British Columbia. Western Journal of Applied Forestry. [*submitted*]

35-YEAR RESULTS FROM THE GLENWOOD TRIAL TO CONTROL ARMILLARIA ROOT DISEASE IN PONDEROSA PINE

C.G. Shaw III[1], D.W. Omdal[2], A. Ramsey-Kroll[2], and L. Roth[3]

INTRODUCTION

By the early 1970's, Armillaria root disease was recognized as the most destructive root pathogen of ponderosa pine in the Pacific Northwest. Forest managers and research pathologists were interested in improving the productivity of severely impacted pine sites and came upon the idea of starving the fungus by removing infected roots during harvest. Evidence that such a procedure might work was gleaned from intensive ground surveys which identified abandoned homesteads where trees were growing well and no root rot was found. Timber of the same age on adjacent uncleared land was often heavily infected with root rot. Working under this assumption, areas within a severely damaged ponderosa pine forest in south-central Washington were treated during a commercial harvest in 1971 to reduce inoculum levels.

METHODS

The study site is located west of Glenwood WA, at an elevation of ca. 900 m in a severely diseased stand of ponderosa pine (SI at 100 yr. is 32 m). The site was mapped and disease was classified into general areas of high and low intensity. There were six treatments, each replicated three times in a block design (Figure 1). The level of sanitation decreased with higher treatment number.

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The six treatments were: 1. Trees pushed out, maximum removal of roots by machine, visible remaining roots picked out by hand; 2. Trees pushed out, maximum removal of roots by machine; 3. Trees pushed out, no further removal of roots; 4. Trees pushed out, large stumps left, otherwise maximum removal of roots by machine; 5. All trees felled, removed by skidding, area cleared of slash, sod scalped, no removal of roots; and 6. Merchantable overstory logged, no further treatment. Trees in treatment 6 provided abundant seed to regenerate the site, but because the treatment differed so markedly in stocking and ground cover from other treatments it was not used in the current analysis.

The entire 3.6 ha tract was divided into three parts. Natural regeneration was destroyed on the central third in 1973 and replaced by a seeding/planting provenance trial. Test provenances were from the local forest, the Deschutes National Forest in central Oregon and the Rogue River National Forest in southwestern Oregon. On the southern third, regeneration was left undisturbed except that up to 1981 dead trees were excavated and examined to determine the cause of death. The northern part was thinned in 1977 to ca. 3 trees/cell and the central planted part was similarly thinned in 1981. All treatments were thinned to one tree per cell in 1992/93.

Each treatment replication had 112 cells in which the effectiveness of the sanitation treatments was evaluated by annually counting mortality. Death of any tree in a cell from Armillaria root disease designated that cell as infested, with the actual mortality levels likely reflecting the minimum component of cells with Armillaria present. After 1992, any mortality would leave a cell un-stocked, whereas prior to 1992, mortality could have left a cell stocked or un-stocked.

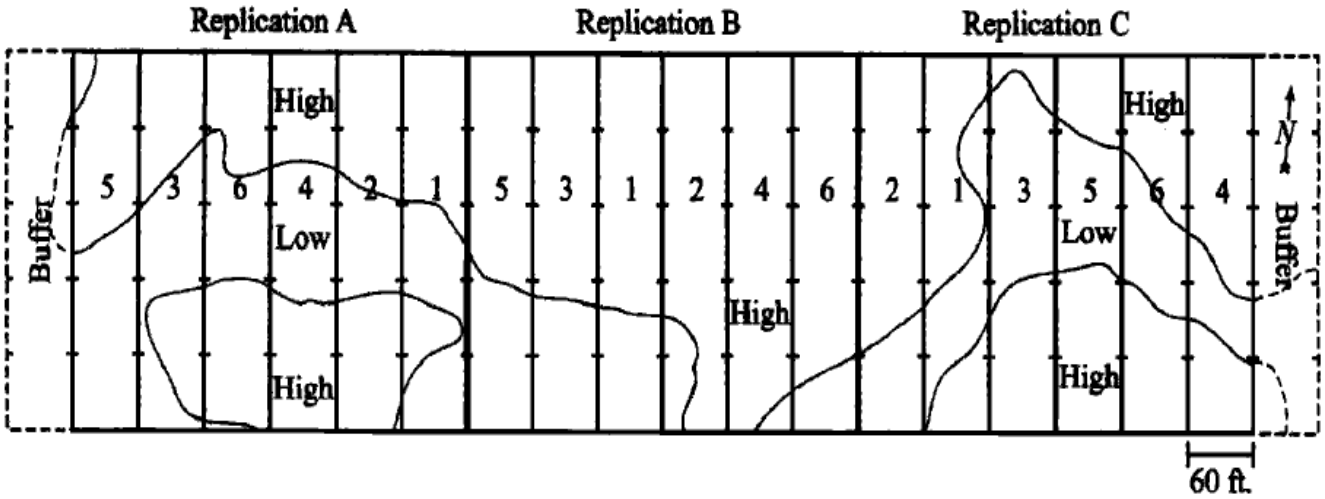

Figure 1: Map of study site with six treatments, each replicated three times in a block design and general areas of high and low disease intensity.

Periodically since the 1980's the designated leave tree in each cell, and the only tree remaining after 1992 was measured for height and diameter.

RESULTS

After 35 years, there was a general tendency for the more intense and thorough root removal treatments to more effectively reduce the occurrence of Armillaria root disease (Table 1). However, even the most intensive treatment, which experienced significantly less disease than most other treatments, had 23% of the area expressing mortality. More than half of the area (nearly 53%) experienced mortality where there was no root removal and just over 40% with push over logging.

In 10/15 treatment/replication combinations, including all three of treatment 5 replications, mortality caused by *A. ostoyae* was found to be clumped (Table 2). That is, cells expressing some mortality from root disease were more likely to be adjacent to one another rather than randomly distributed. It is likely that after 35 years the primary inoculum source is well abated and new infections are the result of secondary inoculum from the current crop of trees. To determine whether clumping is being caused by a likely shift to tree-to-tree spread rather from residual inoculum would require root excavations.

In general, the trees were growing well and there were few significant differences in either height or diameter by root removal treatments (Table 3). Regardless of the root disease treatment, the stand appears to have become more uniform in growth since thinning, and, where planted, the Rogue River seed source performed exceptionally well in this treatment.

CONCLUSIONS

The general hypothesis that removal of root inoculum can reduce levels of root disease is supported by these data in that overall the two most thorough levels of inoculum reduction expressed significantly less root disease mortality after 35 years than the no removal control. However, the only treatment that has promise as an operationally feasible, on the ground management action, treatment #3 (push-over logging) also reduced levels of mortality, but not significantly. From this data it would be difficult to recommend push over logging treatment as an effective or economically viable management strategy for root disease control on these ponderosa pine sites.

Table 1: Percentage of cells with post-treatment mortality due to *Armillaria* through 2007 (35 years after root-removal treatment).

Root removal treatment[a]	Thinned before 1992[b]		Unthinned until 1992[b]		Planted[b]		Pooled[b]		Stocking (%)
1--Push CR	25.5	AB	15.0	A	29.2	A	23.3	A	85
2--Push MR	21.6	A	27.7	AB	43.1	AB	29.8	AB	89
3--Push NR	28.0	AB	44.9	BC	57.4	B	40.3	BC	78
4--Push LS	24.7	AB	46.3	BC	44.4	AB	37.3	B	80
5--Fall NR	44.9	B	55.7	C	59.1	B	52.6	C	73
Pooled	28.9		37.9		46.6		36.1		81

[a]The treatments were (1-Push CR) Trees pushed out, maximum removal of roots by machine, visible remaining roots picked out by hand; (2-Push MR) Trees pushed out, maximum removal of roots by machine; (3-Push NR) Trees pushed out, no further removal of roots; (4-Push LS) Trees pushed out, large stumps left, otherwise maximum removal of roots by machine; (5-Fall NR) Clear logged, sod scalped between stumps, stumps retained.

[b]Within columns, percentages followed by different letters (A-C) differ significantly according to Goodman (1964) confidence intervals (alpha = 0.05).

Table 2: Percentage of cells unstocked in 2007 owing to mortality from *Armillaria*, with (w) and without (wo) a prior history of *Armillaria.* *

Root removal treatment[a]	Thinned before 1992		Unthinned until 1992		Planted		Pooled	
	w	wo	w	wo	w	wo	w	wo
1--Push CR	7	93	50	50	58	42	32	68
2--Push MR	50	50	43	57	64	36	57	44
3--Push NR	38	63	71	29	38	62	44	56
4--Push LS	30	70	64	36	61	39	54	46
5--Fall NR	38	63	54	46	50	50	47	53
Pooled	28	73	57	43	53	47	47	53

*Prior history (w) means the cell experienced some mortality from *Armillaria* prior to the 1992-1993, when all stocked cells were thinned to one tree per cell.

[a]Treatments are more fully defined in notes to Table 1.

Table 3: Mean total height, diameter at breast height (dbh) and standard error, by treatment, of leave-trees 35 years after treatment.

Root removal treatment[a]	ht (m)[b]		dbh (cm)[b]	
1--Push CR	10.2 (0.3)	A	18.7 (0.5)	AB
2--Push MR	11.4 (0.2)	B	19.8 (0.5)	B
3--Push NR	10.2 (0.3)	A	18.2 (0.5)	AB
4--Push LS	9.8 (0.3)	A	16.9 (0.6)	A
5--Fall NR	10.1 (0.3)	A	18.6 (0.6)	AB
Pooled	10.3		18.4	

[a]Treatments are more fully defined in notes to Table 1.

[b]Within columns, means followed by a different letter are significantly different according to Tukey's multiple comparison procedure (alpha = 0.05).

STUMPING FOR CONTROL OF *PHELLINUS*, *TOMENTOSUS* AND *ANNOSUS* ROOT DISEASES: A SYNTHESIS OF RESULTS FROM SOME NORTH AMERICAN TRIALS

Rona Sturrock[1]

INTRODUCTION

Root diseases caused by *Armillaria ostoyae*, *Phellinus sulphurascens*, *Onnia tomentosa* and *Heterobasidion annosum sensu lato* are among the most destructive of biotic disturbances in the forests of western North America. These fungi can persist in diseased stands and their incidence and impact increased if infected stumps and roots (i.e., inoculum) are not properly managed (Sturrock 2000).

The use of machines (e.g., bulldozers, excavators, etc.) to extract stumps and thereby reduce inoculum loads in diseased stands has been tested experimentally, and also applied operationally, for several decades (Figures 1 and 2). The purpose of this communication is to provide an overview of methods and synthesis of results from several stump removal (stumping) trials conducted for the management of *P. sulphurascens* (Table 1), *O. tomentosa* (Table 2), and *H. annosum s.l.* (Table 3) on a variety of sites in western North America. Michelle Cleary's paper has covered several Armillaria root disease stumping trials.

SUMMARY & CONCLUSIONS

Despite the variety of fungi, host species, sites, experimental designs, and equipment, to date, the results from all these trials in western North America indicate that stumping results in a reduction in mortality in the next rotation and an increase in tree growth. These results are in agreement with findings in a recent literature review of stump removal (Vasaitis et al. 2008), which states "that stump removal from clear-felled forest areas in most cases results in a) reduction of root rot in next generation, b) improved seedling establishment and c) increased tree growth and stand productivity". It is important to recognize that stump removal treatments do not result in the **eradication** of root disease in infected forest stands but rather aim to reduce root disease damage to a tolerable threshold. Stumping can be an effective preventive measure against build-up of inoculum potential on many sites and therefore should be considered as an effective, long-term root disease management strategy.

Figure 1: D8 Cat, used to extract stumps in the early days of stumping in western North America.

Figure 2: Linkbelt 4300 hydraulic excavator push-falling a root-diseased tree.

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[1]Canadian Forest Service, Pacific Forestry Centre, Victoria, BC.

Table 1: *Phellinus sulphurascens* (*Ps*, syn. *P. weirii*) trials.

Agency/Investigators & Site Names/Location	**General Description 0f Site(s)**	**Stumping Treatments & Assessments**
USDA FS/W.G. Thies Hoodsport & Apiary, Washington State Gates & Sweethome, Oregon (Thies and Nelson 1988; Thies et al. 1994; Thies and Westlind 2005)	w3.8 – 6.1 ha clearcuts previously naturally regenerated to mainly Douglas-fir (Fd; 40-76 yrs at harvest) w285-603 m average elevation wSilty-cobbly loam soils; site classes II-III	wCrawler tractor with solid or brush blade in fall; stumps/pieces upended; no 2° effort to remove severed roots wFd planted winter; when established, all 239 plots thinned to 2.4 m spacing wFor 7827 trees, dbh, total height, & mortality recorded every 2-5 yrs for up to 27 growing seasons
Results: ***wMortality-***Over all sites, mean mortality due to *Ps* on stumped plots 2.4%; 9.1% on nonstumped plots ***wGrowth-***Stumping increased volume on *Ps*-affected plots by 25.4% Mean reduction in wood volume in *Ps* -affected areas vs non-affected areas was 24.8% ***wOther-***Equipment effects on soil did not reduce Fd survival or growth		
DNR-Washington State/K. Russell Stearns Creek, Washington State (Russell 1991)	w120 yr old Fd stand with 50% + *Ps* infection wSite index 120	wD8 CAT w/brush in fall; "exceptionally large stumps"; Fd planted next spring; when established, 8 plots thinned to ~ 3 m spacing (1100sph) wMortality recorded ~@ 10 & 19 growing seasons post treatment
Results: ***wMortality-***Mortality due to *Ps* on stumped plots 7.0%; 13.5% on nonstumped plots		
CFS/W. Bloomberg & R. Sturrock Cowichan Valley ([C]) & Shawnigan Lake ([S]), BC ([C]Cleary et al. 2012) ([S]Sturrock et al. 1994)	w[C]1.2 ha clearcut CWH site naturally regenerated to Fd, Hw, Cw; ~ 67% trees *Ps*-infected (65 yrs at harvest) w[S]5 ha push-felled CWHxm site naturally regenerated to mainly Fd; ~33% area *Ps*-affected (73 yrs at harvest)	w[C] D8 CAT (Fig. 1); 2 yr BR Fd planted w~1/3 ha plots X 3: control & stumped (1470 sph); stumped & root raked (1600 sph) wMortality recorded ~ every 3-4 yrs for up to 21 growing seasons w[S]Linkbelt hydraulic excavator (Fig. 2) w/live heel & grapple; root balls chainsawed & left upturned or on sides wPlanted to Fd & other spp.; visual monitoring
Results: ***wMortality-***[c]Mortality due to *Ps* on stumped plots 0.9%; 2.7% on stumped/root raked plots; 12.0 % nonstumped plots [s]Site now real estate		
BC Forest Service/S. Zeglen 40+ coastal sites, West Coast Region (*Ps* & *A. ostoyae* on some sites) (Zeglen 1999)	wSeven sites 1999 report coastal BC (6 CWHxm1 & 1 CDFmm); ~3 – 42 ha wSite indices Poor-Med to Good; variable soils	wD7E/8 CAT, some w/brush blade; spring, summer, fall; mostly Fd planted (840 - 1100sph) wTreated strata surveyed for *Ps* & plots established to measure tree growth @ 17 & 20 growing seasons post treatment
Results: ***wMortality & growth-***Stumped strata generally with a lower incidence of RD than nonstumped & better growth by DF ***wIn Progress-***Site data and survey results currently being compiled for remaining 30+ sites stumped using excavator/arm & bucket Second measurement of original 7 sites coming soon		

Table 2: *Onnia tomentosa* (syn. *Inonotus tomentosus*) trials.

Agency/investigators & site names/location	General description of site(s)	Stumping treatments & assessments
BC Forest Service/Nelson Region Mount 7, Golden, BC (Delong 1995)	wICH/MS subzones; silty loam-loam soils; slopes 5-35%	wExcavator pushover in winter; spring planted/Fd, Lw, Spruce wFour treatments (pushover clearcut, pushover light shltrwd, pushover heavy shltrwd, control) X 2, 1 ha plots/treatment
Results: ***wInitial-***"Pushover falling appeared to cause more damage to residuals, advance regeneration and soils as compared to conventional falling." Planted stock not growing well so site ~ inactive		
BC Forest Service/A. Woods Nichyeskwa Creek, BC (FERIC 1996)	w11.5 ha trial site (clearcut ~ 100 km NE of Smithers) previously dominated by subalpine fir & interior spruce wloamy – silt-loam soils	wExcavator/brush rake & hydraulic thumb in Sept.; larger root segments removed; *P. glauca* planted spring at ~ 1600 sph w50 sample trees in each of 11 stumped and 11 unstumped units tagged & measured annually since 1997
Results: ***wMortality & growth-***Stumping reduced mortality & infection by *O. tomentosa* but (to date) not significantly different from unstumped; still too early? Trees in stumped units 11% taller & ~ 20% larger dbh than controls; Alex Woods suggests this partly due to decreased brush competition & soil heating & mixing		

Table 3: *Heterobasidion annosum s.l.* trials.

Agency/investigators & site names/location	General description of site(s)	Stumping treatments & assessments
USDA FS/J. Kliejunas, W. Otrosina, & J. Allison Campground, Big Bear Lake, CA (Kliejunas et al. 2005)	wSix disease centers in proposed campground N shore Big Bear lake, south CA; Jeffrey pine main species	wBackhoe, loader/bucket removed or pushfelled trees; stumps & roots removed by dumptruck; soil removed & large roots removed & put back in holes; 5X3 ft trench adjacent to 2 centers; only one unstumped center wJeffrey pine & *Quercus lobata* planted & several other spp. transplanted & monitored ~ yearly
Results: Mortality-At 12 yrs post treatment, all six stumped/trenches sites disease free while mortality to *Heterobasidion* continues in untreated centers on this site		
Washington State University/G. Chastagner Christmas tree fields, Washington State & OR (Chastagner and Dart 2006)	w21 fields in OR, WA with ~ 34,000 Christmas tree spp. (Noble fir, Fraser fir, Grand fir); previously stocked true firs	wStumps removed at portion of field at 3 sites & one each replanted with Nf, Ff, Gf wMonitored mortality to Annosus for three years
Results: ***wMortality-***Highest mortality in 21 fields was ~ 40%; in three stumped was ~14%		

REFERENCES

Chastagner, G., Dart, N.L. 2006. Effectiveness of stump removal in reducing Annosus root rot losses in Christmas tree plantations. Phytopathology. 96:S166.

Cleary, M.R., Arhipova, N., Morrison, D.J., and others. 2012. Stump removal to control root disease in Canada and Scandinavia: A synthesis of results from long-term trials. *In preparation for Forest Ecology and Management.*

Delong, D. 1995. Shelterwoods in root disease infected stands: Preliminary results – EP 1186. BC Ministry of Forests, Nelson Forest Region, Extension Note 23. Accessed 2/29/12 at http://www.for.gov.bc.ca/rsi/research/nextnotes/rs023.htm

FERIC. 1996. Nichyeskwa Creek tomentosus stumping trial: a short term assessment. Field Note No: Silviculture-91. Forest Engineering Research Institute of Canada. 2 p.

Kliejunas, J.T., Otrosina, W.J., Allison, J.R. 2005. Uprooting and trenching to control Annosus root disease in a developed recreation site: 12-year results. Western Journal of Applied Forestry. 20:154-159.

Russell, K. 1991. Laminated root rot control: stump removal and replanting with Douglas-fir. Informal report presented to Root Disease Committee, Western International Forest Disease Work Conference, August 5-9, 1991, Vernon BC. 4 p.

Sturrock, R.N. 2000. Management of root diseases by stumping and push-falling. Technology Transfer Note 16. Canadian Forest Service. Victoria, British Columbia. 8 p.

Sturrock, R.N., Phillips, E.J., Fraser, R.G. 1994. A trial of push-falling to reduce *Phellinus weirii* infection of coastal Douglas-fir. Canada – British Columbia Partnership Agreement on Forest Resource Development: FRDA Report 217. Victoria, British Columbia. 22 p.

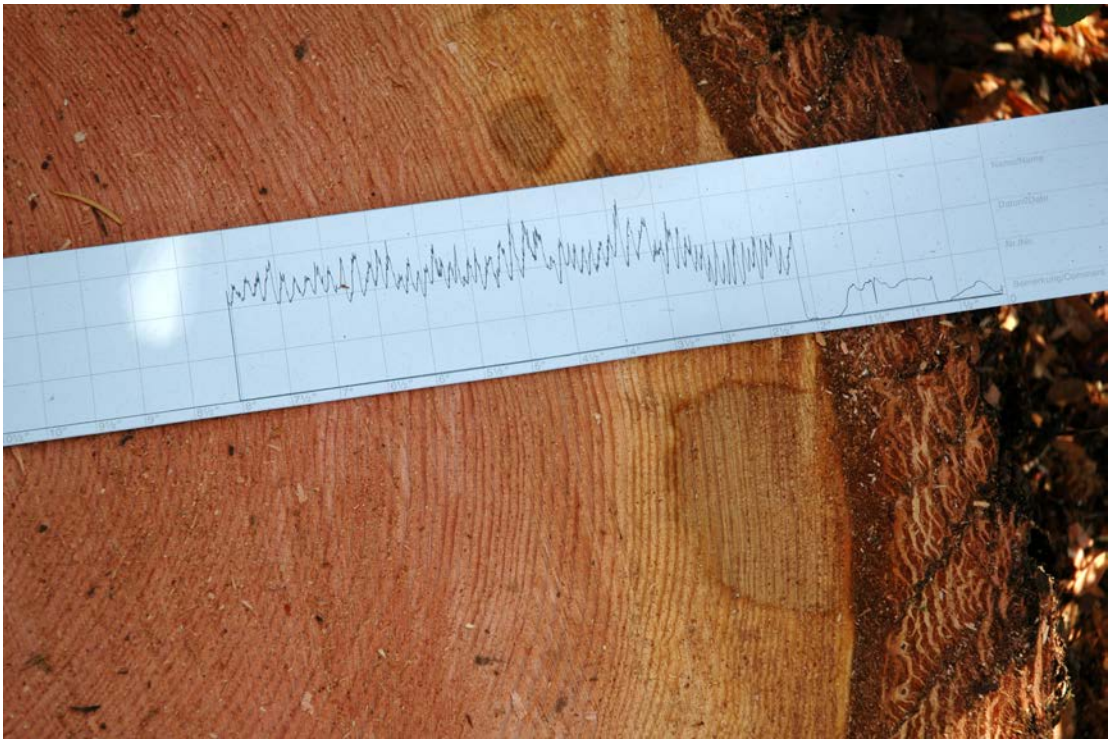
Thies, W.G., Nelson, E.E. 1988. Bulldozing stumps and nitrogen fertilization affect growth of Douglas-fir seedlings. Canadian Journal of Forest Research. 18:801-804.

Thies, W.G., Nelson, E.E., Zabowski, D. 1994. Removal of stumps from a *Phellinus weirii* infested site and fertilization affect mortality and growth of planted Douglas-fir. Canadian Journal of Forest Research. 24:234-239.

Thies, W.G., Westlind, D.J. 2005. Stump removal and fertilization of five *Phellinus weirii*-infested stands in Washington and Oregon affect mortality and growth of planted Douglas-fir 25 years after treatment. Forest Ecology and Management. 219:242–258.

Vasaitis, R., Stenlid, J., Thomsen, I.M., Barklund, P., Dahlberg, A. 2008. Stump removal to control root rot in forest stands. A literature study. Silva Fennica. 42:457-483.

Zeglen, S. 1999. Evaluation of stumping as a root disease treatment in the Vancouver Forest Region: 1977-1980. BC Ministry of Forests, Vancouver Forest Region, Interim Report. 10 p.



IS STUMPING A WISE SOLUTION FOR THE LONG-TERM: THE PROBLEM OF PHENOTYPE-ENVIRONMENT MISMATCH

Geral I. McDonald[1]

"*Without a better mechanistic understanding of how plants and animals work, we can never be assured of an accurate warning of what lies ahead for life on earth.*" (p 197 Denny and Helmuth, 2009)

INTRODUCTION

Expression of root disease in conifers is often associated with forest practices such as planting, thinning, and harvesting. For example, *Armillaria solidipes,* a resident microbe, is "triggered" by these practices or application of fertilizer. On the other hand, the connection to forest practices is not so clear when spores of *Heterobasidion spp*, are delivered to stump surfaces and initiate new infection centers. Natural situations such as ecophysiological maladaptation (McDonald 1991) and insect outbreaks can also trigger expression from resident pest populations. Generally speaking, most current problems concerning root disease pathogens stem from application of management practices and future problems will surely be exacerbated by global climate change. Development of effective management strategies is hampered by poor understanding of landscape level processes ranging from adaptation and evolution to community interactions such as intraspecific and interspecific competition, parasitism, commensalism, and mutualism. These interactions can cause negative or positive impacts on ecosystem function, such as productivity and expression of disease, depending on the particular mix of players at a specific time and place. A potential avenue to understanding and therefore effectively managing these complex living systems is taking shape in a new discipline – eco-evolutionary dynamics (Fussmann et. al. 2007, Kinnison and Hairston 2007, Hendry et.al. 2011, Morris 2011).

A NEW KID ON THE BLOCK

This new discipline has great potential to assist managers of wild resources (Hendry et. al. 2011). Human activity ranging from global climate change to plant and fish breeding can unwittingly create phenotype-environment mismatches, a central tenet of eco-evolutionary dynamics (see Hendry et al. 2010, 2011). Common forest management activities, such as harvesting, fire management, nursery practice, road building, native and non-native pest management, and management of invasive species, can create these mismatches. Development of options to manage these problems has been slow because theories describing how ecology and evolution operate were historically inadequate.

Eco-evolutionary dynamics involves five pivotal concepts. First, the stage was set for creation of new approaches when sequencing of the human genome failed to provide expected gene-based answers and biologists became aware of the ubiquitous nature of phenotypic plasticity (see Fusco and Minelli 2010). Second, a gene-centric theoretical construct, known as the modern syntheses, has provided an inadequate philosophical basis for the study of evolution and genetics for about 70 years. Recently geneticists and ecologists realized that models focused on inheritance via DNA alone yielded incomplete explanations (Day and Bonduriansky 2011). An extended modern synthesis was formulated that includes provisions for: (1) genomes composed of DNA sequences that produce structural and regulatory genes, and transcription factors (genetic inheritance), (2) epigenomes, composed of various chromatin markers, histone modifications, and noncoding microRNA elements, that obtain and forward biotic and abiotic information (nongenetic

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inheritance), and (3) "interpretive machinery", composed of eco-evolutionary feedback loops, that interprets the DNA code to "manage" gene regulation, developmental plasticity, and rapid evolution (see Danchin et. al. 2011, Day and Bonduriansky 2011, Hauser et. al. 2011, Turcotte et. al. 2011).

Since the concepts of nongenetic inheritance and "interpretive machinery" are so important to the following discussion, let us examine how the genetic philosophy of a cell matured from a genetic "black box" (modern syntheses) to a "niche-sensor/actuator" (extended modern syntheses). The niche, in this case, is the realized and/or evolutionary niche (see Holt 2009, Soberon and Nakamura 2009). From about 1930 to 1985 most biologists viewed genetic machinery as a storehouse and dispensary. However, it was believed that cells could "sense" environmental signals and acclimate to those signals in the current generation. From about 1985 to 1990, evidence began to emerge that connected signals to chromatin marks. During the next 10 years, the concept of chromatin remodeling tied the marks to specific cellular behavior or acclimation. Availability of complete genomes (2000 to 2002) quickly led to the ideas that chromatin interacted with gene regulatory networks and that these networks interacted, in turn, with DNA. By 2007 environmental signals were understood to interact with transcription factors as well as ncRNA. In the last 3 years, it has become clear that DNA, gene-regulatory-networks, chromatin, and environmental signals all interact to foster nongenetic inheritance (see Day and Bonduriansky 2011).

The third major element of eco-evolutionary dynamics is "ecological speciation". Cellular mechanisms appear to initiate pre- and post- zygotic suppression of ecologically undesirable gene flow (Hendry et.al. 2007, Wolf et. al. 2010, Nosil and Schluter, 2011). This concept also arose out of the extended synthesis and it supports the expectation of fine-grained, topography driven, and locally adapted intraspecific subpopulations (microrefugia, see Dobrowski 2010). The fourth point, ecological immunology, has revolutionized our understanding of host-microbe interaction (gene regulation networks, evolutionary feedback loops, and rapid evolution) in both plants and animals (see Sadd and Schmid-Hempel 2008, McDonald 2011, Graham et.al. 2011). Reduced fitness resulting from population outcrossing (outbreeding depression) seems to be particularly common regards pests (Allendorf et.al. 2010, Leimu and Fischer 2010). The fifth and final point relates to community interactions. As draft genomes, metagenomes, and metatranscriptomes accumulate, awareness has surfaced that genome x genome interactions are an important characteristic of ecological communities (Yahara et. al. 2010, Mitra et. al. 2011). In total, these five new concepts spawned the pivotal idea that both evolutionary and ecological processes function on similar scales of time and space. The natural integration was eco-evolutionary dynamics.

MECHANISTIC NICHE MODELING

Too complete the picture, new methodologies designed to connect functional traits (Friesen et.al. 2011) to the landscape are taking shape. This is where community ecology and eco-evolutionary dynamics meld. The most popular approach is correlative climate envelope models but little real understanding is gained by their application (Kearney et. al. 2010). On the other hand, mechanistic niche modeling, which arose from animal investigations and is based on biophysical principles, has been applied to landscape analysis of animal populations (Kearney and Porter 2009) and is being generalized through focus on the niche concept (Berg and Ellers 2010, McGill et.al. 2006, Jackson et.al 2009, Webb et.al. 2010, Chown et.al. 2010, Angilletta and Sears 2011). All of the trait-based techniques start by quantifying the organism/environment interface with either continuous or discontinuous reaction norms (species, population, or genotype performance over an environmental gradient). Reaction norms provide a powerful avenue for linking genetic, evolutionary and population factors to environmental factors across all of life (see McDonald et. al. 2005, Aubin-north and Renn 2009, Nicotra et. al. 2010, Alto and Turner 2010, Hendry et. al. 2011,). Even spore germination over a temperature gradient is a continuous reaction norm (McDonald 1996). It is also significant that an analytical methodology is developing to compare and contrast continuous reaction norms (Izem and Kingsolver 2005, Alto and

Turner 2010). As these new approaches develop, so will our understanding of the ecological impact of nearly all forest management activities. As a result, fundamentally different management options will insure truly sustainable forest ecosystems. So, does existing evidence indicate whether or not eco-evolutionary dynamics applies to the question at hand? Is stumping to control root rot a wise solution?

ECO-EVOLUTIONARY DYNAMICS IN FOREST ECOSYSTEMS?

Given that many, if not most, root rot problems in the Pacific Northwest are associated with planting, thinning, natural disturbances and natural phenotype-environment mismatches (see McDonald 1991), one would suppose that the study of eco-evolutionary dynamics would help, providing eco-evo components exist in conifer ecosystems. Evidence shows that open pollinated seed obtained from natural woods-run western white pine exposed to natural blister rust epidemics increased in rust resistance over a short time. Rust incidence decreased at a rate of 0.025/year in field and nursery tests (McDonald et. al. 2004, McDonald unpublished data). Resistance accumulated (proportion of rust free seedlings in artificial inoculations) at almost the same rate as it did in the breeding program.

Nongenetic inheritance (transgenerational epigenetic inheritance) is common in plants (Hauser et. al. 2011) and is known in conifers (Yakovlev et. al. 2010). In yellow monkeyflower, artificial leaf damage initiated increased production of leaf hairs that lasted for 2 generations (Scoville et. al. 2011). Clones of native grasses exhibited rapid trait shifts to tolerate an invader, cheat grass (Gergen et. al. 2011). Two inbred replicate lines of knotweed parents were used to demonstrate nongenetic inheritance of traits influencing drought tolerance (Sultan et. al. 2009). Poplar clones grown in differing levels of drought stress exhibited divergent ecologic function and transcriptomes when grown in well watered and droughty common gardens (Raj et. al. 2011). This study indicates that plants can accumulate variable epigenomes that track the environment in place. Do these results mean that seed banks, such as those formed by Ribes and white pines, track local environments and produce locally preadapted seeds optimized for microrefugia through a combination of epigenetic tracking and nongenetic inheritance? If true, one would expect a link between serious artificial regeneration problems and current nursery and tree breeding practices.

Theory predicts that ecosystems growing in heterogeneous topography should exhibit cryptic subpopulations or microrefugia (Dobrowsky 2011). Collections of western white pine needle tissue at 15 locations in the early 2000s were used to conduct an AFLP molecular marker test (Kim et. al. 2011) of the validity of subpopulations delineated earlier by isozymes. Depending on the type of analysis, early results were confirmed or not. One analysis indicated 12 of the collections represented individual microrefugia and 3 collections were contained within 1 macrorefugium. I conclude available evidence supports the possibility that eco-evolutionary dynamics is an important component of forest ecosystems.

NATURAL PHENOTYPE-ENVIRONMENT MISMATCHES

In 1983, a westwide investigation of the landscape pathology of Armillaria root disease was initiated (McDonald 1991). The behavior of the genus *Armillaria* was related to interactions among hosts and ecological situations by collecting *Armillaria* isolates and host and site data from randomly located plots. Plant community, management history, elevation, slope, aspect, and soil data were used to describe abiotic and biotic character of each .1 acre plot. Using this template, over 800 randomly located plots were installed throughout the conifer forests of the western United States (McDonald unpublished data). The principal goal of this effort was to uncover a connection between *Armillaria* behavior and the forested environment. Plant community classification was used to provide an ecological context in the absence of effective ways to deal with actual abiotic elements such as soil, elevation, slope, aspect, temperature, and moisture or biotic factors such as host species, competition, management activities, and fire (McDonald et. al. 1991, 2000).

Two important initial findings from this effort were that *A. solidipes* distribution and disease expression are related to moisture and temperature gradients. *Armillaria spp*. rarely occurred on either warm and dry or cold and dry sites and both *A. solidipes* and a nonpathogenic and unnamed *Armillaria spp*., North American Biological Species X, commonly co-occurred on cool and mesic to warm and wet sites. On these sites natural stands exhibited little expression of disease whereas planted or thinned stands and natural stands near roads often showed disease (McDonald et.al. 1987). In an effort to further understand *Armillaria* behavior at the landscape level, 631 habitat-types were reduced to 31 subseries or potential vegetation types (McDonald 1998, McDonald et. al. 2000) by utilizing conifer species to indicate soil temperature (continuous soil temperature reaction norm) and understory shrubs and herbs to indicate soil moisture (soil moisture continuous reaction norm). Each subseries can be viewed as a realized niche since each has an abiotic definition (moisture and temperature vectors) and a biotic definition (community assemblage). Next, stand habitat-type data, digital elevation models, satellite imagery, and digital terrain models were coupled to a multivariate technique, most-similar-neighbor, and used to map subseries on the landscape (McDonald et. al. 2003).

Two exceptions to the general rule stated above were expression of *A. solidipes* caused root rot on natural climax Douglas-fir in the Douglas-fir/Dry Herb subseries and on natural climax subalpine fir in the ColdFir/Dry and Moist Herb subseries. The situation with the DF/DH subseries, that is disease expression on ridges and other dry "islands" in a sea of "wet" subseries, led to the ecophysiological maladaptation hypothesis (McDonald 1991). Disease expression on natural climax subalpine fir is clear but the reason is not. It could be that subalpine fir is adapting to a sea of (dry) and disease is being expressed in islands of "wet". Either way would be a natural phenotype-environment mismatch. The finished map provides a view of realized niches in a given landscape as well as *Armillaria* ecological behavior by realized niche. Thus, new mapping techniques mentioned above (Berg and Ellers 2010, McGill et.al. 2006, Jackson et.al 2009, Webb et.al. 2010, Chown et.al. 2010, Angilletta and Sears 2011) offer a powerful new avenue to understand disease on the landscape by assuming that subseries corresponds to realized niches. Of course, this assumption could be tested experimentally.

ANTHROPOMORPHIC PHENOTYPE-ENVIRONMENT MISMATCHES

Can forest practice initiate phenotype-environment mismatches? The idea of locally adapted populations (ecotypes) has been an integral part of forestry practice for many years as evidenced by establishment of elaborate seed zoning programs. However, since significant problems associated with artificial regeneration, thinning, tree breeding and ecological restoration continue to accumulate, some aspect of our basic understanding of forest ecosystems must be missing. The seed zone effort may be failing due to undetected subpopulations because actions of the niche-sensor/actuator were unknown. Common garden experiments show some broad effects but new experimental designs that include comparisons of hybrid to home or transplant to home are required to uncover hypothesized fine-grained niche boundaries.

One place to look for clues about how ecosystems are actually working is management of wild salmon and some plants (see Laikre et.al. 2010, Hutchings 2011, Sgro et.al. 2010) where a similar suite of problems is evident. Important points are: (1) one generation in captivity dramatically reduces survivability of released fry (Christie et.al. 2011), (2) translocation of wild fry from a home river to a foreign river system reduces survivability and other important traits (Hutchings 2011), and (3) comparison of intraspecific local x local against local x away crosses often show outbreeding depression in growth and life history traits (Hutchings 2011). In herbaceous plants (Hufford and Mazer 2003) and fir (Goto et.al. 2011), similar results were observed. In addition, negative effects of translocation and population crossing within species are enhanced when competition and pests are added to the interaction experiments (Leimu and Fischer 2010, Cremieux et.al. 2010). This is not surprising since biotic factors such as pests, competitors, endophyes,

and mycorrhizea are the environmental vectors that define the realized niche (Soberon and Nakamura 2009, Friesen et.al. 2011). These also happen to be the kinds of traits that are often associated with nongenetic inheritance and tend to be most evident early in the life cycle of individuals (Edmands 2007).

ARTIFICIAL REGENERATION

Differing environments like light, density, nutrients, temperature, moisture, microbe populations, pest exposure, endophytes, and competition can push a genotype in various directions (Friesen et.al. 2011). Since many developmental tracks are not reversible, one possible source of phenotype-environment mismatch is the practice of growing seedlings in nurseries in niches far different than those experienced upon planting. This mismatch even includes the shape and size of the seedling's planting space. Also important are increased potential for negative founder effects due to low population breadth of translocated introductions (even an endemic species) and large reduction in screened population by way of the establishment process, e.g. 400 planted and 200 survived compared to 4000 naturals to 200 (Hufford and Mazer 2003). Given the potential for strong interaction between local parents and local environment, the negative consequences of population translocation could be large. A further caveat is that adults and seedlings may have different realized niches where the seedling niche is more restricted (Jackson et.al. 2009).

THINNING

An active niche-sensor/actuator functioning in forest stands could produce some interesting problems. We know that developmental plasticity plays a significant role in plants (Bouvet et.al. 2002, Stinchcombe et.al. 2010) and that specific sets of interactions can develop in genotypes replicated at different locations (Smith et.al. 2011); so it is reasonable to postulate that stands developing at a certain place and time should in some part be expressing their own unique suite of ecosystem interactions that includes the space they occupy. In heterogeneous topography of the Pacific Northwest (where large stand replacing fire is the norm) the functioning realized niche of an even-aged stand could vary in size from about 100m2 to 500000m2 (2Km2). A commercial thinning operation could compact soil, damage trees, and change densities, light, water, wind gradients and trigger new possibilities.

The operation could increase inoculum loads. For example, many western white pine support epiphytic *A. solidipes* yet express little disease. We carried out a seed-tree cut in a 3 hectare 60 year-old western white pine stand over snow in January 1986 to reduce ground disturbance and by May of 1986 numerous stumps showed active new infections of *A. solidipes*. These were existing epiphytic infections, caused by a single genet, that were activated by cutting. During the next 5 years as the stand regenerated to WWP and Grand fir the activated clone killed numerous grand fir and a few WWP and DF naturally regenerating seedlings (see McDonald et.al. unpublished ms, Ross-Davis et. al. These proceedings). The stand currently supports a high density WWP stand and the aggressive *A. solidipes* genet is still actively killing grand fir seedlings but not WWP. Some questions are; what would have happened under a partial cut or a complete stumping and did the cutting trigger a niche-sensor/actuator in the pathogen?

TREE BREEDING

Tree breeding efforts conducted to date have the potential to cause significant damage to coevolved realized niche interaction structures. Seeds are most often produced at orchards located in abiotic and biotic environments far different than those where seedlings will be planted. This interrupts the parent to offspring continuity of the niche-sensor/actuator. One can hypothesize the interruption would be important in a species like WWP where a rotating (3 to 4 years) seed bank is an important source of new natural seedlings. This assumes transgenerational passage of needed information about the local environment through nongenetic inheritance as discussed above. Potential negative outcomes resulting from ongoing tree breeding are caused by failure to test for outbreeding depression and inadequate delineation of a local population structure. Outbreeding is best detected by reciprocal home x away hybrids and home1 x home2 reciprical hybrids grown under natural conditions at both

homes. Another important option would be assessment of population structure with new high-throughput sequencing techniques such as RAD-tags (Emmerson et.al. 2010) to obtain 4,000 to 30,000 markers instead of the customary 100 to 200. Salmon populations separated by as little as G_{st}=0.02 have exhibited significant outbreeding depression (Edmands 2007). A recent study of population structure of western white pine showed pairwise F_{st} values greater than 0.02 for 12 of 15 subpopulations (Kim et.al. 2011).

HIGH-RESOLUTION MAPPING OF *ARMILLARIA* GENETS

Now, I review some evidence provided by the conifer-Armillaria couplet relating to the question at hand. Two unpublished studies of conifer plantations established for ongoing genetic investigations of western white pine and ponderosa pine at Priest River Experimental Forest in northern Idaho may shed some light on how eco-evolutionary dynamics applies northern Rocky Mountain forest ecosystems.

IDA CREEK

A small 2 acre western white pine plantation was established in 1972 from 120 open pollinated WWP families. These woods-run materials were collected to study natural variation in western white pine in support of the Inland Empire blister rust resistance program (described in McDonald et. al. 2004). The plantation was thinned in 1987, at 16 years of age, by removing alternate rows of the 1.2 m^2 spaced plantation. Over 2000 trees were inspected. Stumps of the removed trees were extracted and root systems of remaining trees were inspected for signs and symptoms of *Armillaria spp.* (Figure 1). The 1325 *Armillaria* isolates were subjected to somatic paring to define genets and species. Somatic pairing classifications have been verified by sequencing the IGS2 region of the genome (data on file USFS Moscow Forestry Sciences Laboratory). The 1325 isolates reduced to 7 genets belonging to *A. solidipes* and NABS X (Figure2).

Ecological behavior of genets was ascertained from collection records to provide one detailed geographic snapshot of *Armillaria* populations.

Next, a map of genet and species occurrence was constructed from location data supplied by the 1.2 meter planting grid (Figure 3). In general, low incidence of pathogenic *A. solidipes* and high incidence of saprophytic *Armillaria* NABS X were found. Two genets of *Armillaria* NABS X, a potential biological control agent, occupied most trees and 5 genets of *A. solidipes*, varied from nonpathogenic to significantly pathogenic.

Trees and *Armillaria* isolates

Proportion of trees (2072 total)

	Living	Dead
Clean	.42	.02
Infected	.07	.03
Epiphytic R	.44	.02

Proportion of isolates (1325 total)

	Rhizos	Fans	Rotten wood
Living trees	.77	.10	.01
Dead trees	.06	.04	.02

Figure 1: Ida Creek tree inspections and *Armillaria* isolate collections.

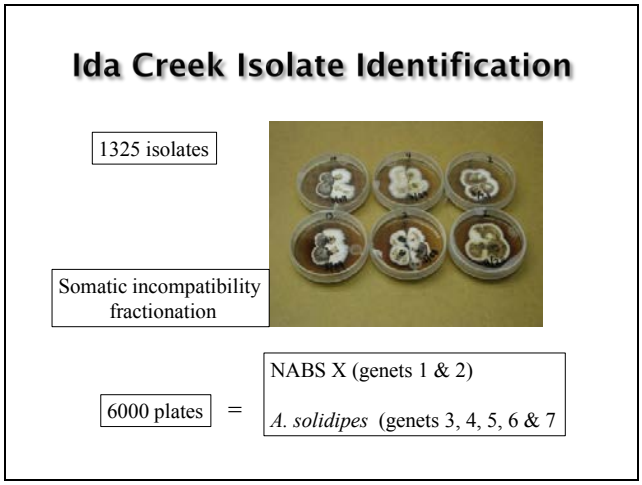


Figure 2: Ida Creek isolate fractionation and identification procedure and results.

Overall ecological behavior and geographical relationships are summarized in Figure 4. Interesting highlights are: (1) neither of the NABS X genets and 4 of the *A. solidipes* genets produced no fruiting bodies during the 10 year observation period, (2) genet 7 produced 4 caps, genet 8 occurred as a single cap (3) NABS X genets freely overlapped *A. solidipes* but not each other, and (4) *A. solidipes*

genets 5 and 7 were geographically split and none of the genets overlapped. Genet variation, in terms of host interaction, was notable (Figure 4) although some genets were underrepresented. Genet 2 dominated with 958 trees with 97% of the occurrences as epiphytic connections on healthy trees and 3% were associated with some resin flow but no fans or rot. *A. solidipes* genet 7 showed disease (no death) on 88% of 129 occurrences and 12% as an epiphyte on healthy trees.

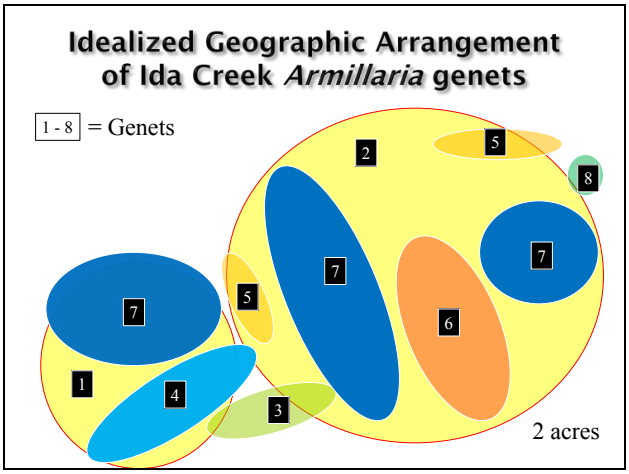


Figure 3: *Armillaria spp* and genets in a 2 acre plantation on the Priest River Experimental Forest in North Idaho.

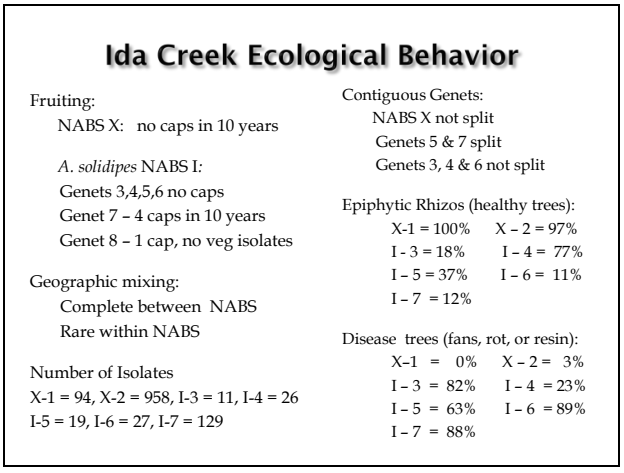


Figure 4: Range of *Armillaria spp* ecological behavior found on the Ida Creek plantation.

PONDEROSA PINE PROVENANCE TEST

A range-wide ponderosa pine provenance planting at Priest River Experimental Forest was established in 1983 on a portion of the site of a 75 year-old planting of exotic conifer species. Nearly all trees in this demonstration plantation were heavily infected with *A. solidipies*. The stand was cut and stumps were removed by excavator and deep roto-tilling. After provenance data collection, trees were extracted at about 8 years of age in the summer of 1990 with the aid of a farm tractor hydraulic lift (McDonald unpublished data). The extracted PP supported numerous infections caused by *A. solidipes* as determined by somatic pairing and IGS2 sequencing. An older range-wide ponderosa pine planting established in 1911, was located about 500 meters NW of the new test. This planting was severely impacted by *A. solidipies* in 1990 (McDonald unpublished data). Eleven percent of the 8 year-old seedlings supported active infections (2% dead, 5% rot and fans, and 4% tiny fans only). Association of infections with seed source has not yet been established. However, about one half of the infected trees supported very small lesions characterized by tiny fans that could not be isolated by our current techniques. Genet and species associations of these fans were not determined. Most lesions, 70%, were not associated with rhizomorphs and therefore could have been initiated from spores. Another interesting finding was that both NABS X and I were isolated from the planting. The main lesson for stumping was that spores could be a significant source of new infections after stumping.

IS CATASTROPHIC NICHE DESTRUCTION A WISE SOLUTION?

Since wholesale removal of large stumps to reduce inoculum loads amounts to catastrophic destruction of a realized niche, one should face the question of eco-evolutionary impact (Weese et.al. 2011). Microbe populations are likely to be significantly affected and their composition will probably significantly influence functional traits of post stumping plant populations (see Friesen et.al. 2011). As discussed above, the perils of artificial regeneration have the potential to be magnified by stumping. The Ida Creek results illustrate potential for interaction complexities just for *Armillaria* species and genets, let alone the remainder of the biosphere. The ponderosa pine example shows potential for significant problems arising from spore reintroductions into a highly disturbed realized niche, even if the disturbed site is regenerated naturally.

Some circumstantial evidence indicates that *Armillaria* NABS X may play a suppressive role regarding *A. solidepes* and that this role may depend on extensive epiphytic rhizomorph networks belonging to a single genet that connects most living woody biomass over 100 to 1000s of square meters (McDonald unpublished data). Further, these networks may be sensitive to disturbance, even stand replacing fire. In the final analysis, the only path to sustainable forest ecosystems may be management systems that emulate natural disturbance regimes (Long 2009), including respect for local realized niches. Otherwise cost of subsidizing ecosystem function may be very high.

Literature discussed and preliminary results presented indicate that an eco-evolutionary dynamic is an important aspect of forested ecosystems. Overlooking or misinterpreting these processes will likely be costly. Significant elucidation of eco-evolutionary dynamics can be uncovered through application of appropriate experimental designs and new genomic tools such as genotyping by sequencing, transcriptome analysis, methylome analysis, metagenomics, genome-wide association studies, and draft sequencing of important interacting species.

REFERENCES

Allendorf, F.W., Hohenlohe, P.A., Luikart, G. 2010. Genomic and the future of conservation genetics. Nature Reviews Genetics. 11:697-709.

Alto, B.W., Turner, P.E. 2010. Consequences of host adaptation for performance of vesicular stomatitis virus in novel thermal environments. Evol Ecol 24:299-315.

Angilletta, M.J. and M.W. Sears. 2011. Coordinating theoretical and empirical efforts to understand the linkages between organisms and environments. Integrative and Comparative Biology. 51:653-661.

Aubin-North, N., Renn, S.C.P. 2009. Genomic reaction norms: using integrative biology to understand molecular mechanisms of phenotypic plasticity. Molecular Ecology. 18:3763-3780.

Berg, M.P., Ellers, J. 2010. Trait plasticity in species interaction: a driving force of community dynamics. Evolutionary Ecology. 24:617-629.

Beisel, C., Paro, R. 2011. Silencing chromatin: comparing modes and mechanisms. Nature Reviews Genetics. 12:123-135.

Bouvet, J-M, Vigneron, P., Saya, A. 2005. Phenotypic plasticity of growth trajectory and ontogenic allometry in response to density for *Eucalyptus* hybrid clones and families. Annals of Botany. 96:811-821.

Christie, M.R., Marine, M.L., French, R.A., Blouin, M.S. 2011. Genetic adaptation to captivity can occur in a single generation. Proceedings of the National Acedemy of Sciences 109(1):238-242.

Chown, S.L., Gaston, K.J., van Kleunen, M., Clusella-Trullas, S. 2010. Population responses within a landscape matrix: a macrophysiological approach to understanding climate change impacts. Evolutionary Ecology. 24:601-616.

Cremieux, L., Bischoff, A., Muller-Scharer, H., Steinger, T. 2007. Gene flow from foreigen provenance into local plant populations: fitness consequences and implications for biodiversity restoration. American Journal of Botany. 97:94-100.

Danchin, E., Charmantier, A., Chanpagne, F.A. 2011. Beyond DNA: integrating inclusive inheritance into an extended theory of evolution. Nature Reviews Genetics. 12:475-486.

Day, T., Bonduriansky, R. 2011. A unified approach to the evolutionary consequences of genetic and nongenetic inheritance. American Naturalist. 178:E18-E36.

Denny, M., Helmuth, B. 2009. Confronting the physiological bottleneck: A challenge from ecomechanics. Integrative and Comparative Biology. 49:197-201.

Dobrowski, S.Z. 2011. A climate basis for microrefugia: the influence of terrain on climate. Global Change Biology. 17:1022-1035.

Drown, D.M., Levri, E.P., Dybdahl, M.F. 2011. Invasive genotypes are opportunistic specialists not general purpose genotypes. Evolutionary Applications. 4:132-143.

Edmands, S. 2007. Between a rock and a hard place: evaluating the relative risks of inbreeding and outbreeding for conservation and management. Molecular Ecology. 16:463-475.

Ellers, J., Marien, J., Driessen, G., Van Straalen, N.M. 2008. Temperature-induced gene expression associated w/ different thermal reaction norms for growth rate. Journal of Experimental Zoology B: Molecular and Developmental Evolution. 310B:137-147.

Emmerson, K.J., Merz, C.R., Catchen, J.M. et.al. 2010. Resolving postglacial phylogeography using high-throughput sequencing. Proceedings of the National Academy of Sciences. 107:16196-16200.

Friesen, M.L., Porter, S.S., Stark, S.C. et.al. 2011. Microbially Mediated Plant Functional Traits. Annual Review of Ecology, Evolution, and Systematics. 42:23–46.

Fusco, G., Minelli, A. 2010. Phenotypic plasticity in development and evolution: facts and concepts. Philosophical Transactions of the Royal Society B: Biological Sciences. 365:547-556.

Fussman, G.F., oreau, M., Abrams, P.A. 2007. Eco-evolutionary dynamics of communities and ecosystems. Functional Ecology. 21:465-477.

Graham, A.L., Shuker, D.A., Pollitt, L.C. et.al. 2011. Fitness consequences of immune responses: strengthening the empirical framework for ecoimmunology. Functional Ecology. 25:5-17

Goergen, E.M., Leger, E.A., Espeland, E.K. 2011. Native perennial grasses show evolutionary response to *Bromus tectorum* (Cheatgrass) invasion. Plos One 6:e18145. Doi:10.1371/journal.pone.0018145.

Goto, S., Iijima, H., Ogawa, H., Ohya, K. 2011. Outbreeding depression caused by intraspecific hybridization beween local and nonlocal genotypes in *Abies sachalinensis*. Restotration Ecology. 19:243-250.

Graham, A.L., Shuker, D.M., Pollitt, L.C., Stuart, K., Auld, J.R., Wilson, A.J., Little, T.J. 2011. Fitness consequences of immune responses: strengthening the empirical framework for ecoimmunology. Functional Ecology. 24:5-17.

Hauser, M-T., Aufsatz, W., Jonak, C., Luschnig, C. 2011. Transgenerational epigenetic inheritance in plants. Biochimica et Biophysica Acta. 1809(8):459-468.

Hendry, A.P., Nosil, P., Rieseberg, L.H. 2007. The speed of ecological speciation. Functional Ecology. 21:455-464.

Hendry, A.P., Lohmann, L.G., Conti, E. et. al. 2010. Evolutionary biology in biodiversity science, conservation, and policy: A call to action. Evolution. 64:1517-1528.

Hendry, A.P., Kinnison, M.T., Heino, M., Day, T., Smith, T.B. et. al. 2011. Evolutionary principles and their practical application. Evolutionary Applications. 4:159-183.

Holt, R.D. 2009. Bringing the Hutchinsonian niche into the 21st century: Ecological and evolutionary perspectives. Proceedings of the National Academy of Sciences. 106:19659-19665.

Hufford, K.M., Mazer, S.J. 2003. Plant ecotypes: genetic differentiation in the age of ecological restoration. Trends in Ecology and Evolution. 18:147-155.

Hutchings, J.A. 2011. Old wine in new bottles: reaction norms in salmonid fishes. Heredity. 106:421-437.

Izem, R., Kingsolver, J.G.. 2005. Variation in continuous reaction norms: quantifying directions of biological interest. The American Naturalist. 166:277-289.

Jackson, S.T., Betancourt, J.L., Booth, R.K., Gray, S.T. 2009. Ecology and the ratchet of events: climate variability, niche dimensions, and species distributions. Proceedings of the National Academy of Sciences. 106:19685-19692.

Jones, E.I., Ferriere, R., Bronstein, J.L.. 2009. Eco-evolutionary dynamics of mutualists and exploiters. American Naturalist. 174:780-794.

Kearney, M., Simpson, S.J., RaubenHeimer, D., Helmuth, B. 2010. Modeling the ecological niche from functional traits. Philosophical Transactions of the Royal Society B: Biological Sciences. 395:3469-3483.

Kearney, M., Porter, W. 2009. Mechanistic niche modeling: combining physiological and spatial data to predict species' ranges. Ecology Letters. 12:334-350.

Kim, M-J., Richardson, B.A., McDonald, G.I., Klopfenstein, N.B. 2011. Genetic diversity and structure of western white pine (*Pinus monticola*) in North America: a baseline study for conservation, restoration, and addressing impacts of climate change. Tree Genetics & Genomes. 7:11-21.

Kinnison, M.T., Hairston Jr, N.L. 2007. Eco-evolutionary conservation biology: contemporary evolution and the dynamics of persistence. Functional Ecology. 21:444-454.

Laikre, L., Schwartz, M.K., Waples, R.S. et.al. 2010. Compromising genetic diversity in the wild: unmonitored large-scale release of plants and animals. Trends in Ecology and Evolution. 25: 520-529.

Leimu, R., Fischer, M. 2010. Between-population outbreeding affects plant defense. PloS ONE 5: e12614. Doi:10.1371/jjournal.pone0012614.

Lingner, T., Abhauer, K.P., Schreiber, F., Meinicke, P. CoMet- a web server for comparative functional profiling of metagenomes. Nucleic Acids Research. 39:W518-W523.

Long, J.N. 2009. Emulating natural disturbance regimes as a basis for forest management: A North American view. Forest Ecology and Management. 257: 1868–1873.

McDonald, G.I., Martin, N.E., Harvey, A.E. 1987. *Armillaria* in the northern Rockies: Pathogenicity and host susceptibility on pristine and disturbed sites. USDA Forest Service, Research Note INT-371. Intermountain Research Station, Ogden, UT. 5p.

McDonald, G. I. 1991. Connecting forest productivity to behavior of soil-borne disease. Pages 129-144 in A.E. Harvey and L.F. Neuenschwander (compilers). Proceedings-Management and Productivity of Western-montane Forest Soils. USDA Forest Service, GTR INT-280. Intermountain Research Station, Ogden, UT.

McDonald, G.I. 1996. Ecotypes of blister rust and management of sugar pine in California. Pages 137-147 in B.B. Kinloch, Jr., M. Marosy, and M.E. Huddleston (eds). Sugar pine. Status, values, and roles in ecosystems. Publication 3362 University of California Division of Agriculture and Natural Resources. Davis, CA.

McDonald, G.I. 1998. Preliminary report on the ecology of *Armillaria* in Utah and the inland west. Pages 85-92 in Proceedings of the 46th Annual Western International Forest Disease Work Conference, Compiled by L. Trummer. USDA Forest Service, Region 10 State and Private Forestry Anchorage, AK.

McDonald, G. I. 2011. After 100 Years Is Coevolution Relevant? Pages 77-90 in M. Fairweather (compiler). Proceeding of the 58th Annual Western International Forest Disease Work Conference; 2010 October 4-8; Valemount, B.C. US Forest Service AZ Zone Forest Health, Flagstaff, AZ.

McDonald, G.I., Harvey, A.E., Tonn, J.R. 2000. Fire, competition, and forest pests: Landscape treatment to sustain ecosystem function. Pages 195-211 in L.F. Neunschwander and K.C. Ryan (technical eds.) Proceedings from the Joint Fire Science Conference and workshop: Crossing the millennium: integrating spatial technologies and ecological principles for a new age in fire management. Volume 2. University of Idaho and the International Association of Wildland Fire, Moscow, Id.

McDonald, G.I., Evans, J.S., Moeur, M., Rice, T.M., Strand, E.K. 2003. Using Digital terrain modeling and satellite imagery to map interactions among fire and forest microbes. Pages 100-110 in K.E.M. Galley, R.C. Klinger, and N.G. Sugihara (eds). Proceedings of Fire

Conference 2000: First National Congress on Fire Ecology, Prevention, and Management. Miscellaneous Publication No. 13, Tall Timbers Research Station, Tallahassee, Fl.

McDonald, G.I., Zambino, P., Klopfenstein, N.B. 2005. Naturalization of host-dependent microbes after introduction into terrestrial ecosystems. Pages 41-57 in Lundquist, J.E. and R.C. Hamelin (Eds.). Forest Pathology: From Genes to Landscapes. The American Phytopathological Society, St. Paul, MN.

McGill, B.J., Enquist, B.J., Weiher, E., Westoby, M. 2006. Rebuilding community ecology from functional traits. Trends in Ecology and Evolution. 21:178-185.

McMahon, S.M., Harrison, S.P., Armbruster, W.S. et.al. 2010. Improving assessment and modeling of climate change impacts on global terrestrial biodiversity. Trends in Ecology and Evolution. 26:249-259.

Mitra, S., Rupek, P., Richter, D.C. et. al. 2011. Functional analysis of metagenomes and metatranscriptomes using SEED and KEGG. BMC Bioinformatics. 12(Suppl 1):S21.

Morris, D.W. 2011. Adaptation and habitat selection in the eco-evolutionary process. Proceedings of the Royal Society B: Biological Sciences. 278:2401-2411.

Nicotra, A.B., Atkin, O.K., Bonser, S.P. et.al. 2010. Plant phenotypic plasticity in a changing climate. Trends in Plant Science. 64:684-692.

Nosil, P., Schluter, D. 2011. The genes underlying the process of speciation. Trends in Ecology and Evolution. 26:160-167.

Raj, S., Brautigam, K., Hamanishe, E.T., O. Wilkins, et.al. 2011. Clone history shapes *Populus* drought response. Proceedings of the National Academy of Sciences. 108(30):12521-12526.

Roelofs, D., Morgan, J., Stirzenbaum, S. 2010. The significance of genome-wide transcriptional regulation in the evolution of stress tolerance. Evolutinary Ecolology. 24:527-539.

Ross-Davis, A., Stewart, J.E., Hanna, J. et.al. 2012. *De novo* assembly and transcriptome characterization of an *Armillaria solidipes* mycelial fan. These proceedings.

Sadd, B.M., Schmid-Hempel, P. 2008. Principles of ecological immunity. Evolutionary Applications. 2:113-121.

Scoville, A.G., Barnett, L.L., Bodbyl-Roels, S., Kelly, J.K., Hileman, L.C. 2011. Differential regulation of MYB transcription factor is correlated with transgenerational epigenetic inheritance of trichome density in *Mimulus guttatus*. New Phytologist. 191:251-263.

Sgro, C.M., Lowe, A.J., Hoffmann, A.A. 2010. Building evolutionary resilience for conserving biodiversity under climate change. Evolutionary Applications. 4:326-337.

Smith, D.S., Bailey, J.K., Shuster, S.M., Whitham, T.G. 2011. A geographic mosaic of trophic interactions and selection: trees, aphids and birds. Journal of Evolutionary Biology. 24:422-429.

Soberon, J., Nakamura, M. 2009. Niches and distributional areas: Concepts, methods, and assumptions. Proceedings of the National Academy of Science. 106:19644-19650.

Stinchcombe, J.R., Izem, R., Heschel, M.S. et.al. 2010. Across-environment genetic correlations and the frequency of selective environments shape the evolutionary dynamics of growth rate in *Impatiens capensis*. Evolution. 64:2887-2903.

Sultan, S.E., Barton, K., Wilczet, A.M. 2009. Contrasting patterns of transgenerational plasticity in ecologically distinct congeners. Ecology. 90:1831-1839.

Turcotte, M.M., Reznick, D.N., Hare, J.D. 2011. The impact of rapid evolution on population dynamics in the wild: experimental test of eco-evolutionary dynamics. Ecology Letters. 14:1084-1092.

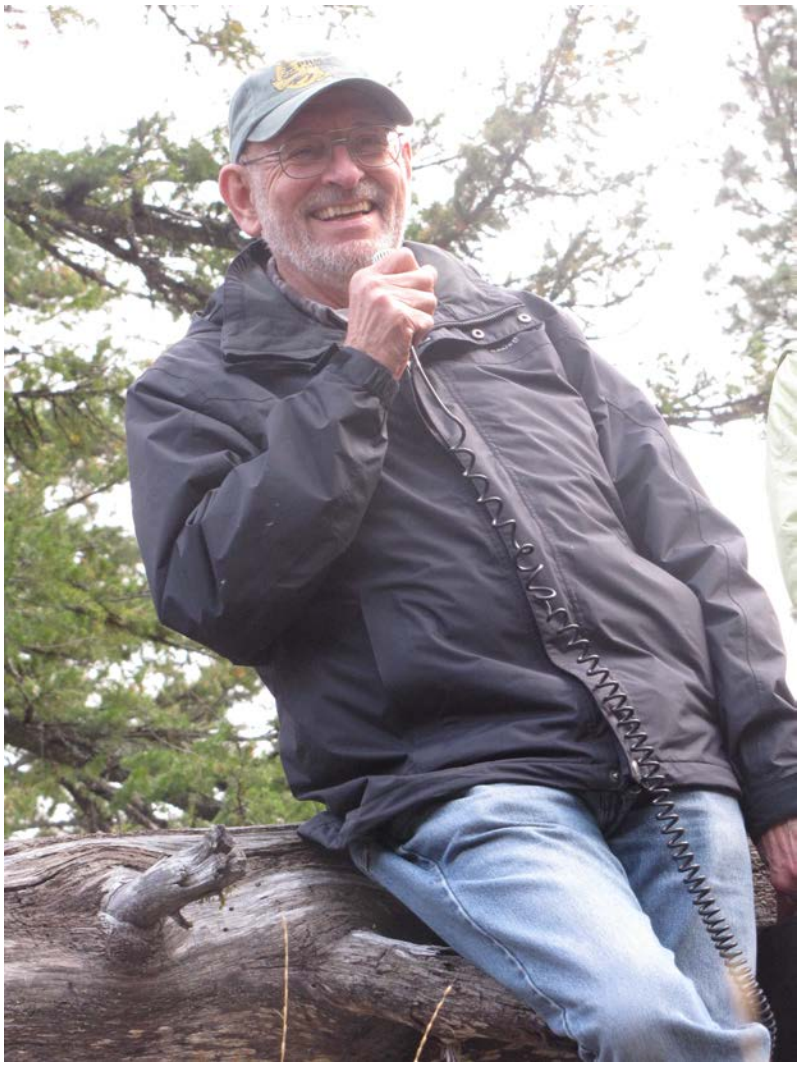
Webb, C.T., Hoeting, J.A., Ames, G.M. et.al. 2010. A structured and dynamic framework to advance traits-based theory and prediction in ecology. Ecology Letters. 13:267-283.

Weese, D.J., Schwartz, A.K., Bentzen, P. et.al. 2011. Eco-evolutionary effects on population recovery following catastrophic disturbance. Evolutionary Applications. 4:354-366.

Wolf, J.B.W., Lindell, J., Backstrom, N. 2009. Speciation genetics: current status and evoloving approaches. Philosophical Transaction of the Royal Society B: Biological Sciences. 365:1717-1733.

Yahara, T., Donoghue, M., Zardoya, R., Faith, D.P., Cracraft, J. 2010. Genetic diversity assessments in the century of genome science. Current Opinion in Environmental Sustainability. 2:43–49

Yakovlev, I.A., Asante, D.K.A., Gunnar, C. et.al. 2011. Differential gene expression related to an epigenetic memory affection climate adaptation in Norway spruce. Plant Science. 180:132-139.



PANEL: PHYTOPHTHORA RAMORUM (SUDDEN OAK DEATH) ERADICATION IN OREGON – WHAT HAVE WE LEARNED IN 10 YEARS?

SUDDEN OAK DEATH ERADICATION SOUTHWEST OREGON: 2001-2011

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ABSTRACT

Sudden Oak Death, caused by *Phytophthora ramorum,* is lethal to tanoak (*Notholithocarpus densiflorus*) and threatens this species throughout its range in Oregon. The disease was first discovered in coastal southwest Oregon forests in July 2001. Since then an interagency team has been attempting to eradicate the pathogen though a program of early detection surveys followed by destruction of infected and nearby host plants. Eradication treatments eliminated disease from many infested sites but the disease continued to spread slowly in a predominantly northward direction. During the 10-year period, the area under quarantine has expanded five times: from 9 mi^2 in 2001 to 202 mi^2 in late 2011. Continued spread of sudden oak death is attributed to the slow development of symptoms in infected trees which hinders early detection, and to delays in completing eradication treatments due to inconsistent funding. A sharp increase in disease in 2010 and 2011 necessitated major changes to the sudden oak death management program and quarantine regulations.

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INTRODUCTION

Sudden oak death is caused by *Phytophthora ramorum,* a pathogen of unknown origin and world-wide importance. It has been causing widespread mortality of coast live oak, tanoak, and black oak in California for nearly two decades, and now occurs in 14 counties there (Rizzo and Garbelotto, 2003; Rizzo et al., 2002). In Europe, where the pathogen formerly occurred only in the nursery trade, it has spread to trees and forests (Brasier and Webber, 2010). The pathogen has potential to spread throughout coastal forests of the US west coast (Vaclavik et al., 2008; Meentemeyer et al., 2004) and to cause considerable ecological and economic damage to the forestry and nursery industries (Hall, 2009). Risk models show that many forests of the world, including the hardwood forests of the eastern United State, are highly susceptible to this pathogen. Accordingly, the pathogen is highly regulated through international and domestic quarantines.

Sudden Oak Death was first discovered in Oregon forests in July 2001 near the coastal city of Brookings, 5 miles north of the California border. Archival aerial photographs revealed tanoak mortality at least one of the infested sites, suggesting that disease probably was present there since 1998 or 1999. At the time of discovery in Oregon we knew of 5 infested sites encompassing a total of 36 acres distributed over an oblong area 2.5 miles (north-south) by 1.2 miles (east-west). Soon after the initial detection we convened an emergency meeting of personnel from the Oregon Department of Agriculture (ODA), Oregon Department of Forestry (ODF), Oregon State University (OSU), and the USDA-Forest Service. Because of the apparently small number of infestations and the unknown potential for damage,

we decided to attempt eradication of the pathogen by cutting and burning all infected and symptomatic host plants in the infested sites. Also at this time, the Oregon Department of Agriculture established an emergency quarantine area of 9 mi^2 from which movement of all host material was prohibited (Goheen et al., 2002).

At the 49th WIFDWC in Carmel, CA, we presented the Oregon sudden oak death situation and plan for eradication. Lively discussion lead to broad support from the members to go forward with the eradication, despite limited understanding of disease ecology and considerable doubt about the long-term outcome. Energized by the support, we embarked on an aggressive program of early detection and eradication. Ten years later, it is clear that we failed in our initial goal of complete eradication of the pathogen from forests. The program now continues with a revised goal of containment and slowing spread, using early detection and eradication as the primary tools for reducing inoculum available for disease intensification and spread.

Ecology of *P. ramorum* in Oregon Forests

P. ramorum produces aerial propagules under wet conditions and mild temperatures. In Oregon its primary host is tanoak which is killed by the pathogen and acts as a source of inoculum throughout the year (Hansen et al., 2008). Many other forest plant species are also susceptible to the pathogen when growing close to tanoak. These include Pacific rhododendron (*Rhododendron macrophyllum*), Douglas-fir (*Pseudotsuga menziesii*) evergreen huckleberry (*Vaccinium ovatum*), red huckleberry (*Vaccinium parvifolium*), Oregon myrtle *Umbellularia californica*, cascara (*Rhamnus purshiana*), and poison oak (*Rhus diversiloba*) (Hansen et al., 2005). They do not appear to be important for disease spread in Oregon forests, at least under the prevailing conditions of the past 10 years.

The disease spreads locally by rain-splash and over long distances (several miles) by wind and wind-driven rain from the canopy of infected tanoaks trees (Reeser et al., 2009). Humans also can spread disease by transporting infected plants or infested materials, but this has not been documented in Oregon forests.

The time between initial infection of tanoak and the development of disease symptoms is not clearly understood in natural forest conditions. Infections on leaves and fine twigs can become apparent within weeks of infection as leave blotches and small lesions, but these are difficult or impossible to detect in standing tanoak trees until infection is abundant and causes discoloration of foliage *en masse*. The time between initial infection in the crown of tanoaks and the development of trunk cankers and tree death tree death appears to range from several months to several years (McPherson et al, 2010; McPherson et al, 2005). This latent period, when the pathogen is present but not readily detectable, is extremely important to the early detection/ eradication program because spore production can occur throughout this period, potentially limiting the effectiveness of eradication treatments.

Early Detection Surveys

The detection program consists of several types of survey, each with their own strengths and limitations. As a group these surveys have proven highly effective. Even though inconsistent funding has impeded the treatment program, we have maintained a consistent detection survey effort since 2001.

Aerial Survey Followed By Ground Checks

Aerial surveys provide the most extensive but least "early" of our detection methods. Four surveys are conducted annually (February, May, July, and October) that extend from the California border north to the Rogue River, and area of approximately 300,000 acres. In the first stage of the survey, observers in a fixed-wing aircraft record the approximate location of recently killed tanoak trees (red-brown foliage) on maps. These records are then used to guide a helicopter to the dead-trees. While hovering over the dead trees the number and condition of the trees are noted and the geographic coordinates determined with hand-held Global Positioning System (GPS) units. Ground crews then

use maps, GPS units and compasses to find the dead trees. All dead-tree locations are visited and trees and plants in the vicinity are checked for symptoms of SOD or other disease. If symptoms are present, two samples of symptomatic plant tissue are collected. One is plated in the field onto *Phytophthora*-selective agar and the other sample is taken to the laboratory for plating and polymerase chain reaction analysis (PCR).

Because aerial surveys are based on the presence of dead trees, they do not offer true early detection. Infested sites detected by this method will have had *P. ramorum* present for months or years prior to the detection. However, because the surveys are flown at frequent intervals and detect recently killed trees, they typically capture new infestations in relatively early stages of development.

Ground Surveys

Ground surveys are very labor intensive but allow detection of an infestation before dead trees are present or visible to aerial observers. They are conducted by two- or three-person crews walking transects spaced 100 to 150 ft apart while looking for symptoms such as bleeding cankers, stem lesions, wilting shoots, leaf spots, and branch dieback on understory plants and live trees. Samples are collected and treated as described for ground checks associated with aerial surveys.

Ground surveys supplement the aerial surveys and provide data necessary to certify areas as "disease-free" to fulfill quarantine requirements for transporting host material. They are undertaken in areas where landowners request surveys or where presence of the disease is likely based on risk maps or proximity to known infestations. Ground surveys of areas with no dead trees present and not located close to infested sites have seldom detected *P. ramorum,* suggesting that sudden oak death is not widespread in areas without dead tanoak trees.

Stream Baiting

Stream baiting with native rhododendron and tanoak leaves is an extensive survey that offers the possibility of detecting *P. ramorum* before tree mortality is evident (Sutton et al., 2009). Stream baiting is carried out in areas considered at risk of new infestation within and beyond the perimeter of the quarantine area. Streams draining known infested sites also are sampled as positive controls. Year-round sampling of approximately 60 bait stations at two-week intervals is interrupted only by summer drought or winter floods. The area of drainages sampled ranges in size from 20 to 8,980 ac and totals 80,000 ac.

In several cases stream baiting indicated an infestation in a drainage area before we detected it by any other means. Follow-up ground surveying eventually lead us to the infested sites. However, there have been a few cases where an infested site occurred in a drainage but the stream baits have been negative for *P. ramorum* (false negative). Conversely, there have been a few instances where we recovered *P. ramorum* from stream baits but have not found upstream infected plants. We do not fully understand the ecology of *P. ramorum* in streams.

Eradication Treatments

Mandatory eradication began in the autumn of 2001 under the statutory authority of the Oregon Department of Agriculture. Funding initially was provided by the USDA-Forest Service and in subsequent years by ODF, USDI-Bureau of Land Management (BLM), and USDA-APHIS. Although there was no direct cost to landowners, no compensation was made for loss of timber or other values. All eradication activities on federal lands managed by the BLM or USDA-Forest Service have been funded by the respective agencies and have been uninterrupted to date.

After initial detection of *P. ramorum* each infested site is surveyed for symptomatic plants and a treatment area delimited. In 2001 and 2002, the treatment area boundary was 50 to 100 feet from infected or symptomatic plants. In subsequent years it was increased to 300 feet, reflecting monitoring data showing that smaller treatment areas often were not large enough to capture the extent of the infestation.

On private and USDA-Forest Service land, eradication treatments consist of felling and burning all host plants within the treatment area as soon as possible after detection. Cutting, piling, and burning are accomplished by hand crews, heavy and light equipment, broadcast burning, and any combination thereof. On BLM land, host plants are cut, piled, partially covered with plastic, allowed to cure, and burned 6 to 14 months later. In early 2011, BLM modified this approach by cutting and burning the actual infected trees immediately and making piles of everything else in the treatment area for burning later.

After the first two years of treatments, tanoak stumps sprouted prolifically and *P. ramorum* was occasionally isolated from the new shoots. In 2004 and 2005, all sprouts from previously burned sites were sprayed with herbicide to kill sprouts. Since 2003, all tanoaks in treated areas (other than those on BLM land where herbicide use was restricted prior to 2011) have been injected with herbicide (imazapyr or glyphosate) prior to felling to prevent sprouting. Follow-up treatments often are necessary to destroy residual host material and stump sprouts that may harbor the pathogen. Upon completion of burning most sites are planted with non-host or conifer seedlings.

Infestations detected in February to May often can be treated immediately if fire precaution levels allow burning and if funds are available. We make this a priority because late winter and early spring are important times for disease spread and intensification. For infestations found in summer and fall, we often start cutting immediately and finish burning when fall rains begin. Our goal has been to complete treatments by the end of December to minimize inoculum availability during winter and spring. It's a good plan, but operationally and administratively it has been difficult to achieve, especially in recent years when funding was inconsistent and the amount of disease had increased.

We were forced to suspend eradication treatments on private land several times because of lack of funds. The first stoppage was from January to May, 2008 on the western part of the quarantine area. Treatments resumed in summer of 2008 and continued through the 2008-2009 winter, but because we gave highest priority to treating new sites near the quarantine boundary, many older sites remained untreated for many months. Because of State budget cuts, we again stopped treatments from April through September, 2009. We resumed treatments at the end of September, 2009 until funds ran out six weeks later. No treatments occurred between mid-November, 2009 and April, 2010. We resumed treatments in April 2010, but by this time we were dealing with a large back-log of untreated or partially treated sites accumulated from 2008 and 2009.

As funds became limiting in late 2010 and 2011, we gave priority to treating sites nearest the quarantine boundary, allowing sites near the center of the quarantine area to remain untreated or partially treated for extended periods of time.

Since 2001, eradication treatments have been completed on approximately 3,000 ac of land, at a cumulative cost of $7.5 million. There has been no compensation to landowners for the value of timber or other resources lost as a result of the eradication treatments. For the period 2001-2009 the area treated for eradication was distributed among landowner groups as follows: private industrial (72%); non-industrial private forests (18%); rural-residential (6%); USDI-Bureau of Land Management (3%); USDA-Forest Service (<1%), and; and State of Oregon (<1%).

Preventative Host Removal

Observations after several years of the eradication program suggested that larger treatment areas would have been more effective that the ones were using. In addition, the idea of a broad host-free zone across the northern part of the quarantine area had been considered as a means of stopping spread northward. Since 2001, 1,400 ac of tanoak have been felled or killed with herbicide in areas of probable disease spread. These host removal activities were voluntary landowner activities supported in part by State or Federal funding.

Eradication Post-treatment Monitoring

In 2008-2010 we surveyed treated sites to determine presence of *P. ramorum* in soil or vegetation. Circular plots (1/20 ac) were established around stumps of known infected trees on sites treated between 2001 and 2008. At each plot 20 one-liter soil samples were collected, returned to the laboratory and baited with leaves of *Viburnum sp*. or *R. macrophyllum*. Presence of *P. ramorum* in the baits was determined by PCR analysis and culturing on *Phytophthora*-selective medium. Host vegetation was examined for symptoms of *P. ramorum*, and a minimum of five plant tissue samples were collected and returned to the laboratory to determine the presence of *P. ramorum* using ELISA, PCR and culturing on Phytophthora-selective media (Goheen et al. 2010; Goheen et al., 2009). We established 145 plots in 2008-2009 and 143 plots in 2010. 109 of these plots were visited in both time periods.

The results suggest that the pathogen was eliminated from many of the sites but persisted on others. In the sample period 2008-2009, *P. ramorum* was not recovered from soil or vegetation on 74 (51%) of the 145 plots sampled. Forty-seven plots (32%) yielded *P. ramorum* from soils only. The pathogen was present in soil and vegetation on 18 plots (12.5%), and on six plots (4.5%), *P. ramorum* was recovered from vegetation only. In the 2010 sampling, *P. ramorum* was not recovered from soil or vegetation on 90 (63%) of the 143 plots sampled. Thirty-six plots (25%) yielded *P. ramorum* from soils only, on 10 plots (7%) the pathogen was present in soil and vegetation, and on seven plots (5%), *P. ramorum* was recovered from vegetation only. All positive vegetation samples were from tanoak in the 2008-2009 sampling period; most of the diseased material was collected from tanoak basal sprouts. Two *P. ramorum*-positive samples of Oregon myrtle were collected in the 2010 monitoring effort along with infected tanoak sprouts. On plots where *P. ramorum* was baited from soil, recovery was generally low, usually only one of 20 soil samples. *P. ramorum* was recovered from soil up to 8 years post-treatment.

Disease Spread, 2001-2011

From 2001 to 2004 the number of new infested sites discovered in surveys remained steady or decreased, suggesting modest success at containment and eradication. In 2005 and 2006 the number of new infested sites and the distance between them began increasing, possibly the result of two consecutive years of unusually wet spring weather which apparently favored spread of the pathogen. Several new sites found during this period were more than two miles from previously known infected trees and outside of the existing quarantine boundary. From 2007 to 2009 the trend in occurrence of new infested sites appeared to stabilize at approximately 60 new disease patches per year, with no new sites outside of the existing quarantine boundary (figure 1). Spread continued to follow a predominantly northward direction of spread.

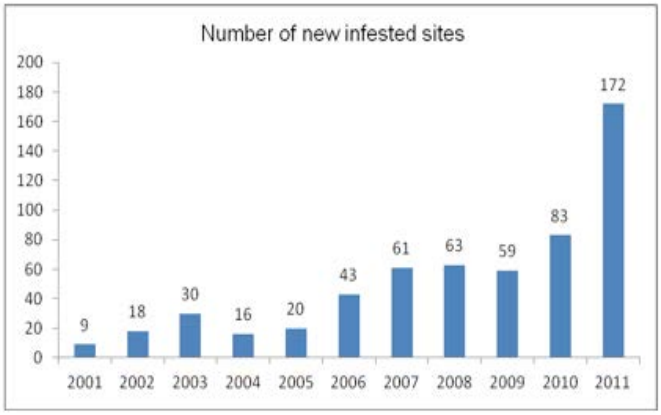

Figure 1: Number of new sites infested with *Phytophthora ramorum* discovered annually between 2001 and 2011 in Curry County, Oregon forests.

In 2010 the number of new sites increased sharply to 83. All new infested sites were well within the existing quarantine area, and most were small with few infected trees, suggesting relatively early detection. Distribution of new sites was uneven with noticeable intensification in the Taylor-Duley creek drainages (west side of quarantine area) where treatment delays had occurred in prior years. Distribution of new infested sites continued to follow the northward pattern of spread.

In 2011 we detected 172 new infested sites, nearly triple the three-year average of new sites detected. The majority of new sites were in the core of the quarantine area, mostly on private land and mainly in one area of rapid disease intensification and spread; the Duley creek – Cape Ferrelo area. Many sites were very close to previous infestations, no doubt the result of delays in completing treatments promptly. In September 2011 one new site was found at Cape Sebastian, 6.5 miles north of the quarantine boundary and 12 miles from the nearest known infested site. At least 25 infected trees were identified at the site, suggesting that the pathogen has been there for at least a year. We do not know if this infestation was the result of natural or human-assistance spread. Treatment of the site was mostly completed by the end of 2011 and involved cutting and burning all host plants in a 28 acre core treatment area and killing all tanoak in a 66 acre buffer area by a combination of herbicide injection and felling. This treatment area is larger than normal, but treatment areas of this size have effectively stopped spread of the pathogen at other locations. It will provide a test to determine if eradication of an isolated infestation is possible.

Disease spread during the 10-year period has been predominantly northward, following the prevailing wind direction during storms and wet weather. If we exclude the Cape Sebastian infestation, the disease has spread from the initial (2001) infestations southward 1.2 miles, and northward and eastward 5.3 and 4.7 miles, respectively. Including the Cape Sebastian infestation increases the northward spread during the period to 17.3 mi. The 2001 quarantine area was 9 mi^2 in size. It has been expanded five times since then, with the most recent expansions to 162 mi^2 and 202 mi^2 occurring in 2008 and 2012, respectively. The current quarantine area and distribution of the disease are show in figure 2.

In many instances new infestations are found very close to eradication sites within a year of treatment. In most cases we believe this is due to latency of the pathogen rather than spread during the treatment process or failure to detect symptomatic trees at the time of delimitation. This problem can be solved somewhat by large treatment area buffers to capture pre-symptomatic infections. In three infestations where we found the disease in early stages and used large treatment areas (25 to 40 acres) the disease has not shown up in the adjacent forest three years post-treatment.

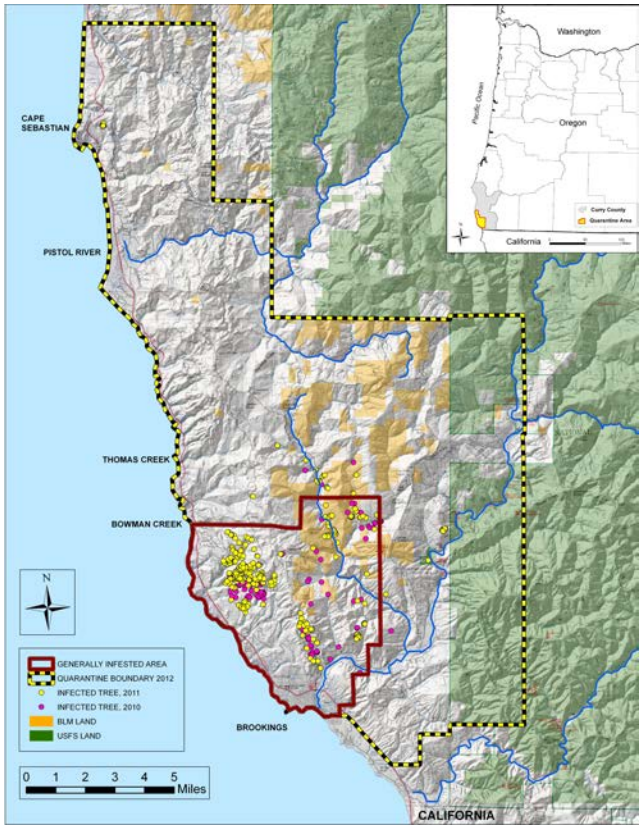

Figure 2: Sudden oak death infestations discovered in 2010 and 2011 in Curry County, Oregon Forests, the new quarantine boundary (yellow outline), and the generally infested area (red outline).

Despite continued spread and intensification of disease, the program has by no means been a failure. In the ten years since first detected, sudden oak death still is confined to a relatively small quarantine area near Brookings. Compared to a similar infestation in Humboldt county California that started in 2003, the Oregon effort has reduced the amount of mortality and disease spread by 5 to 10 times (figure 3).

The total of expenditures from 2001 to 2011 was $11,434,400 for detection surveys, eradication treatments, and administration (does not include university research or the *P. ramorum* nursery program). Approximately $7.5 million of this went toward eradication treatments. Major sources of funds were: USDA-Forest Service, $7,008,400 (59%);

USDI-BLM $2,208,000 (18%); USDA-APHIS,$470,000 (4%); State of Oregon, $2,207,000 (18%), and; Private Industry, $80,000 (1%). Current annual cost of detection surveys and administration is $400,000 (Brookings staff, Salem staff, aerial surveys, lab support). Eradication treatments average approximately $2,500 / acre.

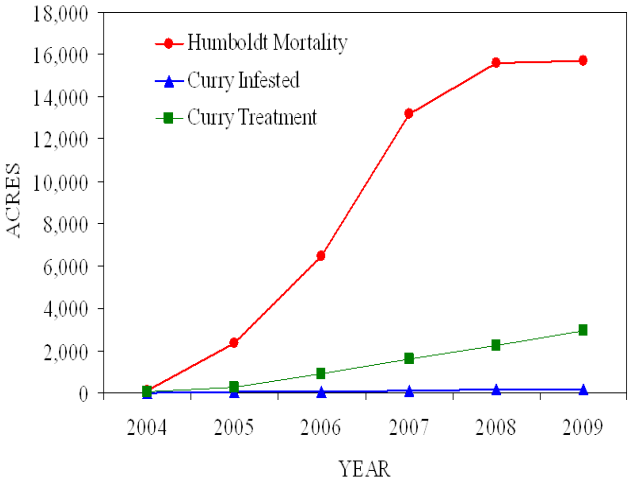

Figure 3: Cumulative area infested with *Phytophthora ramorum* and the area cut and burned in eradication treatments in Curry County, Oregon compared to the area of tanoak mortality in nearby Humboldt County, California where no eradication program was in place, 2004 to 2009.

Costs and Funding

Despite consistent high levels of support from the USDA-Forest Service, several aspects of funding have hindered the eradication effort. Many times funds that were expected early in a federal fiscal year were not available until the federal budget was officially passed, often as late as spring or summer of the following year. Unfortunately, the waiting period was during winter and spring when most disease spread occurs. At other times federal funds were available but the State could not secure the required non-federal matching funds, so we could not accept the funds. Nearly $1 million in federal funds were forfeited for this reason. In addition, when the economy declined in 2008, the State chose to offset budget reductions by returning $265,000 of State General Funds specifically allocated for sudden oak death treatments. The net effect of these funding problems was that we fell farther and farther behind in our eradication treatments, allowing sites that should have been treated promptly to carry over into the following year, creating a backlog of sites requiring treatment. Meanwhile, new, often higher priority sites (in terms of disease spread) continued to appear. In an attempt to recover lost ground and begin host removal in advance of the disease front, we applied for $4.4 million through the American Recovery and Reinvestment Act (ARRA), the so called "stimulus program". We were awarded $2.7 million (no requirement for matching funds) which finally became available in April 2010, allowing us to add staff in Brookings, resume work on the backlog of untreated sites, and complete treatment of all high priority sites identified in 2010. But it may have been too late. The previous delays had allowed the disease to intensify and spread, resulting in the dramatic increase in new infested sites discovered in 2010 and 2011.

Finding non-federal funds to match available federal dollars remains a challenge. In 2011 we were able to obtain matching funds from private landowners through 50-50 cost share program for treatments and in-kind services for work related to sudden oak death management. Even with ample matching funds, the combined state and federal fund sources were not sufficient to continue treating all sites. At the 2010 levels of disease and staffing, we estimated an annual program cost of $2-$2.5 million dollars to continue the early detection surveys and treat infested sites with 300 ft treatment buffers. By mid-2011 it was clear that the dramatic increase in disease and the attendant increase in area requiring treatment on private land would exceed available or expected funds, and a major program change would be necessary.

Changes to the Sudden Oak Death Program

The continued expansion of the disease and associated rising costs of eradication treatments have exceeded our technical and financial ability to cut and burn every new infestation. In early 2011, the Oregon SOD "task force" (ODA, ODA, USFS, & OSU) and stakeholders met and considered various options for a sustainable sudden oak death management program. Options included stopping the program altogether, establishing a broad host-free zone north of the current infestation, extensive aerial application of fungicides, an numerous variants of the existing eradication program.

With funding as a major constraint, we settled on a program with the goal of slowing spread of the disease. Pest spread models suggest that slowing spread is best accomplished by early detection and rapid suppression of new infestations that occur beyond the leading edges of the main infestation. This approach is analogous to treating spot fires when controlling wild fire. Additional benefit can be gained by reducing overall inoculum levels elsewhere within the quarantine area by destroying infected host plants. Reducing inoculum lowers the chance that disease will intensify on-site or be spread long distance naturally or by human activities.

The base function of the program and highest priority for funding is the early detection of infected trees through a variety of survey methods. Eradication treatments will be scaled to funding levels. The highest priority sites (in terms of potential for disease spread) will be treated first while other sites will remain untreated on private land. Federal agencies will continue eradication on all infested sites on their land.

Sudden oak death spreads during periods of wet weather, especially January-June. Our aim is to cut and burn infested sites prior to this period. Because all infestations will not be discovered prior to this period, treatment funds will be allocated as follows: 75% to infestations identified July – December, and; 25% for sites identified January - June. This proportion is based on timing of detections of "outlier" sites in 2009 and 2010. Treatment priorities will be assessed bi-monthly by agency staff conference (ODF, ODA, and USFS). Our current planned annual budget for the program is approximately $1 to $1.4 million.

The new program required several changes to the Oregon quarantine regulations which will become effective in February 2012. The key provisions are described below.

1. Establishes a "generally-infested area" within the quarantine boundary where *P. ramorum* has been commonly found or where the disease has persisted or intensified and complete eradication of the pathogen is impossible or impractical (figure 2). Parts of the generally infested area currently are uninfested, but these likely will become infested over time if host plants are present. The size and shape of the generally infested area will be updated periodically by the ODA and ODF depending on disease distribution and funding available for treatments, and will be available as a map or shape file on the ODA/ODF website. Within the generally infested area eradication treatments are no longer required by the State.

2. Defines two types of infested sites based on their importance for spread of disease:

A. Type 1 sites are infested sites considered to be of highest risk for spread of *P. ramorum* into previously un-infested areas. They typically are located outside of the generally infested area. The highest priority sites are those closest to or beyond the existing quarantine boundary. Eradication treatments are required: all host plants within 50 to 300 ft of infected or symptomatic plants must be cut and burned as soon as possible after the treatment area have been delimited. Cost of treatment will be borne by the State if funds are available.

B. Type 2 sites are infested sites considered to be of less risk for spread of *P. ramorum* into previously un-infested areas. Type 2 sites typically are located inside of the generally infested area. Eradication treatments are not required, but disease suppression through best management practices is encouraged. A 50-50 cost-share program may be available through the ODF to help defray costs of implementing best management practices to reduce disease spread. Host trees within a Type 2 treatment area may be used as firewood within the treatment area.

3. Allows increased utilization of tanoak within the quarantine area:

A. Inside the generally infested area, tanoak maybe used as non-commercial firewood, but it cannot leave the generally infested area.

B. Outside of the generally infested area tanoak cannot leave an infested site or eradication treatment area, but it can be

transported out of the quarantine area if from a "disease free area", which is defined as an area located more than 1/4 mile from the generally infested area or any other infested site, and which has been officially surveyed within the past 6-months and found free of *P. ramorum*.

CONCLUSIONS

Eradication of *P. ramorum* from individual infested forests sites is difficult but not impossible. The disease usually does not persist on infested sites following cutting and burning, but the pathogen frequently is present in soil several years after treatment.

Spread at the landscape continues because latency of the pathogen limits early detection and delays in completing eradication treatments allow disease to intensify and spread between the time of detection and completion of treatments.

In the wet, mild climate of Curry County, spore production has been documented year-round and disease can spread anytime suitable weather conditions occur. Delays anywhere in the detection-treatment process are very costly.

Larger treatment areas can partly compensate for not detecting all infected plants during treatment area delimitation. In several instances where infestations were detected early and eradication treatments applied promptly and over a large area (25 to 40 ac), the disease was eliminated from the site and there was no apparent spread to adjacent forests three years after treatment. Treatment area buffers of 600 ft or more from infected or symptomatic plants probably are necessary to capture these nearby infected plants.

Apparent long-distance spread has been observed with distances of 2 to 3 mi (in one case 12.5 mi) between infested sites and little evidence of infestations between them. The likelihood of long-distance spread increases with amount of source inoculum and the length of time it is present on the landscape. As untreated infestations intensifying and expand, we expect an increasing number of long-distance spread events.

Changes to the quarantine regulations reflect the financial reality of managing an expanding new disease. The initial goal of complete eradication in Curry County is unachievable. Our goal now is to slow spread by 1) early detection and rapid eradication of new infestations that are epidemiologically important; 2) reducing inoculum levels wherever practical through cost-share projects and using best management practices, and; 3) improved education and outreach to prevent spread by humans.

P. ramorum eventually may spread throughout range of tanoak and possibly farther on other host species such as rhododendron and evergreen huckleberry. Pest risk models predict that without control it could eventually spread to 19 western Oregon counties. The pathogen could kill 90% of the tanoak in Oregon and will affect several ecosystems.
As the disease spreads the quarantine area and associated regulations will expand with it. These regulations likely will increase production and shipping costs for the nursery and forest industries, especially when moving host plant material out of the quarantine area or out of state. Markets may be lost as importers of Oregon products enact their own quarantines or decide not to purchase Oregon products because of perceived risk. This already has happened to log exporters and lily bulb growers. The economic justification for continuing a slow-the-spread program is based on the value of preventing or delaying these costs (Hall, 2009).

ACKNOWLEDGMENTS

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REFERENCES

Brasier, C., Webber, J. 2010. Plant pathology: sudden larch death. Nature. 466, 824–825.

Goheen, E.M., Hansen, E.M., Kanaskie, A., McWilliams, M.G., Sutton, W. 2002. Sudden oak death caused by *Phytophthora ramorum* in Oregon. Plant Disease. 86:441.

Goheen, E.M., Hansen, E.M., Kanaskie, A., Sutton, W., Reeser, P. 2009. Persistence of *Phytophthora ramorum* after eradication treatments in Oregon tanoak forests. Pages 173-176 in Proceedings of the 4th international union of forest research (IUFRO) working party. USDA Forest Service, Gen. Tech. Rep. PSW-GTR-221. Pacific Southwest Research Station. Albany, CA.

Goheen, E.M., Kanaskie, A., Hansen, E., Sutton, W., Osterbauer, N., Reeser, P., Osterbauer, N. 2010. Eradication effectiveness monitoring in Oregon tanoak forests. Pages 233-235 in Proceedings of the Sudden Oak Death Fourth Science Symposium. USDA Forest Service. Gen. Tech. Rep. PSW-GTR-229. Pacific Southwest Research Station. Albany, CA.

Hall, K.M. 2009. Cost analysis of quarantine zone size and control policy for an invasive forest pathogen. Master's thesis. Retrieved 30 August 2010 from ScholarsArchive@OSU. http://hdl.handle.net/1957/11964

Hansen, E.M., Kanaskie, A., Prospero, S., McWilliams, M., Goheen, E.M., Osterbauer, N., Reeser, P, Sutton, W. 2008. Epidemiology of *Phytophthora ramorum* in Oregon tanoak forests. Canadian Journal of Forest Research. 38:1133-1143.

Hansen, E.M., Parke J.L., Sutton, W. 2005. Susceptibility of Oregon forest trees and shrubs to *Phytophthora ramorum*: a comparison of artificial inoculation and natural infection. Plant Disease. 89: 63-70.

McPherson, B.A., Mori, S.R., Wood, D.L., Storer, A.J., Svihra, P., Kelly, N.M., Standiford, R.B. 2005. Sudden oak death in California: Disease progression in oaks and tanoaks. Forest Ecology and Management. 213:71-89.

McPherson, B.A., Wood, D.L., Mori, S., Kelly, M., Storer, A.J., Svihra, P. Standiford, R.B. 2010. Survival of oaks and tanoaks after eight years of sudden oak death monitoring in coastal California. Forest Ecology and Management. 259: 2248-2255

Meentemeyer, R., Rizzo, D., Mark, W., Lotz, E. 2004. Mapping the risk of establishment and spread of sudden oak death in California. Forest Ecology and Management. 200(1-3):195-214.

Reeser, P., Hansen, E., Sutton, W., Kanaskie, A., Laine, J., Thompson, M., Goheen, E.M. 2009. Using rain bucket spore traps to monitor spore release during SOD eradication treatments in Oregon tanoak forests. Proceedings of the Sudden Oak Death Fourth Science Symposium, June 15-19, 2009, Santa Cruz, CA. USDA Forest Service. Gen. Tech. Rep. PSW-GTR-229. Pacific Southwest Research Station, Albany, CA

Rizzo, D.M., Garbelotto, M. 2003. Sudden oak death: endangering California and Oregon forest ecosystems. Frontiers in Ecology and the Environment. 1: 197–204.

Rizzo, D.M., Garbelotto, M, Davidson, J.M., Slaughter, G.W. 2002. *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp. and *Lithocarpus densiflorus* in California. Plant Disease. 86: 205-214.

Sutton, W., Hansen, E.M., Reeser, P.W., Kanaskie, A. 2009. Stream monitoring for detection of *Phytophthora ramorum* in Oregon tanoak forests. Plant Disease. 93:1182-1186.

Vaclavik, T., Kanaskie, A., Hansen, E.M., Ohmann, J.L., Meentemeyer, R.K. 2010. Predicting potential and actual distribution of sudden oak death in Oregon: Prioritizing landscape contexts for early detection and eradication of disease outbreaks. Forest Ecology and Management. 260(6):1026-1035.

NEW INFORMATION ON THE EPIDEMIOLOGY OF SUDDEN OAK DEATH

Ebba K. Peterson[1]

INTRODUCTION

The oomycete *Phytophthora ramorum*, causal agent of Sudden Oak Death (SOD), is an exotic pathogen threatening coastal forests of California and Oregon. Known to infect over 60 plant genera, *P. ramorum* causes non-lethal leaf blights or dieback in most hosts (APHIS 2011, Rizzo and Garbelotto 2003). On others, particularly in tanoak, *Notholithocarpus densiflorus*, and the red oaks, *Quercus* section Lobatae, the pathogen causes bleeding stem cankers and eventual death (Rizzo and Garbelotto 2003). Short distance (up to 15m) dispersal via rain splash off sporulating foliar infections has been well documented (Davidson et al. 2005). Nevertheless, the distances between new SOD sites in Oregon indicate that different dispersal mechanisms are responsible for disease spread at different scales. Long distance dispersal within Oregon forests can likely be attributed to either the movement of soil on vehicles, boots and equipment, or the transport of spores in the wind (Hansen et al. 2008). The later mechanism is unprecedented amongst forest *Phytophthora* species leading most researchers to focus their efforts on the epidemiological importance soil movement. The closely related forest pathogen *P. lateralis*, causal agent of root rot of Port-Orford cedar, is known to disperse in soil and has an expected strong association with roads and downstream of where roads and trails cross waterways (Hansen et al. 2000). *P. ramorum* can be recovered from soils at sites treated as part of the SOD eradication program (Goheen et al. 2008), as well as from streams within infested watersheds during all seasons of the year (Sutton et al. 2009).

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The spatial relationship between SOD sites to potential soil introduction points has yet to be investigated, but may indicate if roads or streams are providing opportunities for movement of inoculum. We took two approaches to assess if soil movement can explain the dispersal of *P. ramorum* in Oregon forests: 1) a landscape approach to discern if SOD sites are closer than expected to roads or streams, and 2) a local approach to assess if infection is established and spreading from understory vegetation adjacent to streams.

METHODS

Landscape Spatial Analysis

To assess the spatial relationship between roads or streams and SOD sites, we took geographic coordinates for *P. ramorum* positive locations identified between 2001 and 2010 within the Joe Hall Creek, Ferry Creek and North Fork of the Chetco watersheds. All positive coordinates were then reduced to site coordinates defined as the centroid of all points located within 60 m of one another. We performed a restricted randomization procedure to test the null hypothesis that these sites were no closer to roads or streams than would be expected by chance (spatial independence). This test compares the median distance of all SOD sites to the nearest road or stream (n = 294) to that calculated for multiple reiterations of a random dataset. Statistical likelihood of observing the true median distance under randomness was computed with a 1-tailed randomization test where pseudo-p = k/N; k = the number of random data sets which had median distances less than or equal to the true median distance to roads (those with a median distance closer than observed), N = the total number of randomizations performed (Manly 1991). Each analysis was performed separately for the road and stream layers. Distance to road or stream was calculated in ArcMap; the randomization test was performed in MATLAB.

Stream Association Study

To assess the risk of stream dispersal we hypothesized that for waterways to be source of primary inoculum and contribute to the movement of *P. ramorum* away from streams, foliar hosts must be present both adjacent to and away from waterways. These tissues must also be susceptible to infection by *Phytophthora* spp. Additionally, *P. ramorum* must be recovered more commonly from hosts within the splash or flood line than in foliage away from the water's edge due to an increased risk of exposure to inoculum.

Field surveys were performed along waterways known to contain *P. ramorum* inoculum, as detected by the stream monitoring program (Sutton et al. 2009). Two sets of transects were completed at each of the 15 surveyed locations. At each starting location we ran a 100 to 200 m long transect (hereafter called the 'main transect'), whereby in 10 m intervals the presence or absence of major riparian plant species was recorded if its canopy fell within 2 m of the stream. At each 10 m interval along the main transect, we started an additional 2 m wide, 5 m long transect perpendicular to the stream ('side transect'). In side transects we assessed only the presence of overstory tanoak, and understory tanoak or California bay laurel (*Umbellularia californica*). Understory tanoak and California bay laurel were inspected for symptoms in both transect sets, and up to five leaves per host were gathered from each transect segment. One lesion per leaf was plated in selective media for the identification of *P. ramorum* or other *Phytophthora* spp.

RESULTS

Landscape Spatial Analysis

The 294 SOD sites ranged from <1 to 610 m to the nearest road (median = 100 m), and <1 to 414 m to the nearest stream (median = 71 m). Of the 10,000 randomizations used to assess spatial dependence to roads, the average median distance to roads was 101 m; 4,733 randomizations had a median distance to road that was closer than observed. Sites were not significantly closer to roads than expected by chance (k/N = pseudo-p = 0.4733). Of the 10,000 randomizations used to assess spatial dependence to streams, the average median distance to streams was 88 m; only 14 of the 10,000 randomizations were closer to streams than observed. Sites were significantly closer to streams than expected by chance (k/N = pseudo-p = 0.0014).

Stream Association Study

California bay laurel was the most common foliar host in both transects along the stream and away from the stream. While not as common, tanoak was also present at all locations. Overstory tanoak was present in an average of 10.56% of the 10 m segments observed in the main stream transects (range by location: 0 to 61.54%), and 12.57% of all side transects (range by location: 0 to 24.36%). There was no significant difference in the abundance of understory tanoak (wilcoxon signed-rank test p = 0.7763) or overstory tanoak (wilcoxon signed-rank test p = 0.7547) between the main and side transects.

P. nemorosa was the most common *Phytophthora* species present and was recovered at 12 locations from an average of 26.5% of main transect segments and 24.9% of side transect segments in which a host was present. There was no significant difference in recovery of *P. nemorosa* between main or side transects (wilcoxon signed-rank test p = 0.5044). *P. nemorosa* was not preferentially isolated from either California bay laurel or tanoak (χ^2 = 1.79, d.f. = 1, p = 0.181).

We isolated *P. ramorum* from 28 leaves collected at 4 sites. All 4 sites were in close proximity to current, active infection. *P. ramorum* was disproportionally isolated from tanoak (93% of all *P. ramorum* isolates; χ^2 = 72.5, d.f. = 1, p < 0.0001), and side transects (9.38% of main transect segments vs. 34.21% of side transects with hosts in locations in which *P. ramorum* was recovered). Due to the small number of *P. ramorum* positive locations we lacked sufficient power to determine statistical significance between the recovery of *P. ramorum* in main versus side transects.

DISCUSSION

Roads as Pathways for Inoculum Movement

Human activates along roadways have been implicated in facilitating the introduction of invasive organisms, including well documented examples of invasive *Phytophthora* species (Jules et al. 2002, Kauffmann and Jules 2006). While movement of infested plants by people may have contributed to the initial introduction of *P. ramorum* into Oregon, there is no evidence that *P. ramorum* has continued to spread into adjacent natural ecosystems along roads. Although 50% of SOD sites were within 100 m of the nearest roadway, sites were no more likely to occur closer to roads than expected by chance. This pattern is apparent despite year round access to roads within infested areas, providing opportunity for spread in both wet and dry seasons. Our conclusion of spatial independence to roads is supported, moreover, by the lack of roadside infection in preliminary surveys that have failed to isolate the pathogen from road soils or roadside vegetation within this study area (E.Hansen, unpublished data).

Streams as Pathways for Inoculum Movement

While road dispersal cannot account for the landscape distribution of *P. ramorum* in Oregon, we did observe that SOD sites occurred significantly closer to waterways than expected by chance. We investigated whether undetected understory sporulation may be responsible for moving inoculum away from streams. While understory tanoak, the best indicator of pathogen presence, was equally as common in transects adjacent to streams as away from streams, we recovered *P. ramorum* more commonly from vegetation in side transects away from the splash or flood line. All recoveries were found in areas of known, active SOD infection, and could be attributed to sporulation from upslope, overstory trees identified in the summer of 2011. In the absence of these overstory inoculum sources we failed to recover *P. ramorum*, even immediately downstream of positive samples. These observations support our conclusion that streams are an inoculum sink rather than a source, and stream borne inoculum is not responsible for significant long distance dispersal. Alternative methods of dispersal, for example aerial dispersal, should be investigated as potential contributors to spread.

Jules et al. (2002) found that while foot and animal traffic was responsible for moving inoculum of *P. lateralis* away from streams, vehicle traffic could best explain the introduction of inoculum into new watersheds, especially early in the epidemic. As such, these authors suggested that watersheds without roads had a relatively minimal risk of exposure to inoculum (Jules et al. 2002). Our observations support our conclusion that the geographic expansion of *P. ramorum* in Oregon would not have been slowed through more active road closures. Management aimed at preventing the movement of infested soils – trail and road closures or washing stations – will be ineffective at preventing the movement of inoculum into new stands once *P. ramorum* has established regionally.

REFERENCES

APHIS. 2011. APHIS list of regulated hosts and plants associated with *Phytophthora ramorum*. Published online by APHIS-PPQ/USDA. http://www.aphis.usda.gov/plant_health/plant_pest_info/pram/downloads/pdf_files/usdaprlist.pdf.

Davidson, J.M., Wickland, A.C., Patterson, H.A., Falk, K R., Rizzo, D.M. 2005. Transmission of *Phytophthora ramorum* in Mixed-Evergreen Forest in California. Phytopathology. 95:587-596.

Goheen, E.M., Hansen, E., Kanaskie, A., Sutton, W., Reeser, P. 2008. Vegetation response following *Phytophthora ramorum* eradication treatments in Southwest Oregon Forests. Pages 301-303 In Frankel, S.J. et. al. Eds. Proceedings of the Sudden Oak Death Third Science Symposium. USDA Forest Service, Gen Tech. Rep. PSW-GTR-214. Pacific Southwest Research Station, Albany, CA

Hansen, E.M., Goheen D.J., Jules, E.S., Ullian, B. 2000. Managing Port-Orford-Cedar and the introduced pathogen *Phytophthora lateralis*. Plant Disease. 84(1):4-10

Hansen, E.M., Kanaskie, A., Prospero, S., McWilliams, M., Goheen, E.M., Osterbauer, N., Resser, P., Sutton, W. 2008. Epidemiology of *Phytophthora ramorum* in Oregon tanoak forests. Canadian Journal of Forest Research. 38:1133-1143.

Jules, E.S., Kauffman, M.J., Ritts, W.D., Carroll, A.L. 2002. Spread of an invasive pathogen over a variable landscape: A nonnative root rot on Port Orford cedar. Ecology. 83:3167-3181.

Kauffman, M.J., Jules, E.S. 2006. Heterogeneity shapes invasion: host size and environment influence susceptibility to a nonnative pathogen. Ecological Applications. 16(1):166-175.

Manly, B.F.J. Randomization and Monte Carlo methods in biology. Chapman & Hall / CRC, New York.

Rizzo, D.M., Garbelotto, M. 2003. Sudden oak death: endangering California and Oregon forest ecosystems. Frontiers in Ecology and the Environment. 1:197-204.

Sutton, W., Hansen, E.M., Reeser, P.W. 2009. Stream monitoring for detection of *Phytophthora ramorum* in Oregon tanoak forests. Plant Disease. 93:1182-1186.

CONTRIBUTED PAPERS

SPATIAL AND TEMPORAL PATTERNS OF YELLOW-CEDAR DECLINE IN BRITISH COLUMBIA

Thomas Brian Maertens[1], Amanda B. Stan[2], Claire E. Wooton[3], Lori D. Daniels[1], Brian Klinkenberg[3], and Stefan Zeglen[4]

INTRODUCTION

Patches of dead and dying yellow-cedar (*Callitropsis/Chamaecyparis nootkatensis*) occur on more than 200,000 ha in Alaska and over 45,000 ha in British Columbia (Lamb and Wurtz 2009; Westfall and Ebata 2009). In order to explain processes controlling yellow-cedar decline we summarize recent manuscripts relating to the spatial and temporal patterns of its mortality along the coast of British Columbia (BC).

Symptoms leading to the death of affected trees include formation of root nodules, wood staining, and synchronous crown loss; mortality can be episodic within a stand. However, numerous biotic agents suspected of causing decline have been excluded, including insects, viruses, and more than 90 types of fungi (Hennon et al. 1990). Instead, yellow-cedar decline is attributed to increasing late-winter temperatures. Snowpack insulates soil from below-freezing temperatures, but in some years is now absent at low elevations in late-winter as more precipitation falls as rain rather than as snow. Warming temperatures trigger dehardening of yellow-cedar leaving its shallow roots susceptible to hard frosts. Root damage leads to loss of canopy and death of the tree (Hennon et al. 2006).

METHODS

Landscape-level spatial analyses tested whether topographic variability in declining stands supports the hypothesis that low snowpack contributes to decline. We investigated temporal patterns of decline by assessing changes in the growth and mortality of yellow-cedar alongside changes in climate on the coast of BC.

Remote sensing and Geographic Information System tools were used to analyze spatial patterns of decline within an 800,000 ha study area along the mid-coast of BC. The landscape was stratified by elevation, slope aspect, and slope gradient to sample 108 ecological units with >50% yellow-cedar coverage. Fifteen topographic variables were assessed as to the presence/absence of decline, then the relative importance of contributing variables was quantified using Classification And Regression Tree analyses. The species' vulnerability within the study area was projected on the landscape (Wooton and Klinkenberg 2011).

We extracted cores through bark of 155 surviving trees and 506 standing snags in declining stands (n=10) near the north and south ends of decline in BC. Ongoing tree-ring analyses of dead trees are identifying mortality dates and quantifying decomposition rates; analyses of surviving trees identified dominant patterns of growth. Crucial to the hypothesis of decline is an abrupt injury to yellow-cedar trees, so we identified marker rings measuring less than one standard deviation from the site mean, patterns of extremely low growth consistent among trees and sites (Stan et al. 2011; Maertens and Daniels 2011).

Biologically relevant climatic variables for each sampled site were reconstructed from daily instrumental data (AHCCD 2011) and monthly climate models (ClimateWNA 2011) using linear regressions. In particular, we assessed the extent to which the frequency and intensity of late-winter thaw-freeze events and the amount of snowfall have changed from 1911-2005.

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Response function analysis (Biondi 2004) using monthly temperature, precipitation, and snowfall untangled the climate-growth relationship of surviving yellow-cedar during this period. Climate, growth responses, and the frequency of marker rings were compared during opposing phases of the Pacific Decadal Oscillation (PDO).

RESULTS

GIS and CART analyses determined that, in relation to unaffected stands of yellow-cedar, decline on the mid-coast of BC tends to occur at lower elevations, most commonly between 400 and 700m. Less important contributing factors include southwesterly aspects, proximity to the coast, and areas of less exposure and higher relief (Wooton and Klinkenberg 2011).

Preliminary tree-ring analyses identified marker rings from surviving trees and mortality dates from snags in two stands. Marker rings from four sites on the North Coast were similar to declining sites in Alaska but not expressed in trees on Vancouver Island (Stan et al. 2011). Since 1950 mortality rates and the frequency of narrow marker rings at all sites are increasing.

Consistent with hypotheses of decline, one of the most dramatic changes in climate between 1911 and 2005 is the 2˚C increase in minimum temperatures during late-winter (r=0.30, p<0.005). Now, fewer days <0˚C occur driving an increase in the frequency and intensity of post-thaw freeze events and a decrease in annual snowfall. Correlation analysis shows the strong relation of growing season temperatures (Figure 1). Response function analyses identified spring climate as the primary control on the growth of yellow-cedar.

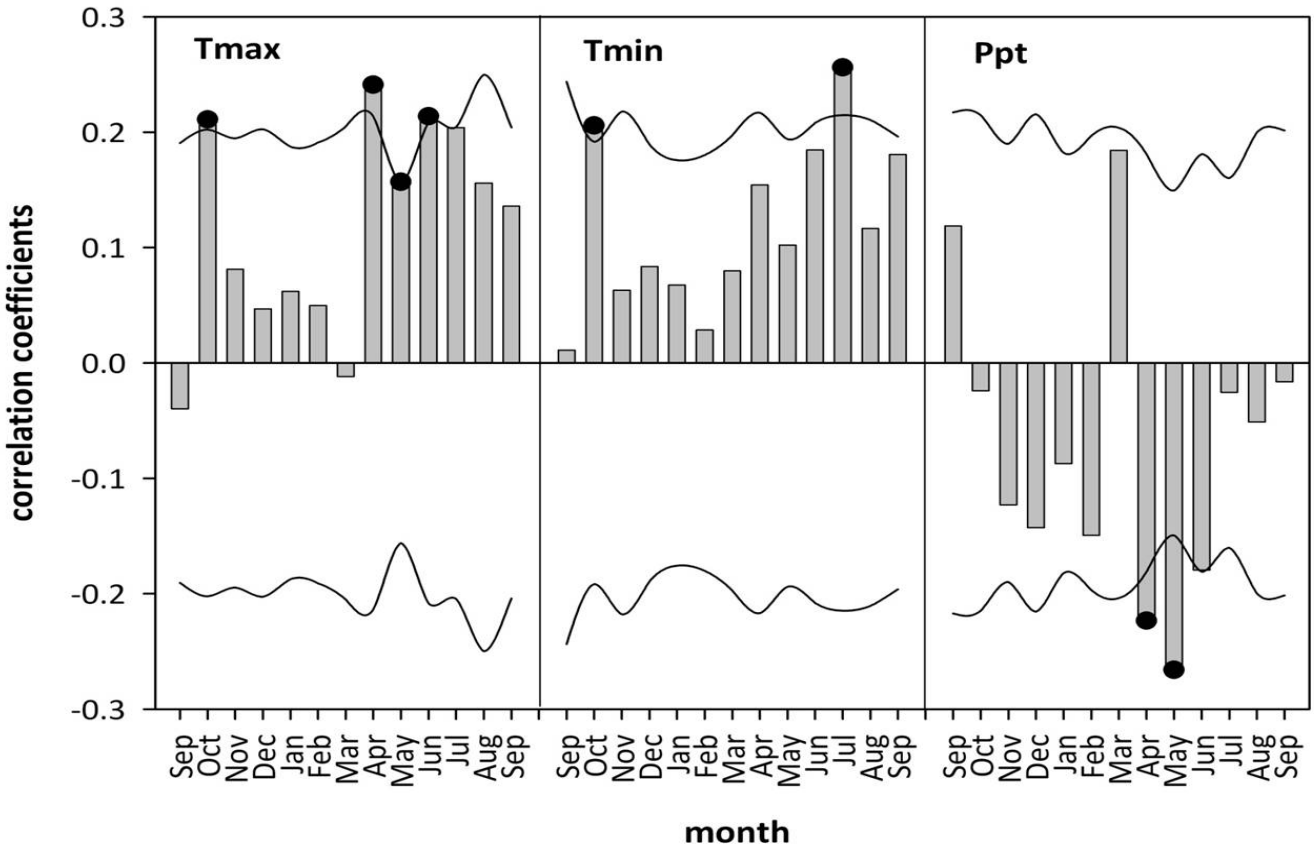

Figure 1: The climate-growth relationship of yellow-cedar from 1911 to 2005 at a declining site. Bars represent strength of correlations, squiggly lines show 95% confidence levels, and dots symbolize significant variables (α=0.05) in the model.

However, the climate and so the strength and even the direction of influence of climate on growth varied according to the PDO. In warm phases, mean late-winter temperatures >0˚C occurred in 55% of years, while in cool phases only 8% of years >0˚C ($t(36)=0.61x10^{-7}$; $p<.000001$). Importantly for yellow-cedar, the intensity and frequency of hard frosts increased and snowfall has decreased in warm phases of the PDO (Maertens and Daniels 2011). Climate-growth relations are also cyclical: more marker rings occur in cool phases of the PDO (9 rings vs. 5 in warm phases) and low temperatures in March become a limiting factor (r = -0.24 vs. -0.02 in the warm phase).

DISCUSSION

Spatial patterns of decline are consistent with hypotheses that areas of low snow accumulation are vulnerable to increased mortality, especially lower elevations (Wooton and Klinkenberg 2011). Aerial photo interpretation is being verified in 2011 and 2012 and the model being expanded to include 252 yellow-cedar stands.

As late-winter temperatures are increasing and annual snowfall is decreasing, we would expect yellow-cedar to deharden earlier coupled with a decrease in accumulated snowpack due to rain-on-snow events. Yellow-cedar trees respond to this late-winter and early-spring variability in climate, which may explain why tree-ring analyses show that in all sampled yellow-cedar stands the frequency of low-growth years increased.

CONCLUSIONS

As spatial patterns of decline coincide with areas of low snow accumulation, yellow-cedar responds to snow and hard-frosts, and mortality rates are increasing, the changing climate will almost certainly affect the distribution of yellow-cedar. With an understanding of yellow-cedar's vulnerability, land stewards can better anticipate effects of climatic change on the composition and structure of these coastal forests to enhance timber productivity and ecosystem protection. These projects help demonstrate that species-specific biological thresholds and extreme weather can combine to abruptly change forest stands (Daniels et al. 2011). We can project vulnerabilities, but expect ecological surprises.

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REFERENCES

AHCCD (2011). Adjusted Historical Canadian Climate Data. Accessed 2/28/11 from http://www.cccma.ec.gc.ca/hccd/.

Biondi, F., Waikul, K. 2004. DENDROCLIM (2002). A C++ program for statistical calibration of climate signals in tree-ring chronologies. Computers & Geosciences. 30:303-311.

Daniels, L.D., Maertens, T.B., Stan, A.B., McCloskey, S.P.J., Cochrane, J.D., Gray, R.W. 2011. Direct and indirect impacts of climate change on forests: three case studies from British Columbia. Canadian Journal of Plant Pathology. 33(2):108-116.

Hennon, P.E., Hansen, E.M., Shaw III, C.G. 1990. Dynamics of decline and mortality of *Chamaecyparis nootkatensis* in southeast Alaska. Canadian Journal of Botany. 68:651- 662.

Hennon, P.E., D'Amore, D., Wittwer, D., Johnson, A., Schaberg, P., Hawley, G., Beier, C., Sink, S., Juday, G. 2006. Climate warming, reduced snow, and freezing injury could explain the demise of yellow-cedar in southeast Alaska, USA. World Resource Review. 18(2):427-450.

Lamb, M., Wurtz, T. (compilers). 2009. Forest Health Conditions in Alaska – 2008. USDA Forest Service Forest Health Protection Report R10-PR-20.

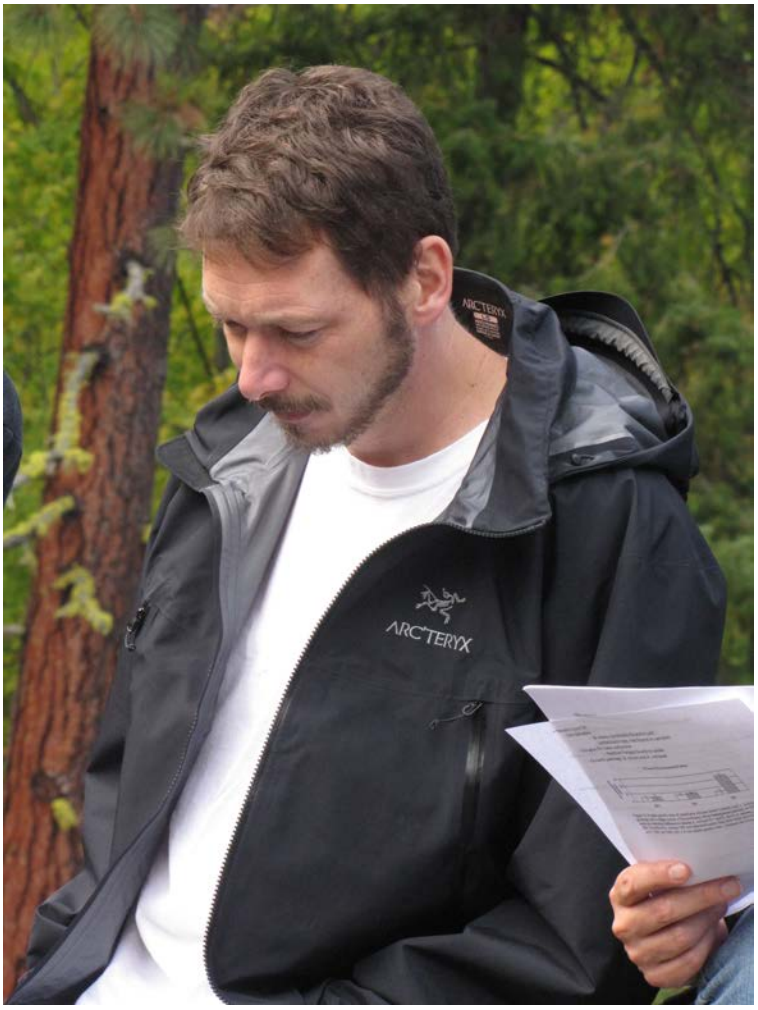
Maertens, T.B., Daniels, L.D. 2011. Climate Change and the Decline of Yellow-Cedar in British Columbia. Report to the British Columbia Ministry of Forests, Lands, and Natural Resource Operations.

Stan A.B., Maertens, T.B., Daniels, L.D., Zeglen, S. 2011. Reconstructing Population Dynamics of Yellow-Cedar in Declining Stands: Baseline Information from Tree Rings. Journal of Tree-Ring Research. 67(1):13-25.

Wang, T. 2010. Climate Western North America. Accessed 9/28.11 from http://www.genetics.forestry.ubc.ca/cfcg/ClimateWNA.html.

Westfall, J., Ebata, T. 2009. 2008 Summary of Forest Health Conditions in British Columbia. British Columbia Ministry of Forests and Range Pest Management Report #15.

Wooton, C.E., Klinkenberg, B. 2011. A Landscape Level Analysis of Yellow-Cedar Decline in Coastal British Columbia. Canadian Journal of Forest Research. 41(8): 1638-1648



HARD PINE STEM RUST HAZARD RATING AND ITS USES IN BRITISH COLUMBIA

Richard Reich[1]

INTRODUCTION

Hazard ratings for forest pathogens are relatively uncommon, and even more uncommon for pine stem rusts (Jacobi et al 1993). Predicting future stand risk to hard pine stem rusts is critical to maintaining lodgepole pine (*Pinus contorta* Dougl.) productivity in British Columbia. The purpose of this project is to develop a hazard rating for hard pine stem rusts in BC. The project itself is designed to determine the value of key predictive variables in modelling risk to rust infection. The study design utilized an empirical approach to test a wide range of variables since the key factors influencing rust risk are currently unknown. A second major purpose is to evaluate the potential for using this hazard rating data to independently verify the reliability of the rust incidence contained in the provincial silviculture database.

METHODOLOGY

Stand selection criteria included random selection of 102 pine leading stands between the ages of 10 and 20 in the Mackenzie Forest District. The survey methodology involved establishment of 20 fixed radius plots (3.99m radius) on a square grid within each plantation. Site factors (slope, elevation, aspect, and mesoslope position), soil parameters, alternate host percent cover, and ecosystem classification variables are collected at the plot level. Rust infection is recorded at the tree level for each of the hard pine stem rusts: western gall rust (WGR), caused by *Endocronartium harknessii* (J.P. Moore) Y. Hiratsuka, comandra blister rust (CBR), caused by (*Cronartium comandrae* Peck), and stalactiform blister rust (SBR), caused by *Cronartium coleosporioides* J.C. Arthur (Ziller 1974).

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RESULTS TO DATE

Site Factors

Major findings of the hazard rating analysis of the 102 young stands indicate that the mean plot elevation was the single most predictive site factor. Rust incidence was negatively correlated with increasing plot elevation for all three rusts. It is postulated that the decrease in rust incidence, associated with increasing elevation, is potentially due to a corresponding decrease in temperature, which potentially limits the rate of the infection process.

Other site factors, by themselves or in combination didn't explain much additional variation in rust incidence.

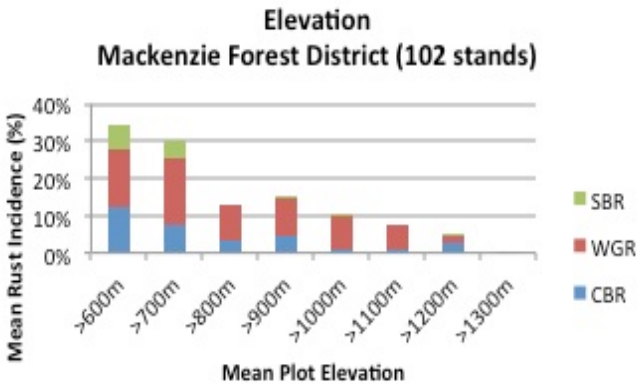

Figure 1: Mean rust incidence by 100m elevation class.

Alternate Hosts

The percent cover of alternate hosts explains a considerable additional amount of variation between sites, and is incorporated into the risk model. However it's effect is somewhat complicated,

due in part, to the fact that the rust inoculum can originate from alternate host plants outside of the plots. The difficulty associated with not knowing how far each plot is from the closest inoculum source prevents precise quantification of the effect. In a separate study, a detailed examination of the proximity relationship to the alternate host was presented for comandra blister rust and *Geocaulon lividum* using repeated annual measurement data from a long term trial (Reich 2011). Figure 2 shows an example of this proximity effect for one of the three trial sites as an example of how important this effect is. At the Holy Cross plot, which was established in 2004, cumulative risk in 2011 (8 growing seasons post establishment) is close to 35% for trees within 1 m of the alternate host, 15% at 3 m away, and less than 5% at 15 m. This magnitude of difference typically overshadows the range of variation observed between ecosystem site series.

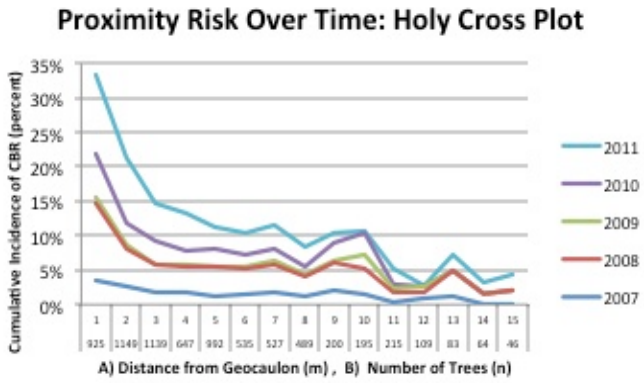

Figure 2: Cumulative incidence of infection by comandra blister rust over time, in relation to proximity to alternate host.

Ecosystem Classification

Plot level biogeoecosystem classification (BEC) was particularly useful in showing trends in rust incidence (Figure 3 & 4). Considerable variation occurred at the site series level, within specific subzone/variants. Rust incidence increased with decreasing soil moisture.

Understanding the mechanism resulting in this trend is important, but understandably complex. Alternate host presence is likely an important component. Most of the alternate hosts are relatively small plants that generally don't grow well under higher competition typically found on increasingly wet sites (Beaudry et al. 1999). However, western gall rust doesn't require an alternate host, and also exhibits higher infection on drier sites. Other variables, possibly including one or more climatic variable, may help to explain this phenomenon.

The considerable variation in rust incidence within the ecosystem classification system alone provides ample evidence of the utility of hazard rating. Using hazard ratings to guide reforestation prescriptions will ensure that high risk sites receive the additional attention that is required.

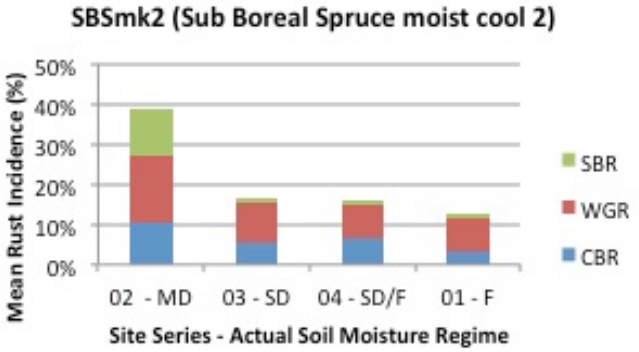

Figure 3: Rust incidence by BEC site series for the SBSmk2 subzone variant.

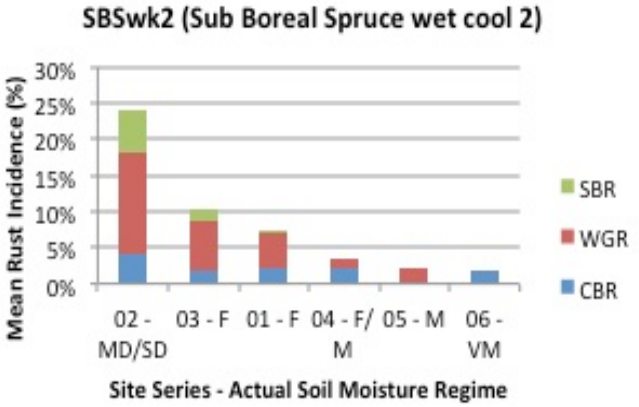

Figure 4: Rust incidence by BEC site series for the SBSwk2 subzone variant.

RESULTS VERIFICATION

In addition to providing guidance for reforestation densities and species composition, hazard rating data can be used to independently verify the reliability of the provincial silviculture database. The database known as RESULTS (**Re**porting **S**ilviculture **U**pdates and **L**and status **T**racking **S**ystem) houses tens of thousands of free growing surveys. A free growing survey is a legal requirement for every plantation established through harvesting of public

forest land, and is naturally a logical source of hazard rating information if the data is deemed reliable.

A comparison of data for stands in the RESULTS database showed that recent data quality was much improved as compared to older data (Figure 5). Annual surveyor training, which started in 2006 in the Mackenzie district, apparently resulted in improved reliability. Mean rust incidence prior to training was 22% of the hazard rating incidence, whereas for the years 2006 to 2009, post annual training, the incidence increased to 81% of the hazard rating incidence.

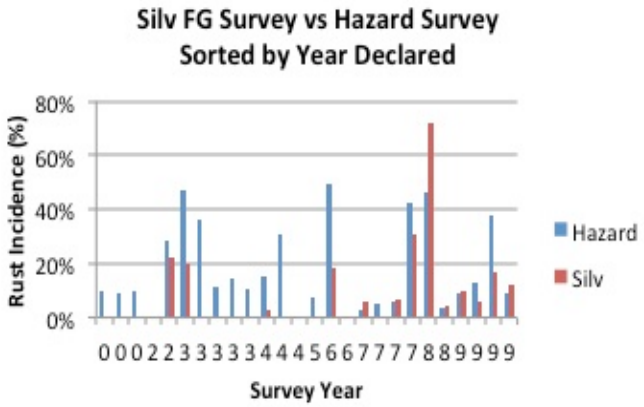

Figure 5: Comparison of accuracy of RESULTS rust incidence data collected before and after training.

This ability to independently verify the reliability of data within the RESULTS database is pivotal to developing extensive hazard data for a large and ecologically complex province such as BC. The RESULTS database may ultimately enable the development of a refined hazard rating based on a much larger dataset than could otherwise be collected through independent efforts.

RESULTS RUST MAPS

Preliminary spatial examination of the RESULTS rust incidence data, using severity rating colour theming on a relief backdrop map, indicates a good correspondence with the spatial patterns expected from the independent hazard rating data. Figure 6 shows an example map created using rust incidence data from the RESULTS database. Colour theming of rust incidence classes for plantations follows an intuitive ranking with the lowest incidence (0-5%) as green, 6-10%; yellow, 11-20%; orange, 21- 30%; red, and >30% as dark red.

These maps are being used as teaching aids for local training sessions of rust identification for silviculture surveyors. They clearly show regional trends, site factor trends (e.g. elevation), local hotspots, and validate the utility of high quality data for overview and local planning. Annual updates will incorporate changes in risk due to a number of factors such as increased surveyor reliability, potential climate change effects, etc.

SUMMARY

Hazard rating surveys provide valuable insight into future stand risk and reveal relationships with high risk sites. Expanding the rust identification training for silviculture surveyors to all high risk districts will help to increase the reliability of the corporate database. Additionally, expanding hazard rating process to other districts will provide independent verification for the use of the RESULTS data for an expanded area. These tools are being incorporated into district rust management strategies.

REFERENCES

Beaudry, L., Coupe, R., Delong, C., Pojar, J. 1999. Plant Indicator Guide for Northern British Columbia: Boreal, Sub-Boreal, and Subalpine Biogeoclimatic Zones (BWBS, SBS, SBPS, and northern ESSF), British Columbia Ministry of Forests, Research Program, Land Management Handbook 46. 139p.

Jacobi, W.R., Geils, B.W., Taylor, J.E., Zentz, W.R. 1993. Predicting the incidence of comandra blister rust on lodgepole pine: site, stand and alternate host influences. Phytopathology. 83:630-637.

Reich, R.W. 2011. Comandra blister rust resistance: a needle in the haystack? In: Fairweather, M. Comp. 2011. Proceedings of the 58th annual Western International Forest Disease Work Conference; 2011, October 4-8; Valemount, BC. US Forest Service, AZ Zone Forest Health, Flagstaff, AZ.

Ziller, W. G. 1974. The tree rusts of western Canada. Canadian Forest Service Publication 1392. 272 pp.

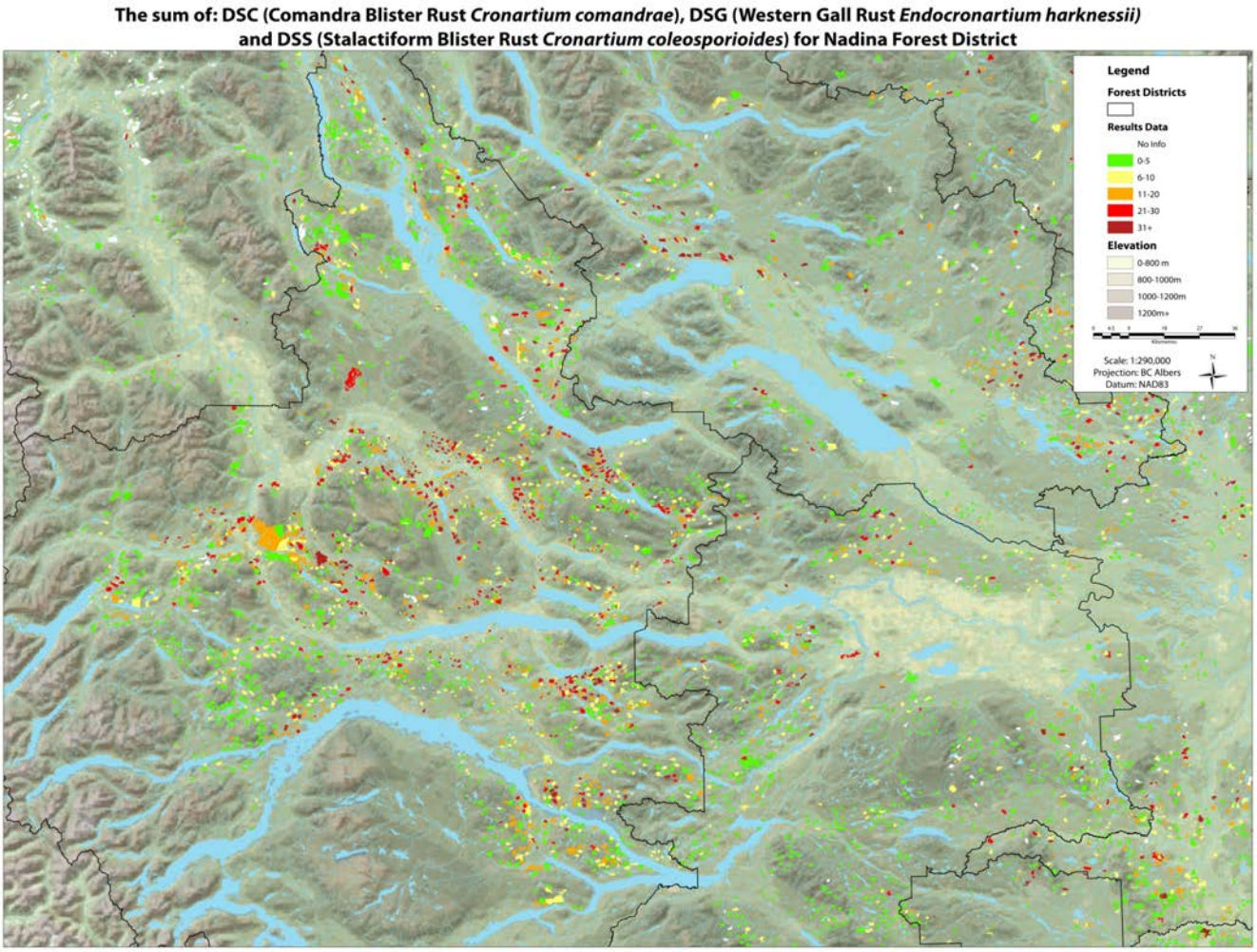

Figure 6: Example map created using rust incidence data from the RESULTS database for portions of the Nadina, Vanderhoof, and Ft St James districts for all 3 hard pine stem rusts combined.

DIEBACK OF *FRAXINUS* SPP. IN EUROPE: HISTORY AND CURRENT SITUATION

Rimvys Vasaitis[1]

Currently, severe dieback of *Fraxinus* spp. is observed in most European countries. The dieback is an emerging disease which results in massive tree mortality, and currently threatens the existence of those tree species over large parts of Europe. Characteristic for this phenomenon is a stepwise pattern of geographic spread in many directions, resembling successive expansion of an alien invasive pathogen. Dieback of *Fraxinus* has first been observed in early 1990s in north-eastern Poland and Lithuania. The disease then expanded northwards to Latvia and Estonia, until in 2007 it has finally reached mainland Finland. In Poland, during 1992-1993 the disease had been only locally observed in the north-eastern parts, then the symptoms started to appear on trees in central and southern regions, and since 1998 intensive dieback occurred all over the country. In 2002, the disease was for the first time recorded in north-eastern Germany, in 2004 in Czech Republic, and in 2005 in Austria. Subsequently, in 2007 *Fraxinus* dieback appeared in Hungary and Slovenia. In 2008 the disease has reached France, and in 2009 it was observed in Croatia and Italy. The most recent reports of ash dieback come from Belgium and the Netherlands, where it has been first observed in 2010.

Similarly, in Scandinavia, in 2002 the disease had only been observed locally in southern Sweden, but by summer 2004 it spread throughout south-east and south, towards western and central parts of the country, ultimately leading to severe symptoms and tree death almost over the whole area of species distribution. In 2003-2004, dieback of *Fraxinus* was first recorded in Denmark, and during 2005-2008 massive tree death was observed all over the country. Finally, between 2006-2008 dieback of *Fraxinus* emerged and intensified in Norway. Consequently, different stages of the epidemics are currently encountered in different parts of Europe: initially emerging phase in the south, west and north, peak in the centre, and post-epidemic period in the central east. To date (2011), ash dieback is known to occur in 24 European countries. Obviously, Europe has a new and alarming forest health problem. *Fraxinus* are important species among deciduous trees available to forest management. The use of other noble hardwoods has already been threatened by health problems: i) *Ulmus* devastated by Dutch elm disease; and ii) *Quercus* subjected to decline due to *Phytophthora*; iii) *Fagus* suffering from bark disease. So far, *Fraxinus* has been seen as a relatively disease resistant tree species and its current decline is a severe setback for noble species forestry. Moreover, *Fraxinus* is of immeasurable value for the nature conservation. Due to special site requirements, it occurs naturally on rich, moist, loamy, calcareous soils, and its stands form a unique landscape component with high biodiversity values.

The disease therefore is of immense concern for practitioners managing *Fraxinus* as forest, landscape and shade tree. For example, in Lithuania, which is one of the areas with the longest dieback history, during 1996-2002 more than 30,000 ha of stands or more than 60% of total *F. excelsior* area, has been affected by dieback, and the majority of those stands had to be clear-felled. According to Statistical Yearbook of Forestry (2009), since 2001 the total forested area covered by *F. excelsior* in Lithuania dropped from 52686 ha to 37680 ha. In 2010, *F. excelsior* was included into the Red Data Book of Sweden. Consequently, the following questions are asked by forest and nursery managers, plant health authorities, nature conservationists, land owners and general public across Europe: i) what is

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happening with *Fraxinus*, and why? ii) what are the long-term consequences of the disease? iii) will it be possible to grow *Fraxinus* in the future? iv) what has to be done, in order to secure its future as economically and ecologically valuable tree species?

The disease-causing agent was identified only in 2006. It was first described as a new fungal species, *Chalara fraxinea*. In 2009, based on morphological and DNA sequence comparisons, *C. fraxinea* was suggested to be the asexual stage (different stage of same biological species) of the ascomycete *Hymenoscyphus albidus*. This was a major surprise, as *H. albidus* is a widely spread and regionally common, saprotrophic fungus with a long history across Europe since 19th century. It has previously been known only as a harmless decomposer of shed *Fraxinus* leaves. However, in 2010 more detailed molecular investigations led to the conclusion that the disease agent belongs to a biologically different entity than *H. albidus*, and consequently, the ash dieback pathogen was described as *H. pseudoalbidus* – a species entirely new to science.

Taking the pattern of successive geographic spread of the disease across Europe into account, *H. pseudoalbidus* seems to be an alien, highly invasive and aggressive fungus, the origin of which remains unknown. Since first observations in central-east Europe, dieback of *Fraxinus* spread across the continent in all geographic directions, and recently the disease almost simultaneously invaded Finland in the north, Belgium in the west, and Italy in the south. This demonstrates wide climatic adaptability of *H. pseudoalbidus*. Yet, this raises the question whether genetic variation in geographic populations of the pathogen is a dynamic character, and if so, to what extent. For example, one could hypothesize that during initial phase and peak of the epidemics, a few aggressive clonal lines prevail in populations of the pathogen, while on weakened diseased trees (host of decreased resistance) in devastated stands there would be a higher genetic diversity. Then, it would also be of interest to compare population structures of *H. pseudoalbidus* with populations of the related saprotroph *H. albidus* in European regions under different stages of the epidemic and from different climatic zones.

Another fascinating feature of *H. pseudoalbidus* is the occurrence of this fungus in different parts of declining trees, as leaves, petioles, buds, bark, wood and roots, where it causes wilt, necroses, cankers, wood discoloration and decay. This makes *H. pseudoalbidus* unique, as no other fungus is known to cause such wide range of symptoms in such a broad range of physiologically, biochemically and physically different plant tissues. Moreover, the fungus has also been detected in symptomless *Fraxinus* leaves. The latter raises the question of whether these were initial, yet latent, infections or if *C. fraxinea* is also a natural endophyte of *F. excelsior*? This would imply universal infection biology and life style of the fungus, possessing fascinating ability to act as a pathogen, saprotroph and endophyte.

Knowledge of metabolic activity of *H. pseudoalbidus* could therefore reveal mechanisms behind its pathogenicity. In addition, the possibility cannot be excluded that the fungus is a natural endophyte of certain *Fraxinus* species outside Europe, which would provide implications regarding origin of the disease. Moreover, preliminary evaluations of clonal seed orchards of *F. excelsior* in Denmark and Sweden, and progeny trials in Denmark provided strong evidence for genetic variation in resistance against the dieback. The potential may therefore be available for breeding for resistance against the disease, if the genetic background is understood. Currently, onging tests are aimed to check if the observed patterns suggest gene variation of qualitative or quantitative nature, and how the initially observed variation between *F. excelsior* clones and progenies evolve over time.

In summary, current research activities on *Fraxinus* dieback in Europe, accomplished by numerous teams, include: i) checking the origin of *H. pseudoalbidus*; ii) assessing its invasiveness; iii) elucidating climatic factors that influence development of the disease; iv) investigating genetic structure and relationships of *H. pseudoalbidus* and *H. albidus* populations; v) infection biology and epidemiology of *H. pseudoalbidus*; vi) direct molecular disease diagnostics from plant tissue; vii) silvicultural management of declining *Fraxinus*

stands; viii) evaluation of long-term phytosanitary consequences in regions devastated by the disease; ix) checking the degree of genetic variation of susceptibility in *Fraxinus*; and x) development of programmes for breeding *Fraxinus* resistance against *H. pseudoalbidus*.

ACKNOWLEDGEMENTS

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Communicating Forest-Pest Impact Trends

Susan J. Frankel[1], David M. Rizzo[2], and Heather K. Mehl[2]

Data on the distribution and impact of forest pests is often difficult for managers and policy makers to access. Therefore, compilations of pest trend information are needed to provide a science-base for forest health management and to determine appropriate programmatic funding levels and priorities. Past pest impact trends also serve as a baseline for understanding the severity of forest insect and disease damage in response to a changing climate.

Between 1990 and 2006, the U.S. Forest Service, Forest Health Protection invested over $6 million in the Pest Trend Impact Plots project. One hundred and thirty-five permanent plot sets were established nationwide to improve pest damage estimates and develop pest models. Over 17 years, plot sets were established and re-measured in stands affected by an array of major forest pests including: Comandra blister rust (33 plots); root diseases (2,822 plots); dwarf mistletoes (409 plots); pitch canker (30 plots); white pine blister rust (98 plots); ozone (57 plots); jack pine budworm (262 plots); spruce beetle (95 plots); western spruce budworm (1023 plots) and others. Data from some of these plot sets has been analyzed and published. However, much of the data has not yet been analyzed and is currently scattered across the country under the management of various pathologists and entomologists, many of whom are nearing retirement.

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Our project, Pest Trend Impacts Plots: Analysis and Extension (WC.AR.09.03, funded by U.S. Forest Service, Forest Health Monitoring Program) aims to interpret, organize and analyze the Pest Trend and Impact Plot data to communicate pest trends to all interested parties. As data becomes available, we aim to check the data for accuracy, incorporate it into an accessible data repository and summarize findings of the study. Where possible, we intend to complete more detailed statistical analysis of individual datasets.

To date, we have gathered 23 datasets and set up a provisional searchable website (http://ucanr.org /sites/PTIPS/) with summaries and reports. We have also analyzed a 30-year, true fir dwarf mistletoe data set from the Sierra Nevada mountains examining growth and mortality trends of red fir dwarf mistletoe (*Arceuthobium abietinum* Engelmann ex Munz f.sp. *magnificae* Hawksworth & Wiens) and white fir dwarf mistletoe (*A. abietinum* f.sp. *concoloris* Hawksworth & Wiens) in even-aged, regenerating stands. The goal of this study was to determine the efficacy of pre-commercial thinning for reducing mortality and increasing radial growth, thereby off-setting potential mistletoe-associated losses. This analysis has highlighted the difficulties that arise when analyzing older datasets. The original study design considered the red and white fir dwarf mistletoe together as if they were one species. Because the species are now considered distinct, we are required to analyze them separately, making many of the plots unusable, and greatly complicating the statistical calculations.

We are eager to work with forest pathologists to provide summaries and analyze data sets so please contact us if you have suitable studies.

CLIMATE CHANGE AND FOREST DISEASES: THE ROLE OF FORESTS IN CLIMATE REGULATION AND OUR ROLE AS FOREST PATHOLOGISTS

Alex Woods[1]

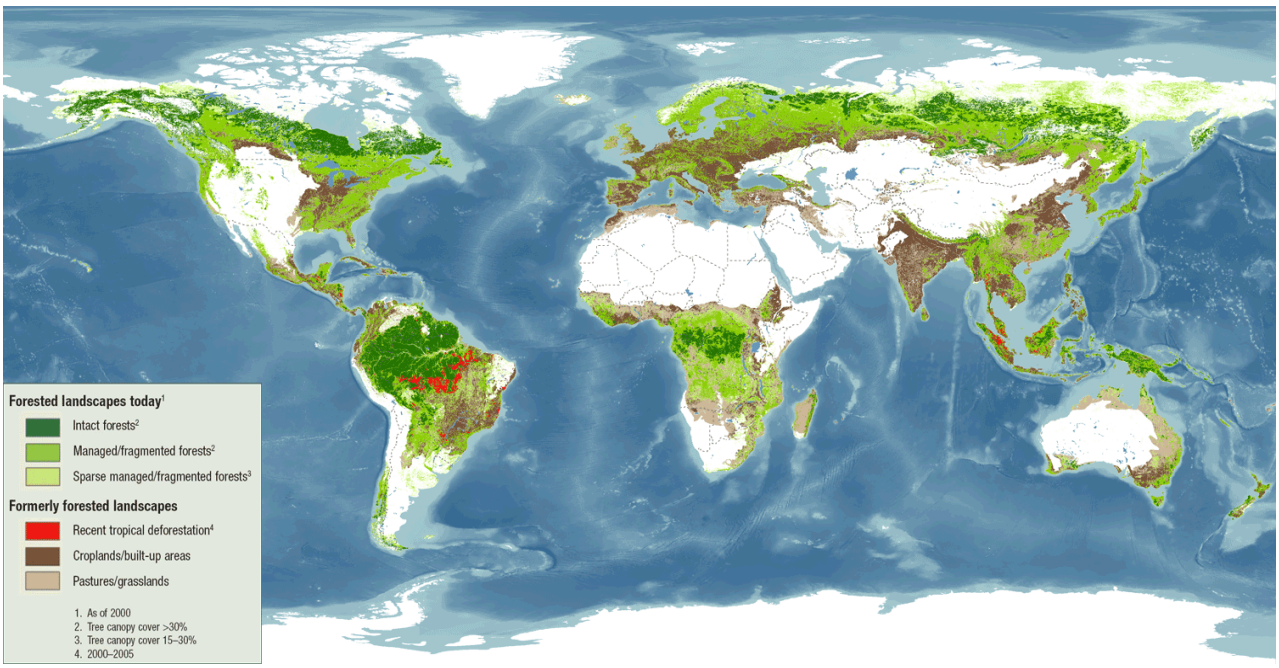

Figure 1: January 2009 - Forested and deforested landscapes of the world, developed by World Resources Institute and South Dakota State University for The Global Partnership on Forest Landscape Restoration (2009).

Forests And Their Importance To Our Collective Well-Being:

Over the past 300 years the extent of forested land area has been reduced by 40% (Millennium Ecosystem Assessment 2005) largely through human efforts to domesticate nature for increased food production (Kareiva et al. 2007). Today less than 30% of the earth's land area is forested (Millennium Ecosystem Assessment 2005). Deforestation continues to be one of the largest contributors to GHG concentrations that drive climate change representing 20% of current global emissions (IPCC 2007) which is more than that emitted by the global transportation sector with its intensive use of fossil fuels.

Most current deforestation is taking place in low-income countries; while in many higher income countries expansion is occurring (Kauppi et al 2006). The biogeophysical land-use forcing of climate in some regions may be of similar magnitude to greenhouse gas climate change (Bonan 2008).

The conversion of forests to grazed land or cultivated crops over centuries has clearly increased food production but this increase has come at a cost given the variety of services that forests provide to societies world-wide. This broad suite of services is often taken for granted. Trees and forests provide superior soil protection and improved water quality relative to other land uses (Malmer et al 2010). Globally, more than half of all wood consumption is used for basic cooking and heating, and over 2.6 billion people depend on that essential forest service (Millennium Ecosystem Assessment 2005). It has been forecast that the convergence of food, fuel and fibre markets will drive change in the world's

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remaining forests (Roberts, Don CIBC World Markets 2008) In forest dominated areas like British Columbia, Canada, forests are primarily seen as a source of revenue resulting from the manufacture of timber for construction. It's possible that this narrow view of forest values is in part responsible for Canada standing out as an exception to the general trend of increased forest growing stocking being linked to high per capita GDP as noted by Kauppi et al (2006).

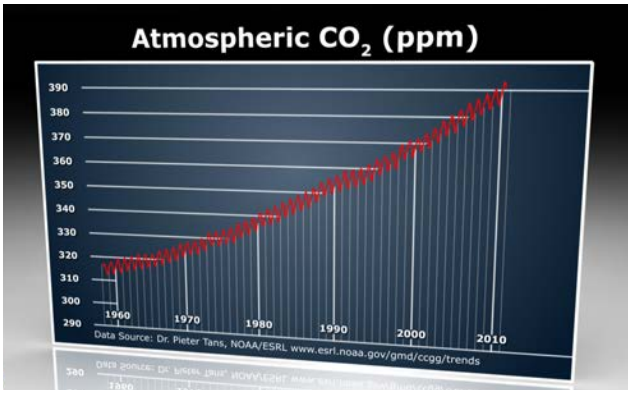

Figure 2: Keeling curve of the concentration of atmospheric carbon dioxide (CO_2) at the Mauna Loa Observatory on the Big Island of Hawaii (Climate Central 2010).

Arguably the most critical service that forests provide to global societies and most life on earth is that of being a key regulator of global hydrologic and carbon cycles (Bonan 2008). The extent to which forests influence global atmospheric CO_2 concentration, is demonstrated by the "Keeling Curve" (http://scrippsco2.ucsd.edu/program_history/keeling_curve_lessons_7.html).

This long term record clearly tracks the increasing global CO_2 concentration over the past five decades. The Keeling curve also shows, in part, how forests affect that fluctuating CO_2 concentration on an annual basis. The jagged nature of the curve is the result of seasons changing and plants either fixing CO_2 in the Northern hemisphere summer, the troughs, or not, in the Northern Hemisphere winter. The majority of land area on the planet is located in the Northern Hemisphere and the largest natural exchanges of CO_2 between the atmosphere the earth's surface occur on land (IPCC 2000). It is the vegetated land area that matters and in this regard forests are, in effect, the lungs of the planet (The Economist, Sept 23, 2010). In addition, boreal, temperate and tropical forests, store 77% of all vegetation carbon stocks yet cover only 28% of land area (IPCC 2007, Table 1).

Table 1: Global carbon stocks in vegetation and soil carbon pools down to a depth of 1 m.

Biome	Area (10^9 ha)	Global Carbon Stocks (Gt C)		
		Vegetation	Soil	Total
Tropical forests	1.76	212	216	428
Temperate forests	1.04	59	100	159
Boreal forests	1.37	88	471	559
Tropical savannas	2.25	66	264	330
Temperate grasslands	1.25	9	295	304
Deserts and semideserts	4.55	8	191	199
Tundra	0.95	6	121	127
Wetlands	0.35	15	225	240
Croplands	1.60	3	128	131
Total	15.12	466	2011	2477

Note: There is considerable uncertainty in the numbers given, because of ambiguity of definitions of biomes, but the table still provides an overview of the magnitude of carbon stocks in terrestrial systems. (IPCC Special Report on Climate Change, Land Use, Land Use Change and Forestry, Chapter 1 Global Perspective) http://www.ipcc.ch/ipccreports/sres/land_use/index.php?idp=3

Given the critical role that forests play in the most fundamental global processes it is clear that when forests are degraded through biotic and abiotic damaging events or removed through land use change, there are direct implications. Science that integrates the many interacting climate services of forests with the impacts of global climate change is lacking but this work must be conducted in order to inform climate change mitigation policy (Bonan 2008). The challenge is great, as climate change threatens forests in all biomes (Millennium Ecosystem Assessment 2005).

Forests Under the Influence of Climate Change

Forest pathogens are just one of the major forest disturbances that are being affected by changes in climate (Dale et al. 2001). The mountain pine beetle epidemic in British Columbia, Canada for example, which has been linked to climate change (Carroll et

al. 2004), has exceeded an area of cumulative attack (red and grey-attack) of 17.5 million ha (Ministry of Forests, Lands and Natural Resources 2010). More broadly, a general warming trend over the past several decades has been identified as the most probable cause behind a widespread increase in tree mortality rates in the western United States (van Mantgem et al. 2009). Increased wildfire activity in the same region of North America has been linked to earlier snow melts and longer fire seasons and both of these climatically driven phenomena trends are consistent with climate change predictions (Westerling et al. 2006). The combined impacts of increased wildfire activity, drought, insects and pathogens are transforming forested landscapes throughout the circumboreal region (Soja et al 2006) but these impacts could possibly be offset by increased net primary production (NPP) resulting from higher CO_2 concentrations (Boisvenue and Running 2006). The prevailing paradigm of current models is that increased global CO_2 concentration, and its fertilizer like effect, drives terrestrial carbon sinks, but the degree to which this remains the case is one of the greatest uncertainties in simulating the carbon cycle in the 21st century (Bonan 2008). Improving our understanding of the influence of forest pathogens on the global carbon cycle is a great challenge but it could be one of the greatest services the profession of forest pathologists could provide.

Forest Pathogens and Climate Change: Dothistroma Needle Blight; A Signal of Change?

The epidemic of Dothistroma needle blight caused by the fungus *Dothistroma septosporum* infecting lodgepole pine in northwest British Columbia, Canada, is considered one of the best supported examples of a plant disease/host relationship being affected by climate change (S. Chakraborty pers. comm. Nov 11, 2010). The foliar disease outbreak has lead to the failure of 10% of lodgepole pine plantations in the affected area and more importantly has resulted in mortality in mature lodgepole pine host trees (Woods et al. 2005). A native foliar disease killing mature native host trees suggested that a major disruption in the balance of the host/pathogen/environment relationship, or disease triangle, has taken place. Supporting information for each of the three primary factors of were well documented. In terms of the host, based on lake sediment cores collected in the area and the pollen grains found within those samples, it is known that lodgepole pine has been a component of the forests in northwest BC for the past 9000 years (Gottesfeld et al 1991). Forest management over the past several decades has increased the percentage of host in managed stands as compared to the unmanaged forest (Woods 2003). The pathogen has been recorded in the area since the mid-1960s (Molnar 1963) but recently discovered dendrochronological evidence suggests that Dothistroma is native to the area and has been affecting lodgepole pine in northwest BC for at least the past 174 years (Welsh et al 2009).

Dothistroma needle blight is one of the most thoroughly studied foliar pathogens of conifers and its epidemiology is well understood (Bradshaw 2004). Conidiospores of the fungus are released whenever temperatures are above 7C and host needles are wet (Sinclair et al 1987) Temperatures between 15-20 C combined with prolonged moisture are optimal conditions for infection (Peterson 1973, Gadgil 1974). Woods et al (2005) found a pronounced increase in mean summer precipitation during the period of time that the current epidemic had developed, 1998-2002, as compared to the 1961-1990 climate normal. They also found that the area most severely impacted by the foliar disease was the area that had experienced the greatest increase in summer precipitation, as much as 30% greater than the 1961-1990 normal. In addition, Woods et al (2005) found that previous records of Dothistroma needle blight coincided with previous years of elevated summer precipitation and that the largest spike in mean summer precipitation over the weather record period occurred during the current, most severe outbreak of the foliar disease.

The increased incidence and severity of Dothistroma needle blight attack in northwest BC has coincided with a similar pattern of damage from the pathogen in other areas of the Northern Hemisphere. Since the late 1990s there has been a dramatic increase in the extent and severity of the disease in Britain where it is causing damage to several *Pinus* species (Brown and Webber, 2008). There too, increases in disease extent and severity have been linked to increases in summer precipitation and three to five

or more consecutive days of warm rain. Over the past decade, reports of Dothistroma needle blight being identified for the first time have occurred in the following European countries: Belgium, Netherlands, Denmark, Norway, Sweden, Finland, Estonia, Lithuania, Czech Republic, Slovakia, Bosnia, The Ukraine and Turkey (pers comm. L. Jankovsky 2010 10 27). The example of the increasing extent and severity of Dothistroma needle blight and its relationship to changing weather patterns provides a clear example of what climate change could do to host pathogen relationships. The lessons learned from this example, however, have for the most part only become clear in hindsight.

Environmental Drivers of Foliar Disease

The relationships between summer precipitation, overnight minimum temperatures, and foliar fungi, are key to understanding the development of most foliar diseases. Many fungi are favoured by moist conditions during the growing season due to an enhancement of spore production and in many cases spore dispersal by rain splash (Lonsdale and Gibbs 1996). Links between increased summer precipitation and outbreaks of Dothistroma needle blight have been well documented in New Zealand (Gadgil 1977), the US Midwest (Peterson 1973) and northwest BC (Woods et al 2005). Overnight minimum temperature is also considered a strong environmental driver of foliar disease development (Coakley *et al*., 1999; Harvell *et al*., 2002). Woods (2011) has shown how both of these environmental drivers have been on an increasing trend that would favour foliar disease development in central British Columbia, Canada. Field observations appear to support the link between these climate trends and the incidence and severity of a variety of foliar diseases in that province (Woods 2011).

Environmental Drivers of Forest Declines

Increases in the extent and severity of attack of foliar diseases are typically found in association with increases in precipitation but the opposite environmental trend, that of drought and its link to wide-spread climatically driven forest die-offs are more prevalent in the literature. In times of heat stress and drought, forests are able to maintain lower surface radiative temperatures and generally more moderate environmental conditions compared to croplands because of the ability of trees and their deep roots to access deeper reservoirs of soil water (Bonan 2008). The ability of forests to continue to do so under climate change may be threatened by the severity of recent drought events which have been the result of high summer-time temperatures rather than a precipitation anomaly (Anderegg et al 2012). An increase in temperature is one of the most consistently predicted climatic changes in a CO2 enriched atmosphere (IPCC 2007). Regional warming and consequent drought stress have been suggested as the primary driver of a widespread increase of tree mortality rates in the temperate forests of the Western United States (van Mantgem et al 2009). Drought- induced water stress has also been identified as the dominant cause for an observed reduction in the biomass carbon sink of the western Canadian boreal forest which could convert these forests to net carbon sources if climate change induced droughts continue to intensify (Ma et al 2011). Drought induced, regional scale dieback of forests has emerged as a global concern that is expected to escalate under model predictions of climate change (Michaelian et al 2011). The loss of the ecosystem services associated with these global forest die-off events including the loss of sequestered carbon could exacerbate climate change through atmospheric feedbacks (Allen et al 2010).

Warming alone, in the absence of increased drought, may have one of the largest influences on the global carbon budgets of forests. Increased minimum temperatures in tropical forests are highly negatively correlated with Net Primary Productivity (NPP). As minimum temperatures increase NPP decreases and tropical forests globally become sources rather than sink for carbon (Clark et al 2003).

The Disease Triangle under the influence of Climate Change

While some areas of the globe are experiencing increases in precipitation others are experiencing the opposite. Accurately predicting changes in precipitation patterns due to global warming is simply not possible (IPCC 2007). Given the direct link between forest pathogen epidemiology and

precipitation, how can we make predictions of future disease behaviour? Concepts such as the disease triangle (Gaumann 1950) can serve as a guide, but it can also help illustrate the extent of the uncertainty. For many forest diseases we simply do not have enough detailed information on the epidemiology of the fungi and their relationship with the environment to be able to predict what may happen as that environment changes. We can recognize that when that change takes place, the long established relationships between pathogens and host will be disrupted. How those disruptions will play out and how environmental thresholds being broken will influence host/pathogen dynamics is very difficult to predict. A safe bet is that the species with the shortest life-spans, the pathogens and not the host tree species, will be favoured (Harvell et al 2002).

Forest Management in the Face of Unprecedented Uncertainty

Much of the knowledge of forests, forest management and forest pathogens has been acquired over a period of climate stability (Millar et al 2007). It has, until the last decade or so, been assumed by most forest scientists and practitioners that we live in a relatively unchanging environment. Now we know this premise no longer holds true. To accept that we simply cannot fully understand and accurately predict what will happen to forests under the influence of an unstable environment is a necessary first step. But we cannot be paralyzed by uncertainty, and we must not use uncertainty as an excuse not to act. Once we acknowledge the extent of uncertainty associated with the most basic levels of our understanding of trees and forests, we can start to build a framework from which to make decisions.

We can no longer assume that if we plant a given tree species, a species that may have lived for millennia in an area, it will survive to maturity 80 years from now. For example, at the town of Burns Lake in the central interior of British Columbia, Canada, eighty years from now the average minimum winter temperature could be 1 to 6 ^{o}C warmer, the average maximum summer temperature could be 3 to 6 ^{o}C warmer and the amount of summer precipitation could either be similar to that experienced over the climate normal of 1961-90 or 36% greater (source ClimateWNA v4.62 courtesy of V. Foord BC Forest Service). The wide range in these estimates reflects the level of uncertainty associated with future CO_2 emission scenarios but the implications of these model scenarios illustrates a range of environmental conditions that encompasses much of the environmental niche for lodgepole pine subspecies *latifolia* (Rehfeldt et al 1999). In other words, in 80 years the conditions in this location could largely be unsuitable for lodgepole pine even if the best adapted provenances were planted today (O'Neill et al 2008). More to the point, by the 2080s, the total area deemed suitable for lodgepole pine throughout western North America could be reduced to as little as 17% of its current distribution (Coops and Waring (2011).

Despite the daunting degree of uncertainty associated with climate change, forests and forest management there are still logical steps that can be taken, there are some "known knowns". We know the critical roles forests play in the fundamental carbon and hydrological cycles (Bonan 2008), and that any potential successes achieved in climate change mitigation will likely involve forests (Nabuurs et al 2007). We know that when forests are damaged or destroyed by disturbance events and agents not only is the carbon they sequestered lost to the atmosphere (Kurz et al 2008) but so too is the ability of those same forests to absorb CO_2 from the atmosphere until they are re-established. We know too, that any time there is a radical shift in climate, trees become more susceptible to pathogens and disease, regardless of the climatic zone (D. Lodge pers comm. Oct. 2010).

Given these foundational tenets there are some logical next steps. It is clear that empirical based models, for almost any biological system including forests, developed over a period of relative climate stability may not accurately predict future conditions given climate change. The environmental conditions, under which the organisms grew, as the empirically measured data that was used to develop the models was collected, no longer exist. Process based models

must instead be developed and refined as these can be more readily adapted to complex changing conditions (Korzukhin et al 1996). If we don't know for certain which disease or insect disturbance agent will be the next to benefit from the changing climate we would be wise to diversify the plantations we are currently creating (Thompson et al 2009). Diversity can be incorporated into managed stands, both within and among species as well as in terms of stand structure. Forest management will be most effectively practiced on the young managed stands where direct interventions in species selection and stand density can still be employed. Most forest and forest species will have to adapt to climate change autonomously (Spittlehouse and Stewart 2003) so we would be wise to focus our efforts on the young managed stands where we can have an influence. We should plant as wide a variety of species as is currently suitable for the local environment and add to that mix, species which over the next rotation period are forecast to be suitable in the future climate. Given the inherent risks associated with taking these chances, moving species while the environment itself is in flux, it is clear that we will need to increase monitoring (Metsaranta et al 2011). This increased monitoring will need to take place at a level of resolution that will capture changes in forest pest and pathogen behaviour while the damage levels are low so that management efforts can adapt quickly. Forestry in general will need to follow an adaptive management approach where forest managers are trained to expect the unexpected (Millar et al 2007). This will require a very different mindset than that which foresters and forestry have traditionally followed.

Maintaining healthy forests in order to help increase human resilience to climate change over this century will be increasingly important but at the same time increasingly more difficult to accomplish. As weather patterns become more unpredictable and extreme, the role of forest ecosystems in regulating both the carbon and hydrologic cycles will increasingly be seen as their most critical services to global societies. To achieve climate mitigation and adaptation goals there must be a reduction in rates of deforestation (and related emissions), increased efforts in forest restoration (http://www.ideastransformlandscapes.org/what-we-do/) and a greater emphasis directed towards maintaining today's healthy forest cover. The challenge ahead is to ensure that forests remain healthy for as long as possible in the face of the impending changes to Earth's climate so that they can remain productive and sustainable (Alfaro et al 2010). The study and practice of forestry including the fields of forest pathology, and entomology have never been in a position of greater importance to societies.

REFERENCES

Alfaro, R.I., Hantula, J., Carroll, A., and others. 2010. Forest Health in a changing environment. Pages 113 to 134 in G. Mery, et al. editors. Forests and Society – Responding to Global Drivers of Change. International Union of Forest Research Organizations (IUFRO), Vienna, Austria.

Allen C.D., Macalady, A.K., Chenchouni, H., and others. 2010. A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. Forest Ecology and Management. 259:660-684.

Anderegg, W.R.L., Berry, J.A., Smith, D.D., and others. 2012. The role of hydraulic and carbon stress in a widespread climate-induced forest die-off. Proceedings of the National Academy of Sciences. 109(1):233-237.

Boisvenue, C., Running, S.W. 2006. Impacts of climate change on natural forest productivity-evidence since the middle of the 20th century. Global Change Biology. 12:1-21.

Bonan, G.B. 2008. Forest and Climate Change: Forcings, feedbacks, and the climate benefits of forests. Science. 320:1444-1449.

Bradshaw R.E. 2004. Dothistroma (red-band) needle blight of pines and the dothistromin toxin: a review. Forest Pathology. 34:163-185.

Brown, A., Webber, R. 2008. Red band needle blight of conifers in Britain. UK Forestry Commission Research Note FCRN 002 1-7.

Carroll, A.L., Taylor, S., Régnière, J., Safranyik, L. 2004. Effects of climate change on range expansion by the mountain pine beetle in British Columbia. Pages 223-232 In T.L. Shore et al. editor(s). Mountain Pine Beetle Symposium: Challenges and Solutions, October 30-31, 2003, Kelowna, British Columbia, Canada. Information Report BC-X-399. Natural Resources Canada, Canadian Forest Service, Pacific Forestry Centre, Victoria, British Columbia.

Clark, D.A., Piper, S.C., Keeling, C.D., Clark, D.B. 2003. Tropical rain forest tree growth and atmospheric carbon dynamics linked to interannual temperature variation during 1984-2000. Proceedings of the National Academy of Sciences. 100:5852-5857.

Coakley, S.M., Scherm, H., Chakraborty, S. 1999. Climate change and plant disease management. Annual Review of Phytopathology. 37:399-426.

Climate Central. 2010. Keeling curve. Downloaded March 29, 2012 from http://www.climatecentral.org/gallery/graphics/keeling_curve/.

Coops, N.C., Waring, R.H. 2011. A process-based approach to estimate lodgepole pine (*Pinus contorta* Dougl.) distribution in the Pacific Northwest under climate change. Climatic Change. 105:313-328.

Dale V.H., Joyce L.A., McNulty S., and others. 2001. Climate change and forest disturbances. BioScience. 51(9):723-734.

Gadgil P.D. 1974. Effect of temperature and leaf wetness period on infection of *Pinus radiata* by *Dothistroma pini*. New Zealand Journal of Forest Science. 4(3):495-501.

Gadgil P.D. 1977. Duration of leaf wetness periods and infection of *Pinus radiata* by *Dothistroma pini*. New Zealand Journal of Forest Science. 7(1):83-90.

Gäumann E. 1950. Principles of plant infection. New York Hafner Publishing Company.

Global Partnership on Forest Landscape Restoration. 2009. Forest Landscape Restoration: map and analyze restoration potential – figure: map. Downloaded March 29, 2012 from http://www.ideastransformlandscapes.org/large-map/.

Gottesfeld A.S., Mathews R.W., Gottesfeld L.M.J. 1991. Holocene debris flows and environmental history, Hazelton area, British Columbia. Canadian Journal of Earth Science. 28:1583-1593.

Harvell C.J., Mitchell, C.E., Ward, J.R., and others. 2002. Climate warming and disease risks for terrestrial and marine biota. Science. 296:2158-2162.

IPCC. 2007: Climate Change 2007: Synthesis Report. Contribution of Working Groups I, II and III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change [Core Writing Team, Pachauri, R.K and Reisinger, A. (eds.)]. IPCC, Geneva, Switzerland, 104 pp.

Kareiva, P., Watts, S., McDonald, R., Boucher, T. 2007. Domesticating Nature: Shaping Landscapes and Ecosystems for Human Welfare. Science. 316: 1866-1869.

Kauppi, P.E., Ausubel, J.H., Fang, J., and others. 2006. Returning forests analyzed with the forest identity. Proceedings of the National Academy of Sciences. 103(46):17574-17579.

Korzukhin, M.D., Ter-Mikaelian, M.T., Wagner, R.G. 1996. Process versus empirical models: which approach for forest ecosystem management. Canadian Journal of Forest Research. 26(5):879-887.

Kurz, W.A., Dymond, C.C., Stinson, G. and others. 2008. Mountain pine beetle and forest carbon feedback to climate change. Nature. 452:987-990.

Lonsdale, D., Gibbs, J.N. 1996. Effects of climate change on fungal diseases of trees. Pages 1-19 In Frankland, J.C et. al. eds. 1996. Fungi and environmental change. Cambridge: Cambridge University Press.

Ma, Z., Peng, C., Zhu, Q., and others. 2012. Regional drought-induced reduction in the biomass carbon sink of Canada's boreal forests. Proceedings of the National Academy of Sciences. 109(7):2423-2427.

Malmer, A., Ardö, J., Scott, D., Vignola, R., and Xu, J. 2010. Forest cover and global water governance. Pages 75-93 in G. Mery, et al. editors. Forests and Society – Responding to Global Drivers of Change. International Union of Forest Research Organizations (IUFRO), Vienna, Austria.

Metsaranta, J.M., Dymond, C.D., Kurz, W.A., Spittlehouse, D.L., 2011. Uncertainty of 21st century growing stocks and GHG balance of forest in British Columbia, Canada resulting from potential climate change impacts on ecosystem processes. Forest Ecology and Management. 262:827-837.

Michaelian, M., Hogg, E.H., Hall, R.J., Arsenault, E. 2011. Massive mortality of aspen following severe drought along the southern edge of the Canadian boreal forest. Global Change Biology. 17:2084-2094.

Ministry of Forests, Lands and Natural Resources Operations. 2011. Facts about B.C.'s mountain pine beetle. Retreived March 29th from http://www.for.gov.bc.ca/hfp/mountain_pine_beetle/Updated-Beetle-Facts_Apr2011.pdf.

Millar, C.I., Stephenson, N.L., Stephens, S. 2007. Climate change and forest of the future: Managing in the face of uncertainty. Ecological Applications. 17(8):2145-2151.

Millennium Ecosystem Assessment. 2005. Forest and Woodland Systems. Pages 585-621 in Ecosystems and Human Well-being: Current state and trends. Island Press, Washington DC.

Molnar A.C. 1963. Annual report of the forest disease survey for British Columbia. Canadian Department of Forestry, Forest Entomology and Pathology Branch.

Nabuurs, G.J., Masera, O., Andrasko, K., and others. 2007. Forestry. Pages 541-584 In: Metz, B., et al. editors. Climate Change 2007: Mitigation. Cambridge University Press, New York, NY.

O'Neill, G.A., Hamann, A., Wang, T. 2008. Accounting for population variation improves estimates of the impact of climate change on species' growth and distribution. Journal of Applied Ecology. 45:1040-1049.

Peterson G.W. 1973. Infection of Austrian and ponderosa pines by *Dothistroma pini* in Eastern Nebraska. Phytopathology. 63:1060-1063.

Rehfeldt G.E., Ying C.C., Spittlehouse D.L., Hamilton D.A. Jr. 1999. Genetic responses to climate in *Pinus contorta*: Niche bredth, Climate Change, and reforestation. Ecological Monograph. 69(3):375-407.

Sinclair W.A., Lyon H.H., Johnson W.T. 1987. Diseases of trees and shrubs. Ithaca, New York: Cornell University Press.

Soja, A.J., Tchebakova N.M., French N.H.F. 2007. Climate-induced boreal forest change: predictions vs current observations. Global and Planetary Change. 56:274-296.

Spittlehouse, D.L., Stewart, R.B. 2003. Adaptation to climate change in forest management. BC Journal of Ecosystems and Management. 4(1):11.

Thompson, I., Mackey, B., McNulty, S., Mosseler, A. 2009. Forest Resilience, Biodiversity, and Climate Change. A synthesis of the biodiversity/ resilience/ stability relationship in forest ecosystems. Secretariat of the Convention on Biological Diversity, Montreal. Technical Series no. 43.

van Mantgem, P.J., Stephenson, N.L., Byrne, J.C., and others. 2009. Widespread Increase of Tree Mortality Rates in the Western United States. Science. 323:521-523.

Welsh, C., Lewis, K., Woods, A.J. 2009. The outbreak history of Dothistroma needle blight: an emerging forest disease in northwestern British Columbia, Canada. Canadian Journal of Forest Research. 39(12):2505-2519.

Westerling, A.L., Hidalgo, H.G., Cayan, D.R., Swetnam, T.W. 2006. Warming and earlier spring increase western US forest wildfire activity. Science. 313:940-943.

Woods, A.J. 2003. Species diversity and forest health in northwest British Columbia. The Forestry Chronicle. 79(5):892-897.

Woods, A.J., Coates, K.D., Hamann, A. 2005. Is an unprecedented Dothistroma needle blight epidemic related to climate change? BioScience. 55(9):761-769.

Woods, A.J. 2011. Is the health of British Columbia's forests being influenced by climate change? If so, was this predictable? Canadian Journal of Plant Pathology. 33(2):117-126.

THE EASTERN FILBERT BLIGHT EPIDEMIC IN THE PACIFIC NORTHWEST: SURVEY VERSUS BIOLOGY

Jay W. Pscheidt[1], Pat Grimaldi[1], and Ross Penhallegon[1]

ABSTRACT

Since its initial discovery in 1970, eastern filbert blight (*Anisogramma anomala* (Peck) E. Muller) of European hazelnut (*Corylus avellana* L.) has slowly spread through the Pacific Northwest. During this 35 year period, five different surveys have detailed the location and spread of the disease. Most surveys focused on finding diseased orchards but one focused on finding shipments of diseased nursery stock (such as ornamental contorted hazelnuts) in disease free areas. Two maps were generated using Macromedia Flash (MX 2004) that compile all of the survey data and animate disease spread over time.

One map shows the annual location of diseased hazelnut orchards and nursery stock as discovered during the surveys. An estimate of the first year of infection was made for each diseased orchard based on the observed disease severity upon discovery and adjusting for the two year life cycle of the fungus. For individual sites, this estimate indicated that first detection lagged first infection by 2 to 10 years. A second map animates the location of diseased hazelnut orchards for the year of first infection and depicts fungal spread over time. The resulting maps demonstrate a pattern of movement where the disease 'jumps' into new regions 7 to 10 years before initial detection. Due to these long distance 'jumps' and detection delays, growers are encouraged to start disease management tactics before the disease is found in their orchards.

Map animation can be found at the following address: http://oregonstate.edu/dept/botany/epp/EFB/location/map1.htm.

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THOUSAND CANKER DISEASE OF WALNUT-A VIEW FROM THE EAST

William E. Jones[1]

Thousand cankers disease (TCD) is a disease complex of *Juglans spp.* (walnut trees) that is comprised of a fungal pathogen (*Geosmithia morbida*) that is vectored by the Walnut twig borer (*Pityophthorus juglandis*) (Tisserat *et al.*, 2009a). The common name of the disease refers to the numerous, small cankers that form around the beetle galleries that given sufficient numbers can coalesce and girdle trees and branches. TCD is apparently native to the western United States and Mexico, but is not native to the Eastern U.S. The threat that TCD poses is not necessarily to its original hosts such as Arizona walnut (*Juglans major*), but to new hosts such as European walnut (*Juglans regia*) or black walnut (*Juglans nigra*) that is found throughout the eastern United States in the forms of naturally established or planted trees. Interest and concern over TCD has grown exponentially in the East following discovery of the disease near Knoxville, Tennesse (TN) in July 2010. As of 2011, TCD has been detected within the native range of *Juglans nigra* in several counties in Tennessee and Virginia and Pennsylvania (PA). One primary concern is the potential for the insect and disease to spread throughout the range of Eastern black walnut, a highly valued tree species. This poses an interesting dilemma for both regulatory and land management agencies as TCD is apparently a native disease complex, even while the disease is a non-native exotic to black walnut in its natural range (Tisserat *et al.*, 2009b). Very few good examples for successful eradication of a newly established invasive pest exist for natural ecosystems, so a general approach of "wait and see" has apparently been established as the *de facto* standard for new infestations within the native range of black walnut.

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The purpose of this paper is to summarize what is currently known about Thousand Cankers Disease infestations on black walnut in the eastern United States.

Immediately following discovery of TCD in Tennessee in 2010 a concerted effort was made between State and Federal agencies to look for the disease throughout eastern TN and begin the search for other infestations in other locales. For a county to be considered as being "TCD positive" both the insect and the fungal agent must be recovered from a declining or recently killed walnut. As of 2011, the counties where TCD is known to be found in Tennessee are: Anderson, Blount, Knox, Loudon, and Sevier (the greater Knoxville area) (Figure 1). In Virginia, TCD has been found in Chesterfield, Colonial Heights, Goochland, Hanover, Henrico, and Powhatan counties (the greater Richmond area). In the USDA Forest Service's Region 9 only Bucks County, PA is currently known to possess any infestation of TCD. Figure 2 depicts all counties that were formally surveyed in 2011 for TCD in and around the Knoxville, TN infestation. All other known infestations of TCD occur west of the Mississippi River and are outside of the scope of this paper.

Almost every road segment in eastern Tennessee was surveyed for TCD in the summer of 2011 via road survey to identify symptomatic trees (Graves, *et al.,* 2009). In addition, nearby counties in Kentucky (KY) and North Carolina (NC) were also intensively surveyed (Figure 2). In Virginia, the discovery of TCD in the Richmond area in 2011 left little time to conduct a survey this year, however, the Richmond area was intensively scouted for symptomatic trees and a trapping method and pheromone were deployed. There were five counties identified in VA in 2011 as being positively identified for being positive for TCD infestations, all within the greater Richmond area.

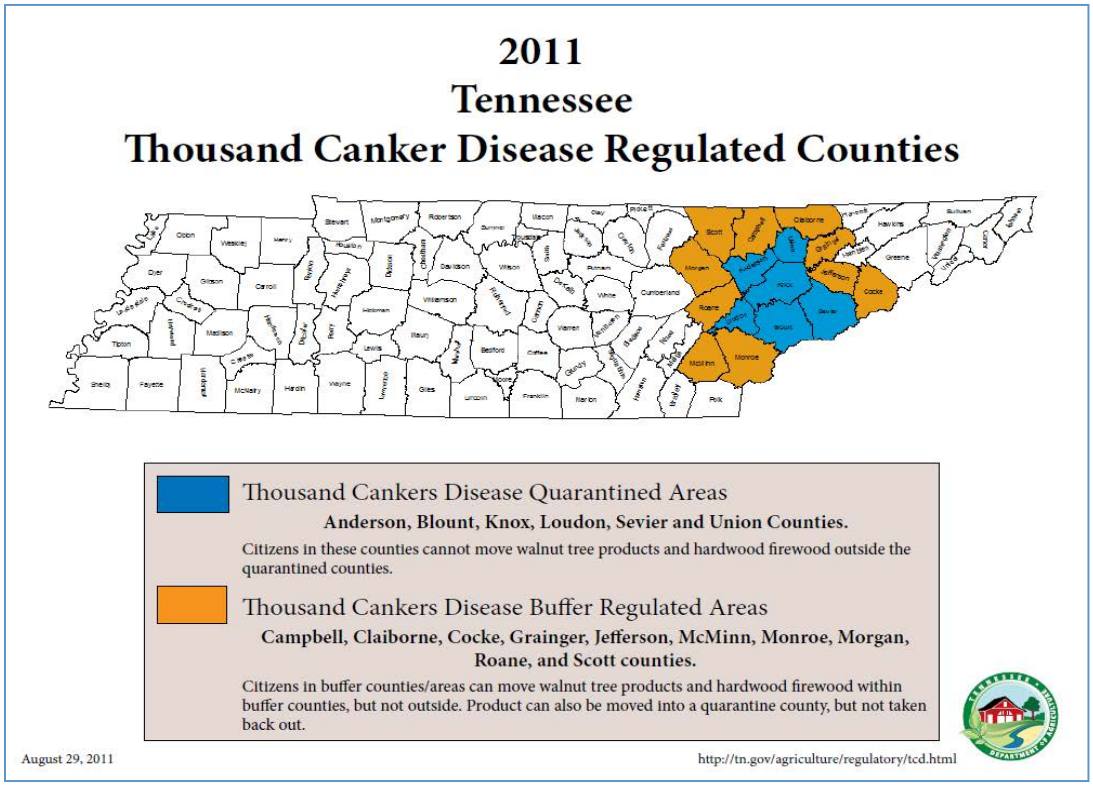

Figure 1: Extent of the TCD quarantine for walnut in Tennessee with counties impacted by the disease (blue) and buffer regulated areas.

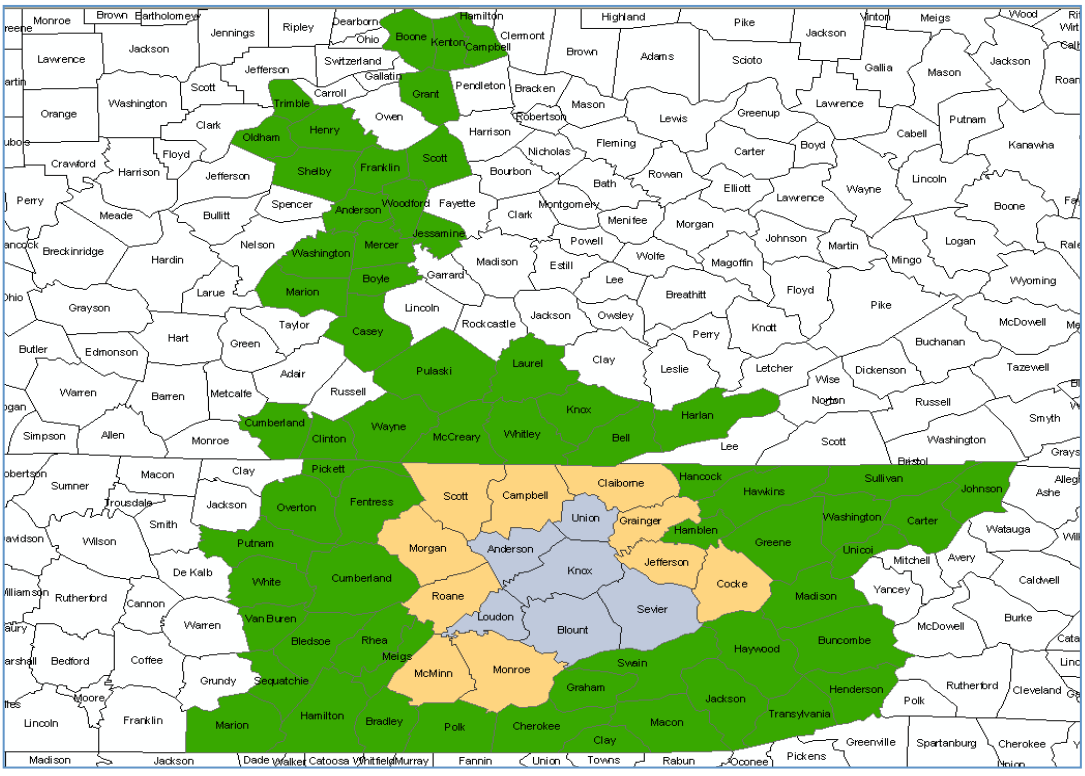

Figure 2: Counties in Kentucky, North Carolina and Tennessee that surveys for TCD were conducted in 2011 (green), buffer regulated areas (tan) and counties with known infestations (blue).

One additional county was identified as TCD positive in Bucks County, PA in 2011 as well. However, the PA survey was cut short due to severe weather and end of season effects on foliage that prevented a more thorough search for symptomatic trees.

The future of TCD in the East is far from certain but the potential for impacts are potentially very large as the composite value of black walnut in the United States is tremendous. Thousand cankers disease as a new disease poses a dire, but uncertain future for black walnuts within their native range. Indeed, even in the original disease description there was uncertainty about the role and identity of the *Fusarium spp.* Within the *Geosmithia* cankers (Tisserat, *et al.*, 2009a). Very little is known about TCD, and much basic work remains to be completed as the disease was only described in 2008 and new discoveries are occurring at a much more rapid pace than new developments in research. If the three known areas of general infestations in the East do indeed represent three distinct outbreaks of the disease, the differences in approaches the many different State and Federal agencies involved in control, management and research should serve as a lesson for future the handling and management of future tree diseases and offer some hope for control of the disease spread and impacts. If however, the three known infestations of TCD in the East only represent a small part of the current expanse of the disease, given reports of mortality rates for black walnut trees planted throughout the western United States, particularly in Colorado where monitoring and disease recognition has the longest records, the predicted outlook for black walnut is grim.

REFERENCES

Graves, A.D., Coleman, T.W., Flint, M.L., Seybold, S.J. 2009. Walnut twig beetle and thousand cankers disease: Field identification guide, UC-IPM Website Publication. Accessed 11/21/2009 from http://www.ipm.ucdavis.edu/PDF/MISC/thousand_cankers_field_guide.pdf

Tisserat, N., Cranshaw, W., Leatherman, D., Utley, C., Alexander, K. 2009a. Black walnut mortality caused by the walnut twig beetle and thousand cankers disease. Plant Health Progress. 8/11/09. 10 pp.

Tisserat, N., Cranshaw, W., Leatherman, D., Utley, C., and Alexander, K. 2009b. Black walnut mortality in Colorado caused by the walnut twig beetle and thousand cankers disease. Phytopathology. 99(6 Supplement):S128.

FIELD STUDIES AGREE AND EXTEND GREENHOUSE STUDY RESULTS OF HOST RESISTANCE TRIALS OF DOUGLAS-FIR TO ARMILLARIA ROOT DISEASE

Mike G. Cruickshank[1] and Barry Jaquish[2]

SUMMARY

Selection for positive breeding traits in conifers mainly considers tree height growth as the dominant trait for early selection. More recently, tree breeders recognize the need to incorporate insect and disease resistance traits into selection programs. A previous greenhouse seedling study (Cruickshank et al. 2010) indentified heritable survival traits in half-sibling Douglas-fir [Pseudotsuga menziesii var. glauca (Beissn.) Franco] families after challenge by *Armillaria ostoyae* (Romagn.) Herink.

In the current study, we field inoculated five poor and good survivors of same five families indentified from the greenhouse study (Table 1). The five chosen families existed as 22-year-old trees that were field inoculated at the root collar as with *A. ostoyae,* 15 trees per family. After five years, the tree root collars were excavated, lesions measured, and stem increment cores were taken at 1.3 m from infected and uninfected trees to determine how the trees reacted to the fungus. Tolerant families (larger lesions, families 421 and 423) had low to no growth impacts from root collar fungal damage (p=0.026) compared to families with resistance (smaller lesions). Resistant families suffered reduced stem radial growth with increasing collar damage, but had smaller lesions and lower collar girdling. One disease tolerant family limited proportional root collar girdling but not lesion area by forming vertically shaped lesions (Table 1, family 423). Trees with vertically shaped lesions would be more difficult to girdle and kill, but apparently this mechanism also has low cost to growth. Although the sample size was small, the results also suggested a tradeoff between resistance and tolerance so that larger sized lesions were evident on tolerant families and smaller lesions on resistant families.

The results suggest that families respond differently to disease and considering resistance and tolerance in programs of multi-trait index selection might be warranted. Disease tolerance is not well studied in conifers, but the combination of both resistance and tolerance traits may help to maximize both survival and growth from enemy threat at the stand or landscape level.

REFERENCES

Cruickshank, M.G., Jaquish, B. Nemec, A.F.L. 2010. Resistance of half-sib interior Douglas-fir families to *Armillaria ostoyae* in British Columbia following artificial inoculation. Canadian Journal of Forest Research. 40: 155-166.

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Table 1: Root collar reactions for half-sibling Douglas fir seedlings inoculated with *A. ostoyae.*

Half –sib family	421	422	423	514	620
Number of field trees (healthy, infected)	5, 14	4,7	6, 13	9, 4	6,6
Mean proportionate collar girdling (min, max)	0.35 (0.01, 0.6)	0.06 (0.01, 0.14)	0.2 (0.01, 0.7)	0.15 (0.01, 0.4)	0.12 (0.03, 0.3)
Mean collar lesion area (cm^2) (min, max)	147.5 (2.0, 363.7)	18.2 (0.7, 42.1)	175.2 (7.9, 552.6)	43.03 (3.6, 138.5)	21.59 (1.8, 52.4)
Proportion of 6-year-old greenhouse seedlings surviving 3 years post *A. ostoyae* inoculum placement	0.12	0.47	0.35	0.07	0.44

INFLUENCE OF ROOT DISEASE ON POTENTIAL FIRE BEHAVIOR IN AN EASTERN WASHINGTON PONDEROSA PINE FOREST

Nathan Johnson[1] and Robert Edmonds[1]

ABSTRACT

Root disease and wildfire are important disturbance agents in western North American forests. This research quantifies the effect of Armillaria root disease on potential wildfire behavior in ponderosa pine (*Pinus ponderosa*) forests near Glenwood, Washington. Fire behavior was modeled using the Fuel Characteristic Classification System (FCCS) based on data collected in plots with and without Armillaria and with differences in stand structure and composition. Modeling predicted that both crown and surface fire intensity would be lower in areas with root disease. This research highlights the need to consider multiple competing effects when assessing the impact of forest diseases on wildland fire.

INTRODUCTION

The effect of root disease on fire behavior has been largely unquantified. Knight (1987) developed a conceptual framework showing how disease affects forest flammability and Lundquist (2007) provided evidence from the Black Hills of South Dakota that root disease increases surface fuel loads. Fields (2003) found increased fuel loadings associated with root disease in central Oregon, and Hessburg et al. (1994) hypothesized a similar effect in eastern Washington forests. While surface fuels certainly affect potential fire behavior, root disease likely affects fire in other ways. Holah et al. (1997) showed that understory vegetation changes with root disease in western Oregon, and his may be true in Washington as well. Overstory canopy cover may be reduced reducing needle inputs, but shrub cover may increase. All these variables interact to affect potential fire behavior but most studies have looked at only a few of them.

The Fuel Characteristic Classification System (FCCS) provides a holistic approach to quantifying potential fire behavior that was unavailable to previous researchers (Ottmar et al. 2007). It allows for customization of fuelbeds at various canopy layers based on forest structure and composition variables measured in the field. This modeling approach, within the context of Knight's (1987) conceptual model, was used to test the assumption that root pathogens can increase flammability in dry forests of the western US.

METHODS

Field data were collected on forest structure and composition for 15 paired healthy and diseased plots located in actively managed ponderosa pine (*Pinus ponderosa*) stands near Glenwood, Washington. Armillaria root disease is the primary disturbance in the area. Structural data were tested using paired t-tests (alpha=0.05) and were used to generate custom fuelbeds for FCCS. Weather, slope and fuel moisture parameters were held constant. Model outputs for infected and uninfected stands were compared.

RESULTS AND DISCUSSION

Canopy cover is reduced in plots with Armillaria relative to healthy plots (Table 1). There was no evidence of increased abundance of ladder fuels (unpublished data). These results lead to a decrease in the intensity of crown fire behavior (Table 2).

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Table 1: Differences in some structural variables (1, 10, 100 and 1000 hr woody fuels, and needle litter, grass and overstory canopy cover) used for fire behavior modeling with FCCS.

	"Diseased" mean	"Healthy" mean	Difference (SD)	P-value
1 hr (0-0.6 cm) (Mg/ha)	0.50	0.28	0.22 (.27)	0.22
10 hr (0.6-2.5 cm) (Mg/ha)	2.69	2.10	0.59 (.68)	0.20
100 hr (2.5-7.6 cm) (Mg/ha)	6.94	2.45	4.49 (1.66)	**0.008**
1000 hr (>7.6 cm) (Mg/ha)	1.61	.67	.94 (.42)	**0.004**
Needle litter cover (%)	55.7	88.4	-32.7 (7.5)	**0.0003**
Grass litter cover (%)	14.5	2.9	11.6(5.4)	**0.02**
Overstory canopy cover (%)	53	78	-25 (6.43)	**0.001**

Table 2: FCCS potential crown fire behavior for healthy and Armillaria diseased ponderosa pine stands. Pacific ponderosa is the default fuelbed for the Glenwood area and is included for reference. "Overall potential" is an integrated measure of fire potential ranked on a 0-9 scale, 9 being the highest potential. The three crown-fire sub-metrics are on the same scale.

	Pacific-Ponderosa	Glenwood "Healthy"	Glenwood "Diseased"
		Crown fire potential	
Overall Potential	4	4	3
Initiation potential	3.5	2.6	2.4
Crown-crown transmissivity	7.2	8.7	6.3
Spread potential	2.7	3.2	2.2
		Surface fire potential	
Overall Potential	6	5	4
Reaction Intensity (kW/m^2)	644	965	868
Flame length (m)	1.1	1.3	1
Rate of spread (m/min)	2.7	2	1.2

Greater fuel loading was detected in 100 and 1000 hour fuels but not in 1 and 10 hour fuels. This is consistent with the results of other studies investigating the effect of disease on surface fuels (Fields 2003, Hoffman et al. 2007). Lower needle loading, and less area covered by needles on the forest floor were detected in plots with Armillaria; however, these plots had more grass litter ground cover (Table 1). The overall effect of these differences along with differences in shrub and herb layer abundance and composition (unpublished data) led to decreased potential surface fire behavior in areas with Armillaria (Table 2).

CONCLUSIONS

The effect of Armillaria on forest structure and composition is complex and so is the resulting effect on potential fire behavior. While Armillaria increases surface fuel loading this study shows that other effects, such as reducing canopy cover and needle-litter may offset increased surface fuels and act to reduce fire behavior intensity. Although this study is limited by examining only one forest type at the stand level, it does point out the need to broaden the focus of research on the effect of diseases on fire behavior. Diseases affect many of the drivers of potential fire behavior. They may increase fire

severity in one way while simultaneously decreasing it in another. Fire behavior models such as FCCS provide a tool to examine multiple competing effects and provide a more complete picture of the effect of a disease on potential fire behavior.

REFERENCES

Fields, K. 2003. Impact of Armillaria and Annosus root diseases on stand and canopy structure, species diversity, and down woody material in a central Oregon mixed-conifer forest. M.S. thesis. Oregon State University, Corvallis, OR. 166 pp.

Hessburg, P.F., Mitchell, R.G., Filip, G.M. 1994. Historical and current roles of insects and pathogens in eastern Oregon and Washington forested landscapes. USDA, Forest Service, General Technical Report PNW-327. Pacific Northwest Research Station, Portland, OR. 72 pp.

Hoffman, C., Mathiasen, R., Sieg, C.H. 2007. Dwarf mistletoe effects on fuel loadings in ponderosa pine forests in northern Arizona. Canadian Journal of Forest Resources. 37:662-670.

Holah, J. C., Wilson, M.V., Hansen, E.M. 1997. Impacts of a native root-rotting pathogen on successional development of old-growth Douglas fir forests. Oecologia. 111:429-433.

Knight, D.H. 1987. Parasites, lightning, and the vegetation mosaic in wilderness landscapes. Pages 59-83 in MG Turner (ed) Landscape Heterogeneity and Disturbance. Springer-Verlag, New York.

Lundquist, J.E. 2007. The relative influence of disease and other small-scale disturbances on fuel loading in the Black Hills. Plant Disease. 91(2):147-152.

Ottmar, R.D., Sandberg, D.V., Riccardi, C.L., Prichard, S.J. 2007. An overview of the fuel characteristic classification system—quantifying, classifying, and creating fuelbeds for resource planners. Canadian Journal of Forest Research. 37:1–11.

POPULATION GENETIC ANALYSIS OF *LEPTOGRAPHIUM LONGICLAVATUM* A PATHOGEN ASSOCIATE WITH THE MOUNTAIN PINE BEETLE *DENDROCTONUS PONDEROSAE*

L. Farfan[1], C. Tsui[1], Y. El-Kassaby[1], and R. Hamelin[1]

The mountain pine beetle (MPB) and its fungal symbiont (*Leptographium longiclavatum*) have destroyed over 16 million ha of pine forests in Canada, the largest epidemic in recorded history. Fungal symbionts could play an important role in epidemics by reducing tree defence response following beetle colonization. We investigated the genetic structure of *L. longiclavatum* isolated from various populations in western North America using microsatellite markers. Based on Bayesian clustering inference, we found that there are two clusters that are concordant with geographic origin. One cluster comprises individuals from Northern sites where the beetle-fungus complex has recently established, and a second cluster is found along the Rocky Mountains. This distribution pattern is best explained by geographic origin and is concordant with the patterns observed in the beetle and with *Grosmannia clavigera*, another important, pathogenic fungal symbiont of the MPB. The general agreement in north-south differentiation of *L. longiclavatum* and *G. clavigera* populations, as well as the MPB suggests the dependence of fungal dispersal on their bark beetle vector and similar demographic processes in these two fungi. This information is important for disease management and surveillance.

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LANDSCAPE PATTERNS OF BALSAM WOOLLY ADELGID OCCURRENCE & SUBALPINE FIR MORTALITY, OLYMPIC PENINSULA, WA

Karen M. Hutten[1], Christian E. Torgersen[2], Andrea Woodward[3], Robert E. Kennedy[4], and Justin Braaten[4]

INTRODUCTION

Subalpine fir (*Abies lasiocarpa* [Hook.] Nutt.) trees are experiencing visible decline on the Olympic Peninsula, WA (USA). Annual aerial detection surveys (ADS) conducted jointly by Washington State Department of Natural Resources (DNR) and the United States Forest Service (USFS) have identified extensive areas of defoliation and mortality in the subalpine fir zone and attribute this decline to infestations of an exotic insect, the balsam woolly adelgid (BWA) (*Adelges piceae* [Ratzeburg]) (Ciesla 2006; USDA 2010). As many as 79-98% of true fir trees (*Abies* spp.) have been killed in some locations of the Appalachian Mountains and Cascade Range since the introduction of BWA to North America from Europe in the early 1900's (Mitchell and Buffam 2001; Witter and Ragenovich 1986). A similar pattern of adelgid-induced decline may be occurring on the Olympic Peninsula since the first documented sighting in 1969.

Tree mortality is complex, and involves multiple predisposing, inciting and contributing factors that interact across scales (Manion 1981; Wu and Loucks 1995). Forests world-wide have been experiencing mortality due to a combination of climate factors, and insect and disease outbreaks (Berg et al. 2006; van Mantgem et al. 2009; Allen et al. 2010). On the landscape level, tree mortality can create spatial heterogeneity, diversity, and natural cycles in forest vegetation and succession that support ecosystem stability; however, a change in one or more biotic or abiotic disturbances that exceeds the historic range of variability can disrupt natural cycles and permanently alter an ecosystem (Holling et al. 2001; Seastedt et al. 2008; Turner 2010).

Climate change and exotic insects are two factors with potential to cause permanent ecosystem change. Insect-induced defoliation stresses host trees, reduces carbohydrate production and tree growth, compromises tree health, kills pre-weakened or otherwise infected trees and makes healthy trees vulnerable to attack by endemic agents (Houston 1992; McDowell et al. 2008). The etiology may not be initially clear due to numerous interacting agents and varied environmental conditions.

The balsam woolly adelgid is now a part of subalpine fir ecosystem dynamics as a slow but consistent defoliator. There are currently no effective methods for controlling BWA on a broad scale (Mitchell and Buffam 2001). Similar to the aphid, the adelgid feeds by inserting sucking mouth parts into phloem cells and injecting hormones that alter cambial cell growth (Doerksen and Mitchell 1965). The flow of water and nutrients to branches is reduced, resulting in defoliation, growth reduction, and dieback. Subalpine fir is susceptible to winter desiccation, early and late frost, abnormal warming events (Manion 1981), windthrow (Alexander et al. 1984), and infestation by bark beetles and wood decay fungi (Franklin and Mitchell 1967; Goheen and Willhite 2006); trees with decreased growth due to defoliation are more susceptible to environmental stressors and pathogens (Houston 1992).

The objectives of our research are to (1) identify environmental conditions and disturbance agents associated with subalpine fir mortality, (2) determine the distribution and role of balsam woolly

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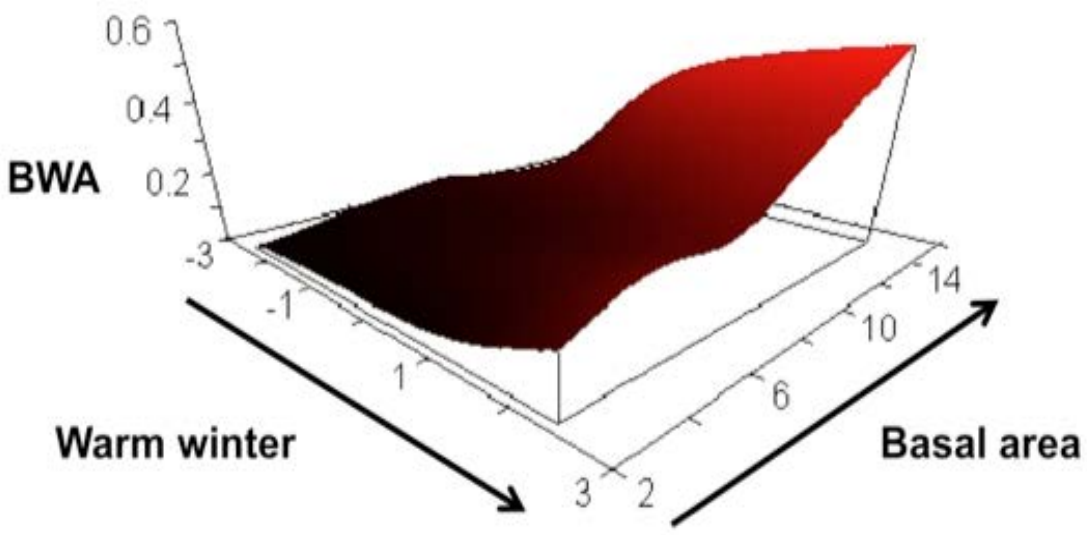
adelgid in subalpine fir decline, and (3) identify susceptible forested areas on the Olympic Peninsula. Preliminary results address objectives two and three. Initial observations and research plans are included to provide an overview of the more comprehensive mortality study currently in progress.

METHODS

This study is being conducted in the subalpine fir zone of the Olympic Peninsula, a mountainous region in northwestern Washington (USA) which is isolated from adjacent mountain ranges. Aerial detection surveys (Ciesla, 2006) and satellite imagery processed by LandTrendr (LT) (Kennedy et al. 2010) were used to identify and differentiate areas of moderate to high tree mortality from areas of no detected mortality.

A total of 105 quick-assessment plots were located within 10 sites, with approximately 12 plots per site. At each site four plots were randomly distributed within each of three polygon types: (1) ADS mortality, (2) LT vegetation decline, and (3) no detected mortality. Overlap occurred between ADS and LT polygons when plots were located in both polygon types.

Only plots with BWA sign or symptoms, and more than two tree-sized (≥ 12.7 cm dbh) subalpine fir were used to assess relationships between BWA severity and stand and environmental variables. It was assumed that BWA absence was a function of dispersal rather than tree resistance, and that plots without BWA had simply not been exposed. Of 105 plots, 54 plots fit our criteria, 41 plots had no subalpine fir, four plots had 1-2 subalpine fir only, and six plots had no BWA signs or symptoms.



Predictor	Tolerance %	Sensitivity
Basal area	14	0.6078
Warm winter	21	0.3379
Warm days	27	0.1736

Figure 1: Factors associated with severity of BWA symptoms as determined by non-parametric multiplicative regression (NPMR).

Stand data collected within the 10-m radius plots included stand age, height, elevation, slope, aspect, average BWA gout-size, seedling and sapling tally by species, and cone-bearing counts for subalpine fir. Tree-level data included tree species, status, diameter (dbh), crown class, health or decay class, presence of BWA insects, gouting and crown collapse, and other damage agents. Boles were assessed visually on the lower 2 m of the stem. USFS Forest Inventory and Analysis (FIA) protocol were followed for data collection, but not for plot design (USDA 2007).

Climate variables were downloaded from PRISM and included 30-year monthly and annual averages of minimum temperature (TMIN), maximum temperature (TMAX), and precipitation (PPT) from 1971 to 2000 (PRISM Climate Group 2004). To reduce the 39 climate variables into fewer composite variables representing dominant plot data axes, principal components analysis (PCA) was conducted using PCORD (McCune and Mefford 2011). Two composite variables were used as explanatory variables in regression analysis.

Non-parametric multiplicative regression (NPMR) (McCune and Mefford 2009) was used to determine associations between the response variable, BWA severity, and explanatory variables, which included landscape- and stand-level climate and environmental conditions. Balsam woolly adelgid severity was calculated by taking the proportion of subalpine fir trees, seedlings, and saplings with signs or symptoms of BWA and multiplying by a factor of gout-size-class 1-3 (i.e., the increase in millimeters of branch radius), after dividing by 3 to standardize the severity index to one. This measure of BWA severity allowed for a quick assessment at each plot, and was an effective representation for areas that had sparse stem infestation, variable symptom occurrence on both overstory and understory subalpine fir, and consistent plot gout size.

Non-parametric multiplicative regression was also used to investigate the relationship between proportions of subalpine fir in a plot within different decay classes, live tree health classes, and explanatory variables, which included environmental variables and mortality agents.

PRELIMINARY RESULTS

Preliminary analyses indicated that BWA was distributed throughout the subalpine fir zone on the Olympic Peninsula, with 10% of subalpine fir plots lacking symptoms or sign. Dense stem infestations of BWA were rare (*n=5*) in our study area, and trees with small gouts were generally not defoliated. BWA gout severity increased with temperature and total stand basal area (Figure 1). The preliminary results did not demonstrate a direct and significant statistical relationship between BWA and subalpine-fir deaths (plot-level data), nor did other agents prove to be significant predictors of mortality with the current data collection and analysis methods.

Disturbance agents commonly found in study plots, in addition to BWA, included mechanical damage from snow, wind and falling trees, endemic bark beetles (fir root beetle *Pseudohylesinus granulatus* [LeConte], silver fir beetle *Pseudohylesinus sericeus* [Mannerheim], western balsam bark beetle *Dryocoetes confusus* [Swaine], and fir engraver *Scolytus ventralis* [LeConte]), fungal pathogens (*Armillaria* spp. and *Heterobasidion occidentale* sp. nov.), burl-formation, and bear damage. Some mortality agents common in the Cascade Range were nonexistent or rare in subalpine fir on the Olympic Peninsula, e.g. western spruce budworm (*Choristoneura occidentalis* [Freeman]), mistletoe (*Archeuthobium abietinum* [Englman] Hawksworth & Wiens), and virulent *Armillaria* spp.

DISCUSSION

Understanding mortality dynamics requires an interdisciplinary approach for evaluating interactions among hosts, multiple disturbance agents, and climate. Multiple factors must be acknowledged and teased apart to determine their contribution to specific mortality patterns. Environmental and stand characteristics associated with BWA symptom severity provided preliminary indications of where BWA may play the largest role in tree mortality. Severity was high where warm winter temperatures may be causing earlier snowmelt and consequently greater tree volume growth. This is consistent with observations that BWA has a greater impact in productive stands, during warm years, and where trees have experienced growth release (Johnson et

al. 1963; Mitchell and Buffam 2001). Subalpine fir trees in these susceptible locations may be at risk of extirpation (Franklin and Mitchell 1967). Severely affected stands may provide opportunities for experimental management activities or aid the search for BWA-resistant trees. Our research will continue to fine-tune specific temperature and tree growth dynamics related to spatial distribution of BWA severity. Two additional explanatory variables (moisture deficit and predominant wind patterns) will be evaluated in subsequent analyses.

Additional analyses are needed to address tree mortality and potential predictors. Forested areas with subalpine fir (64 plots) appeared to have higher mortality than areas without subalpine fir (41 plots), indicating that subalpine fir is the target host for one or more damage agents. Although stand-level analyses failed to elucidate significant associations, we hypothesize tree-level analysis will help identify relationships between tree health/mortality and associated insects and disease that were not detected in stand averages. Dendrochronology and satellite imagery will also be used to investigate temporal patterns of mortality and decline along a highly visible mountain ridge in the northern part of the Olympic Peninsula. Growth rate reductions and mortality not explained by known climate and growth relationships (Peterson et al. 2002; Ettl and Peterson 1995) may be the result of increased insect or disease activity (Lemieux and Filion 2004). TimeSync (Cohen 2010), a Landsat-image-processing technique, will allow us to observe vegetation decline and recovery trends detected by Landtrendr algorithms (Kennedy et al. 2010) for 90 m^2 grid cells around our study plots. Identification of spatial and temporal trends may further help to identify mechanisms associated with forest decline (Meigs et al. 2011).

REFERENCES

Alexander, R.R., Shearer, R.C., Sheppard, W.D. 1984. Silivical characteristics of subalpine fir. USDA Forest Service, General technical report RM 115. Rocky Mountain Forest and Range Experiment Station, Fort Collins, CO. 29 pp.

Allen, C.D., Macalady, A.K., Chenchouni, H., and others. 2010. A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. Forest Ecology and Management. 259(4):660-684.

Berg, E.E., Henry, J.D., Fastie, C.L., De Volder, A.D., Matsuoka, S.M. 2006. Spruce beetle outbreaks on the Kenai Peninsula, Alaska, and Kluane National Park and Reserve, Yukon Territory: Relationship to summer temperature and regional differences in disturbance regimes. Forest Ecology and Management. 227:219-232.

Ciesla, W.M. 2006. Aerial signatures of forest insect and disease damage in the western United States. USDA Forest Service, FHTET-01-06. Forest Health Technology Team, Ft. Collins, CO.

Cohen, W.B., Yang, Z., Kennedy, R.E. 2010. Detecting trends in forest disturbance and recovery using yearly Landsat time series: 2. TimeSync – Tools for calibration and validation. Remote Sensing of Environment. 114:2911-2924.

Doerksen, A.H., Mitchell, R.G. 1965. Effects of the balsam woolly aphid upon wood anatomy of some western true firs. Forest Science. 11(2):181-188.

Ettl, G.J., Peterson, D.L. 1995. Growth response of subalpine fir to climate in the Olympic Mountains, Washington, U.S.A. Global Change Biology. 1:213-230.

Franklin, J.F., Mitchell, R.G. 1965. Successional status of subalpine fir in the Cascade Range. USDA Forest Service Research Paper PNW-46. Pacific Northwest Forest and Range Experiment Station, Portland, OR. 16 p.

Holling, C.S. 2001. Understanding the complexity of economic, ecological, and social systems. Ecosystems. 4:390-405.

Houston, D.R. 1992. A Host-stress-saprogen model for forest dieback-decline diseases. Pages 3-19 In P.D. Manion and D. Lachance eds. Forest Decline Concepts. The American phytopathological Society. St. Paul, MN.

Johnson, N.E., Mitchell, R.G., Wright, K.H. 1963. Mortality and damage to pacific silver fir by the balsam woolly aphid in southwestern Washington. Journal of Forestry. 61: 854-860.

Kennedy, R.E., Yang, Z., Cohen, W.B. 2010. Detecting trends in forest disturbance and recovery using yearly Landsat time series: 1. LandTrendr – Temporal segmentation algorithms. Remote Sensing of Environment. 114: 2897-2910.

Lemieux, C., Filion, L. 2004. Tree-ring evidence for a combined influence of defoliators and extreme climatic events in the dynamics of a high-altitude balsam fir forest, Mount Megantic, southern Quebec. Canadian Journal of Forest Research. 34(7):1436-1443.

Manion, P.D. 1981. Tree disease concepts. Prentice-Hall. Englewood Cliffs, NJ. 399 pp.

McCune, B., Mefford, M.J. 2011. PC-ORD. Multivariate Analysis of Ecological data. Version 6. MjM Software, Gleneden Beach, OR.

McCune, B., Mefford, M.J. 2009. HyperNiche. Version 2.0. MjM Software, Gleneden Beach, OR.

McDowell, N., Pockman, W.T., Allen, C.D., and others. 2008. Mechanisms of plant survival and mortality during drought: why do some plants survive while others succumb to drought? New Phytologist. 178:719-739.

Meigs, G.W., Kennedy, R.E., Cohen, W.B. 2011. A Landsat time series approach to characterize bark beetle and defoliator impacts on tree mortality and surface fuels in conifer forests. Remote Sensing of Environment. 115:3707-3718.

Mitchell, R.G., Buffam, P.E. 2001. Patterns of long-term balsam woolly adelgid infestations and effects in Oregon and Washington. Western Journal of Applied Forestry. 16(3):121-126.

Peterson, D.W., Peterson, D.L., Ettl, G.J. 2002. Growth responses of subalpine fir to climatic variability in the Pacific Northwest. Canadian Journal of Forest Research. 32:1503-1517.

PRISM Climate Group. 2010. Oregon State University, http://prism.oregonstate.edu. Map created 4 Feb 2004.

Seastedt, T.R., Hobbs, R.J., Suding, K.N. 2008. Management of novel ecosystems: are novel approaches required? Frontiers in Ecology and the Environment. 6(10):547-553.

Turner, M.G. 2010. Disturbance and landscape dynamics in a changing world. Ecology. 91(10):2833-2849.

USDA Forest Service. 2007. Forest inventory and analysis national core field guide, Volume 1: Field data collection procedures for phase 2 plots, version 4.0. Available online at http://fia.fs.fed.us/library /field-guides-methods-proc/.

USDA Forest Service and Washington Department of Natural Resources. 2010. Forest health highlights in Washington. Accessed 2/29/12 from http://www.fs.usda.gov/Internet/FSE_DOCUMENTS/stelprdb5304130.pdf.

van Mantgem, P.J., Stephenson,N.L., Byrne, J.C., Daniels, and others. 2009. Widespread increase of tree mortality rates in the Western United States. Science. 323:521-524.

Witter, J.A., Ragenovich, I.R. 1986. Regeneration of Fraser Fir at Mt. Mitchell, North Carolina, after depradations by the balsam woolly adelgid. Forest Science. 32(3):585-594.

Wu, J., Loucks, O.L. 1995. From balance of nature to hierarchical patch dynamics: a paradigm shift in ecology. The Quarterly Review of Biology. 70(4):439-466.

OBSERVATIONS ON SEEDLING AND FOREST HEALTH IN FUJIAN PROVINCE, CHINA

Will Littke[1]

Since 2004, a concentrated effort has been underway to develop the forest regeneration capacity of the forest industry in Central Fujian Province. Coastal areas suitable for warm climate Eucalyptus species (*E. grandis* and *E. urophylla*) have been planted for several decades, but inland and at elevations in excess of 1000 meters are not suited for these species. Our regeneration goals include: (1) Establish through a joint-venture forestry operation structure, facilities and cultural management practices to support seedling production within the Yongan forestry region; (2) develop training opportunities for local scientists and foresters on regeneration and forest health issues; (3) meet regeneration goals for maintaining forest cover, forest productivity, and products; and (4) document regeneration issues on the path to forest sustainability.

Photo 1: Landscape of Yongan forest region, Fujian, China.

Yongan forest region is composed of valleys and ridge tops. Intensively managed exotic plantations are in close proximity to reserve natural hardwood forests (Photo 1). Central Fujian is a dominated by steep mountain slopes, 2000+ mm of rain, and a Mediterranean climate. Summer highs range from +40 C to -8 C in winter. The region contains a mosaic of plantation forests composed of exotic Eucalyptus and native species (Photos 2 and 3).

Photo 2: *Eucalyptus grandis* plantation at approx. 300 meters age 11 years.

Photo 3: "Native" Chinese fir plantation (*Cunninghamii lanceolata*) age 40 years.

Nursery seedling production is nearly 100% container because a) fumigation is too expensive; b) few fungicides are approved for soil use; c) farmable land is too valuable and used for food production; d) nursery soil management infrastructure is lacking; and e) few commercial tree species would benefit from the bare-root option. Containerized seedling production facilities tailored to Eucalyptus can easily be managed for species like pine or fir. To date, seedling regeneration has been very successful with loblolly pine plantations (*P. teada*) exceeding 2

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meters in height at age 2 and some Eucalyptus species exceeding 4 meters in year one (Photos 4 and 5).

Photo 4: Loblolly pine in second year after planting.

Photo 5: *Eucalyptus dunnii* clone exceeding 4 meters in 1st year after planting, Fujian, China.

Regeneration and Forest Health Issues: To date the most serious issues concern cold damage to sensitive *Eucalyptus* species. In 2010-2011 a sudden cold event plunged much of the area in to record cold temperatures. Several thousand hectares were killed outright and many more were severely damaged. This has caused a reassessment of the planting guidelines for many Eucalyptus clones and has increased the research on more cold tolerant genotypes (Photo 6).

Photo 6: *Eucalyptus grandis* killed by exposure to cold events during winter 2010-2011.

Early regeneration issues with loblolly pine were caused by poor stock quality. A change to cultural management, fertilization and proper conditioning has resulted in few issues related to stock performance. Pine sawfly larvae caused defoliation has appeared in some plantations, but it is infrequent and not economical to control. Likewise, Eucalyptus seedlings can be damaged by rodent, crickets and termites, but losses do not affect stocking levels.

Soils generally contain high iron concentrations and can cause serious phosphorus deficiency. Fertilization (NPK) at the time of planting and at intermediate stand age have largely overcome these difficulties.

Due to the relatively young age of these stands (~1-10 years) we are currently seeing very little in the way of forest disease. This is typical for "new forests" which go through a period of relative quiescence with regard to forest pests. However, the future is certain to change as new pest introductions occur. There are many regeneration challenges in Fujian that remain, but none that cannot be solved without cooperative research and application of good science.

Species trials

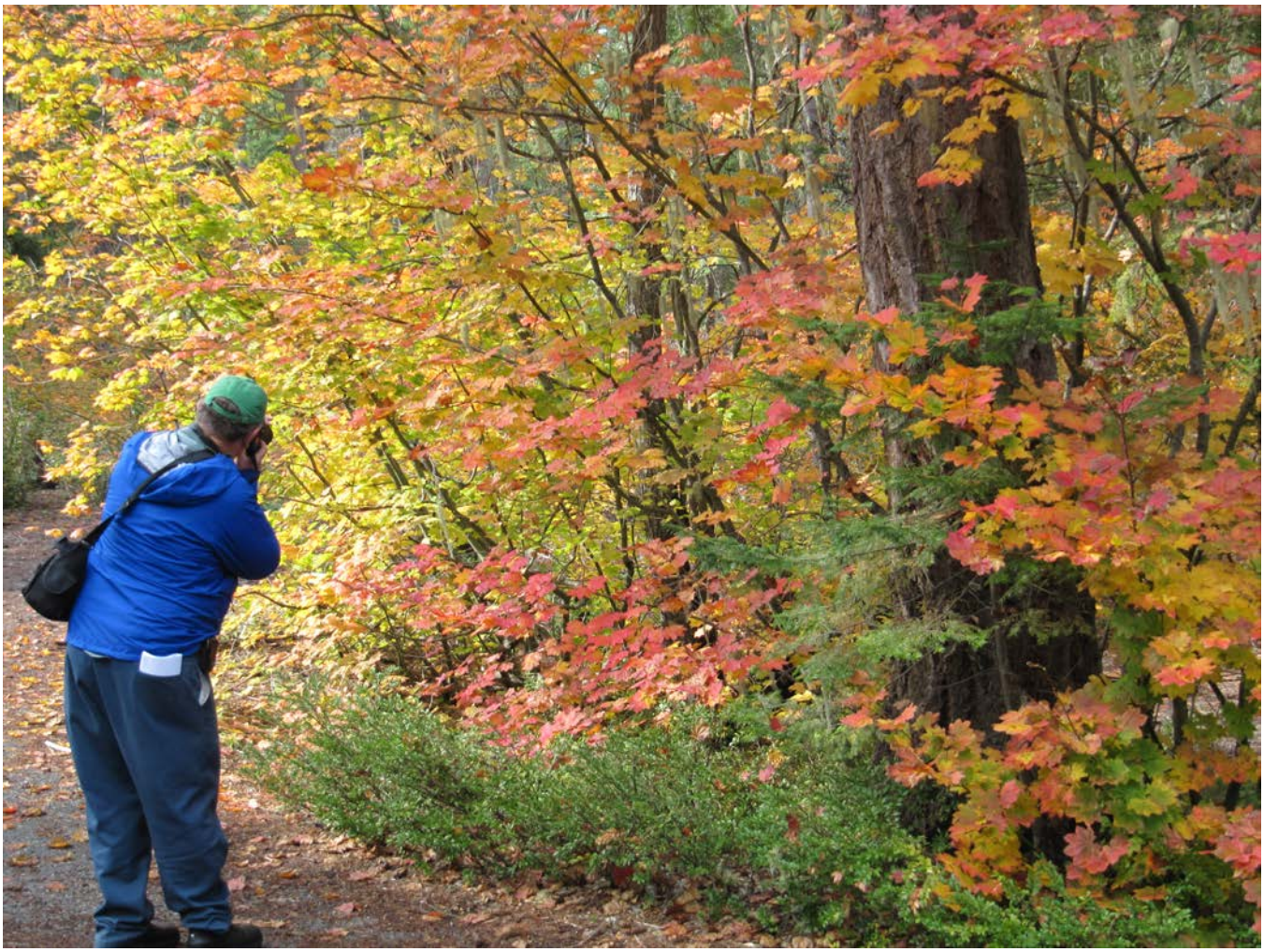
Functional balance between forestry and agriculture

Reserve management and conservation of native hardwoods

Challenges

Native and exotic pests

Establishing unit setbacks to protect water resources



POSTER PAPERS AND ABSTRACTS

DETERMINING THE GEOGRAPHIC AND HOST RANGES OF *DOTHISTROMA* SPECIES IN THE USA

J.A. Walla[1] and I. Barnes[2]

Dothistroma needle blight (DNB) has been reported or collected from at least 35 states and 19 pine hosts in the USA. However, confusion in fungal nomenclature, similarity of DNB symptoms with other diseases, and utilization of morphological traits that are now known to overlap, have resulted in not knowing where DNB pathogen species occur in the USA and on which hosts. The goal of this project is to rectify that, i.e., to determine the geographic and host ranges of *Dothistroma* species in the USA using molecular methods. We are requesting DNB samples from as wide a range of settings (e.g., geographic, environmental, stand type), and from as many host species in each setting, as possible. An APHIS-approved protocol is in place for transportation of samples for this project, which includes the use of double tyvek envelopes for shipment of samples to an already-permitted lab (North Dakota State University Plant Diagnostic Lab). Additional collection coordinators for the project are also needed. If you can help by making or arranging for collections by fall, 2012, please take or request sampling envelopes and a protocol, and contact us with any questions.

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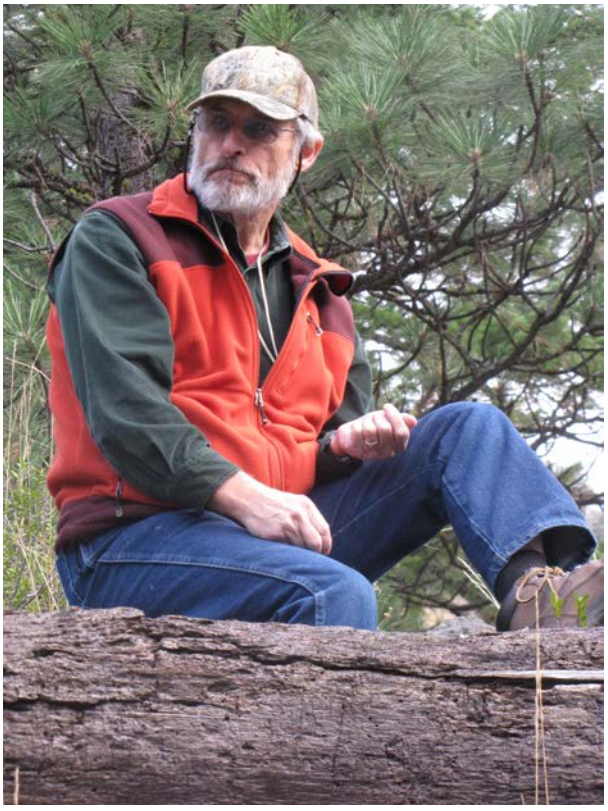

A BIOCLIMATIC APPROACH TO PREDICT GLOBAL REGIONS WITH SUITABLE CLIMATE SPACE FOR *PUCCINA PSIDII*

J.W. Hanna[1], R. Neves Graça[2], M.-S. Kim[3], A.L. Ross-Davis[1,4], R.D. Hauff[5], J.Y. Uchida[6], C.Y. Kadooka[6], M.B. Rayamajhi[7], M. Arguedas Gamboa[8], D.J. Lodge[9], R. Medel Ortiz[10], A. Lopez Ramírez[10], P.G. Cannon[11], A.C. Alfenas[12], and N.B. Klopfenstein[1]

ABSTRACT

Puccinia psidii, the cause of eucalypt-guava-'ohi'a-myrtle rust, can infect diverse plants within the Myrtaceae, and this rust pathogen has the potential to threaten numerous forest ecosystems worldwide. Known occurrence records from Brazil, Uruguay, Paraguay, Costa Rica, USA (Hawaii, Florida, and Puerto Rico), and Japan were used to develop bioclimatic models for predicting suitable climate spaces of this rust on a global scale. Four separate models were developed to predict current distribution of suitable climate space for *P. psidii* : (1) sample points based on all rust occurrence points in our dataset (All model), (2) sample points based on our dataset for occurrences of the rust on guava (*Psidium guajava*) from South America only (Guava model), (3) sample points based on our data set for occurrences of the rust on eucalypt (*Eucalyptus* spp.) from South America only (Eucalypt model), and (4) sample points based on our dataset for occurrences of the rust from multiple hosts in Hawaii only (Hawaii model). The "All model" was also projected for the 2050s (based on years 2040-2069) using the A1B SRES (Special Report on Emission Scenarios) scenario and CCCMA-CGCM 3.1 (Canadian Centre for Climate Modeling and Analysis – third generation coupled global climate model). These models can help determine points of origin, evaluate potential pathways of spread, predict areas at risk for *P. psidii* introduction, and predict potential future risks for new introductions of *P. psidii* under climate-change scenarios.

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BACKGROUND

Puccinia psidii is the cause of eucalypt /guava/'ohi'a/myrtle rust (hereafter referred to as eucalypt rust) disease of many host species in the Myrtaceae family, including guava (*Psidium* spp.), eucalypt (*Eucalyptus* spp.), rose apple (*Syzygium jambos*), and 'ohi'a (*Metrosideros polymorpha*) (Farr and Rossman 2010). First reported in 1884 on guava in Brazil (Winter 1884; MacLachlan 1938), the rust has since been detected in other South American countries (Argentina, Colombia, Paraguay, Uruguay and Venezuela), Central America (Costa Rica and Panama), Mexico, the Caribbean (Cuba, Dominica, Dominican Republic, Jamaica, Puerto Rico, Trinidad and Tobago, Virgin Islands), USA (Florida, California, Hawaii), and most recently Japan and Australia (Coutinho et al. 1998; Uchida et al. 2006; Kawanishi et al. 2009; Carnegie et al. 2010; Graça et al. 2011). Of present concern is the recent introduction of the rust pathogen to Hawaii, where it infects an endemic tree species known as 'ohi'a, the dominant tree species in Hawaii's remnant native forests. Recent analyses on the genetic diversity of *P. psidii* also

suggest several genetically distinct groups/races of this rust distributed throughout the world (Graça et al. 2011). Because only a single *P. psidii* genotype is known to exist in Hawaii (Graça et al. 2011), the introduction of additional rust genotypes or races could further threaten forests in Hawaii (Loope and La Rosa 2008). Eucalypt rust poses serious threats to several hosts in the Myrtaceae including *Eucalyptus*, a genus native to Australia and elsewhere, which is planted extensively in numerous tropical and subtropical countries (Coutinho et al. 1998; Graça et al. 2011). Previous climate-based studies have developed strong approaches for predicting areas at risk to *P. psidii* in Australia (Booth et al. 2000). Despite the potential threats to numerous forest ecosystems worldwide and the expanding geographic range of this disease, more information is needed about the potential global distribution of suitable climate space for this pathogen under present and changing climates. Bioclimatic modeling methods to predict present and future suitable climate spaces for many tree species have already been developed (Rehfeldt et al. 2006). Similar approaches can be used to predict areas where the pathogen is climatically well-adapted for comparison with areas of host (Myrtaceae) adaptation and maladaptation.

OBJECTIVES

The objectives of this study are to 1) determine the distribution of suitable climate space and potential sources for *P. psidii*; 2) predict future distributions of suitable climate space for *P. psidii* under climate-change scenarios; and 3) evaluate possible pathways of pathogen spread, and areas at risk for *P. psidii* establishment. Information from this approach will also be used to predict potential future areas at risk for *P. psidii* establishment under climate-change scenarios.

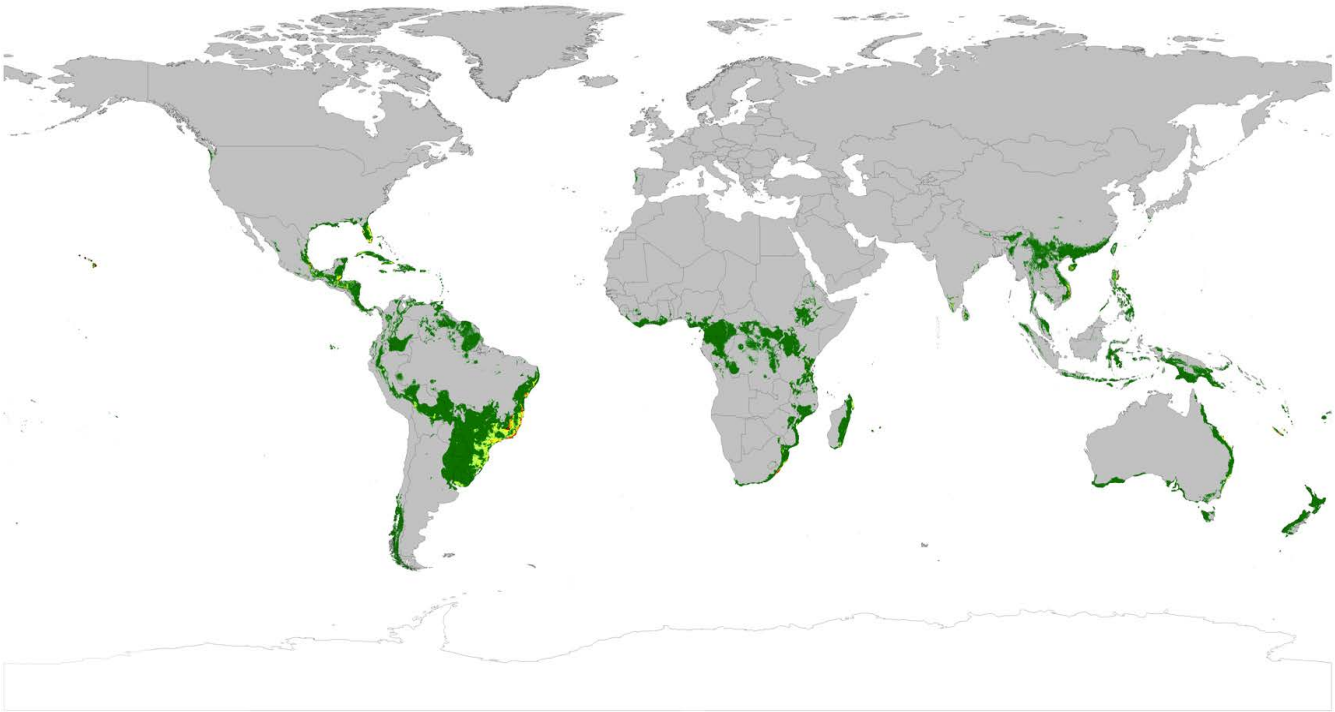

Figure 1: Global prediction of current (based on data from 1950-2000) suitable climate space for *Puccinia psidii* using 327 geo-referenced occurrence records from Brazil, Uruguay, Argentina, Paraguay, United States, Japan, and Costa Rica.

Bioclimatic Modeling

We used a maximum entropy approach with 327 geo-referenced occurrence records of *P. psidii* from Brazil, Uruguay, Paraguay, Costa Rica, USA and Japan. MaxEnt (Maximum Entropy Species Distribution Modeling) version 3.3.3e (Phillips et al. 2006) was used to predict the current distribution of *P. psidii* using high-resolution (30second = 1km) climate surfaces of 19 bioclimatic variables (e.g., annual mean temperature, annual precipitation, precipitation coldest quarter, etc.). These data were obtained from WorldClim (worldclim.org) and are based on interpolations of observed data from 1950-2000 (Hijmans et al. 2005). The future prediction model was then projected onto statistically downscaled (delta method) future climate surfaces for the 2050s (years 2040-2069) using the A1B SRES (Special Report on Emission Scenarios) scenario and CCCMA-CGCM 3.1 (Canadian Centre for Climate Modeling and Analysis – third generation coupled global climate model; Ramirez and Jarvis 2010). Four separate models were developed to predict current distribution of *P. psidii* based on (1) sample points of all occurrences of eucalypt rust in our data set (All model), (2) sample points of our occurrence data for eucalypt rust on guava (*Psidium guajava*) in South America only (Guava model), (3) sample points of our occurrence data for eucalypt rust on eucalypt (*Eucalyptus* spp.) from South America only (Eucalypt model), and (4) sample points of our occurrence data for eucalypt rust from multiple hosts in Hawaii only (Hawaii model).

Bioclimatic Prediction Maps

Predicted current global distribution of suitable climate space for *P. psidii* based on "All model" is shown in Figure 1. The predicted current global distribution map shows several geographic areas that may be at risk for *P. psidii* establishment, including parts of Africa, Australia, and Southeast Asia (Figure 1). Current distribution of suitable climate space for *P. psidii* in Hawaii based on the "All model" (Figure 2a) shows more areas with suitable climate space for *P. psidii* compared to predicted distribution based on the "Hawaii model" (Figure 2c). Projections of the "Eucalypt model" and "Guava model" on Hawaii show an almost inverse distribution compared to the "Hawaii model" (Figure 2c, 2d, and 2e).

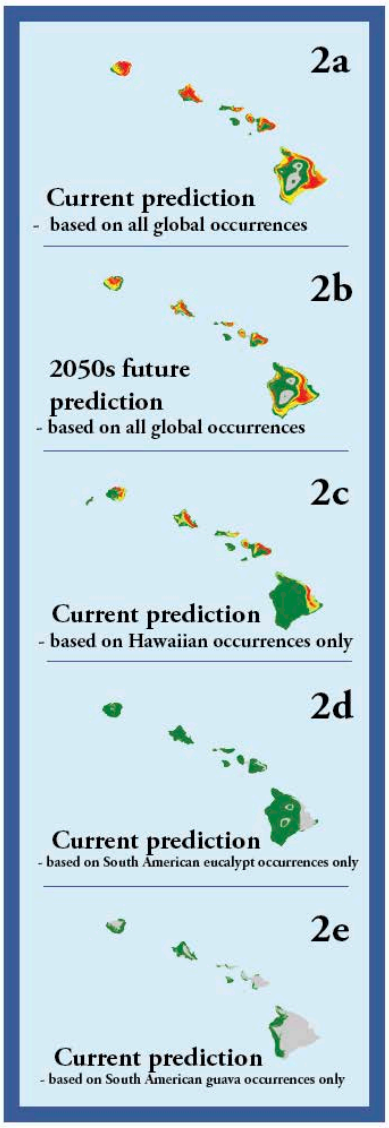

Figure 2a-e: Hawaiian *Puccinia psidii* predictions. Figure 2a: Detail of Figure 1. Figure 2b: Projected future suitable climate space for *P. psidii* using statistically downscaled (delta method) future climate surfaces for the 2050s (years 2040-2069), using CCCMA-CGCM 3.1 global circulation model, and the A1B SRES scenario. Figure 2c: Prediction of current suitable climate space for *P. psidii* based on 94 occurrence records from Hawaii (multiple host species). Figure 2d: Prediction of current suitable climate space for *P. psidii* based on 82 occurrence records from *Eucalyptus* spp. in South America. Figure 2e: Prediction of current suitable climate space for *P. psidii* based on 63 occurrence records on guava (*Psidium guajava*) in South America.

When considering that the *P. psidii* genotype in Hawaii has not been found on eucalypt or guava, these projections further indicate that introductions of new *P. psidii* genotypes from eucalypt and guava from South America or elsewhere pose an additional risk to potential hosts and new geographic areas of the Hawaiian Islands. The "Hawaii model" predictions of geographic areas with suitable climate for the single-genotype, Hawaiian *P. psidii* race (Graça *et al*. 2011) can provide insights into potential origins of the Hawaiian *P. psidii* race, especially when compared to the "Eucalypt model" and "Guava model" (Figure 3a, 3b, and 3c).

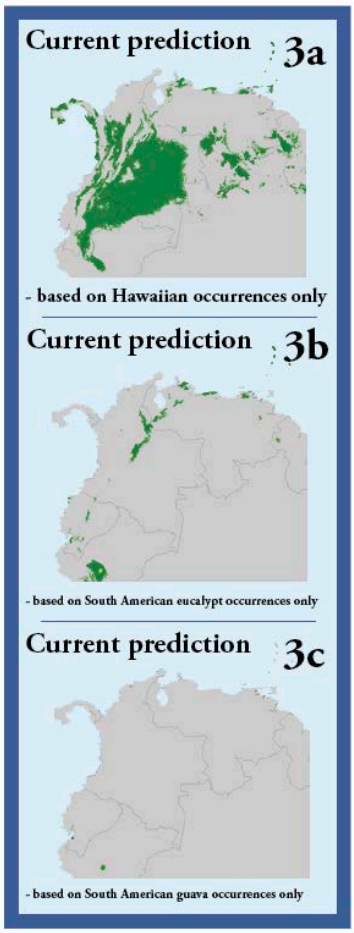

Figure 3a-c: *Puccinia psidii* predictions for northwestern South America. Figure 3a: Prediction of current suitable climate space for *P. psidii* based on 94 occurrence records from Hawaii (multiple host species). Figure 3b: Prediction of current suitable climate space for *P. psidii* based on 82 occurrence records from *Eucalyptus* spp. in South America. Figure 3c: Prediction of current suitable climate space for *P. psidii* based on 63 occurrence records of guava (*Psidium guajava*) in South America.

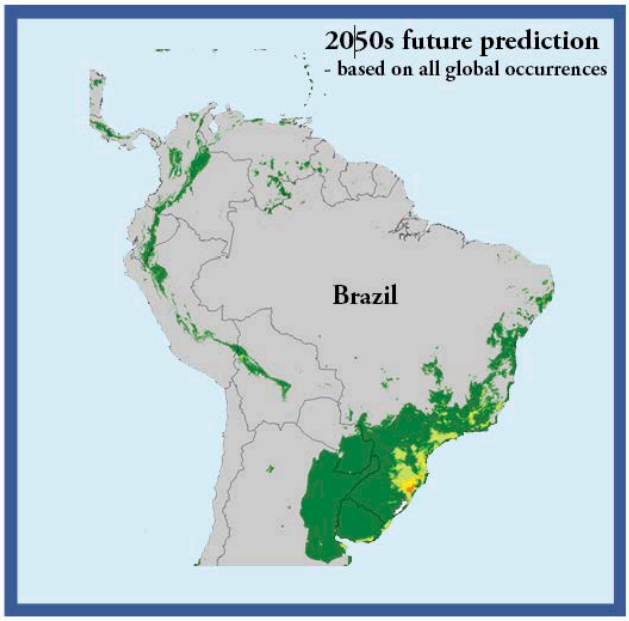

Figure 4: 2050s prediction of suitable climate space for *Puccinia psidii* in Brazil (using the same methods as Figure 2b).

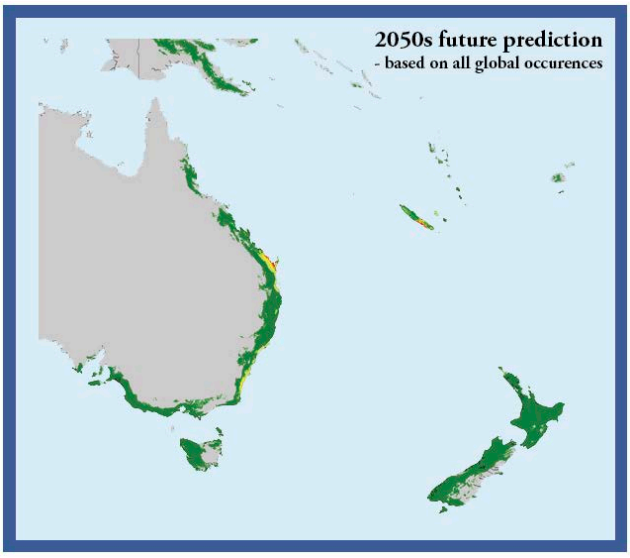

Figure 5: 2050s prediction of suitable climate space for *Puccinia psidii* in eastern Australia and New Zealand (using the same methods as Figure 2b).

The future distribution (2050s) of *P. psidii* based on the "All model" shows little change in suitable climate space for Hawaii (Figure 2b), greatly decreasing suitable climate space for Brazil in as little as 40 years (Figure 4), and relatively unchanged or slightly increasing climate suitability for *P. psidii* in Australia (Figure 5). As demonstrated here, this approach can be used to model *P. psidii* by host or genetically distinct group/race to determine whether individual *P. psidii* groups require disparate

environmental conditions. Combining climate-based, species-distribution modeling with genetic data can yield powerful insights into future distribution and potential invasive spread of forest disease. Additionally, predictions of the present and future distribution of *P. psidii* can help guide forest managers to implement appropriate forest practices to manage eucalypt rust according to current and future climates. This study is of great relevance for one-half of the forested land area of the tropics and sub-tropics, where the 4,500 species of Myrtaceae grow naturally and/or are actively cultivated. This area includes all countries that have significant investments in eucalypt forestry. Thus, information from this study can help identify areas at risk for *P. psidii* establishment, based on climatic envelopes. This information can also be used to help prevent introductions of *P. psidii* to global populations of Myrtaceae within regions that are at risk for *P. psidii* establishment.

ACKNOWLEDGEMENTS

This project was partially funded by Special Technology Development Program (R5-2011-04: "Guava rust (*Puccinia psidii*): evaluating pathways of spread and assessing future risk), Joint Venture Agreement (07-JV-11221662-285), Forest Health Protection – Region 5, and the Western Wildland Environmental Threat Assessment Center.

REFERENCES

Booth, T.H., Old, K.M., Jovanovic, T. 2000. A preliminary assessment of high risk areas for *Puccinia psidii* (Eucalyptus rust) in the Neotropics and Australia. Agriculture Ecosystems & Environment. 82:295-301

Carnegie, A.J., Lidbetter, J.R., Walker. J., Horwood, M.A., Tesoriero, L., Glen, M., Priest, M. 2010. *Uredo rangelii*, a taxon in the guava rust complex, newly recorded on Myrtaceae in Australia. Austalasian Plant Pathology. 39:463-466.

Coutinho, T.A., Wingfield, M.J., Alfenas, A.C., Crous, P.W. 1998. Eucalyptus rust: A disease with the potential for serious international implications. Plant Disease. 82:819-825.

Farr, D.F., Rossman, A.Y. 2010. Fungal Databases, Systematic Mycology and Microbiology Laboratory, ARS, USDA. Retrieved 5/4/10 from http://nt.ars-grin.gov/fungaldatabases/.

Graça, R.N., Alfenas, A.C., Ross-Davis, A.L., Klopfenstein, N.B.,and others. 2011. Multilocus genotypes Indicate differentiation among *Puccinia psidii* populations from South America and Hawaii. Pages 131-134 In Fairweather, M.L. Comp. Proceedings of the 58th Annual Western International Forest Disease Work Conference; 2010 October 4-8; Valemount, BC. US Forest Service, AZ Zone Forest Health, Flagstaff, AZ.

Hijmans, R.J., Cameron, S.E., Parra, J.L., Jones, P.G., Jarvis, A. 2005. Very high resolution interpolated climate surfaces for global land areas. International Journal of Climatology. 25:1965-1978.

Kawanishi, T., Uematsu, S., Kakishima, M., Kagiwada, S., Hamamoto, H., Horie, H., Namba, S. 2009. First report of rust disease on ohia and the causal fungus, *Puccinia psidii*, in Japan. Journal of General Plant Pathology. 75:428-431.

Loope, L., La Rosa, A.M. 2008. An analysis of the risk of introduction of additional strains of the rust *Puccinia psidii* (`Ohi´a rust) to Hawai´i. U.S. Geological Survey Open File Report 2008-1008, Reston, VA.

MacLachlan, J.D. 1938. A rust of pimento tree in Jamaica, B.W.I. Phytopathology. 28:157-170.

Phillips, S.J., Anderson, R.P., Schapire, R.E. 2006. Maximum entropy modeling of species geographic distributions. Ecological Modelling. 190:231-259

Ramirez, J., Jarvis, A. 2010. Decision and Policy Analysis Working Paper No.1 Centro Internacional de Agricultura Tropical International Center for Tropical Agriculture. Cali, Colombia.

Rehfeldt, G.E., Crookston, N.L., Warwell, M.V., Evans, J.S. 2006. Empirical Analyses of Plant-Climate Relationships for the Western United States. International Journal of Plant Sciences. 167:1123-1150.

Uchida J.; Zhong, S.; Killgore, E. 2006. First report of a rust disease on Ohi'a caused by *Puccinia psidii* in Hawaii. Plant Disease. 90:524.

Winter, G. 1884. Repertorium. Rabenhorstii funi europaei et extraeuraopaei. Cent. XXXI et XXXII. Hedwigia. 23:164–172.

SUDDEN LARCH DEATH? LARCH SUSCEPTIBILITY TO *PHYTOPHTHORA RAMORUM* IN OREGON FORESTS

Everett Hansen[1], Joe Hulbert[1], Paul Reeser[1], Wendy Sutton[1], and Alan Kanaskie[2]

To test the susceptibility of Japanese larch (*Larix kaempferi*) as well as our North American native Western larch (*Larix occidentalis*) to the strains of *P. ramorum* found in Oregon Forests, we exposed potted seedlings to natural inoculation under infected tanoak trees in Curry County Oregon. Douglas-fir (*Pseudotsuga menziesii*) was also included for comparison. We determined that exposure of potted seedlings beneath infected tanoak during periods of sporulation provides an effective and realistic susceptibility test. Three hundred and six dormant, 2-yr-old seedlings, representing the three different species, were transplanted into gallon pots and exposed to *P. ramorum*. The seedlings were distributed among ten different sites in Curry County on March 1st, 2011 while still dormant, and returned to Corvallis at the end of May, after budburst. Control seedlings, not exposed to inoculum, remained in Corvallis. Symptoms consistent with *P. ramorum* infection developed on most exposed seedlings of Douglas-fir and western larch (88% of Douglas-fir and 98% of western larch). The main symptom observed was wilting or collapse of the new shoot. Results for Japanese larch were complicated by the low vigor of the seedlings. About half of the seedlings (including controls) died without bursting bud, and another 30% died shortly after bud burst. *P. ramorum* was isolated from advancing margins of lesions on twigs at the base of new growth of most of the Douglas-fir and western larch seedlings at the end of the study. It was not recovered from any control seedlings. Recovery of *P. ramorum* from Japanese larch was low (25-35 %).

Both Douglas-fir and western larch seedlings had similar percentages of *P. ramorum* recovery and both were infected at all 10 of the field sites. *P. ramorum* was isolated from lesions on 71 of the 102 (69.6%) Douglas-fir seedlings and from 63 of the 102 (61.8%) western larch seedlings. We concluded that Western larch is susceptible to the Oregon forest lineage (NA1) of *P. ramorum* under environmental conditions prevalent in the epidemic area of SW Oregon. Japanese larch is also susceptible, although the degree of susceptibility cannot be determined from this experiment.

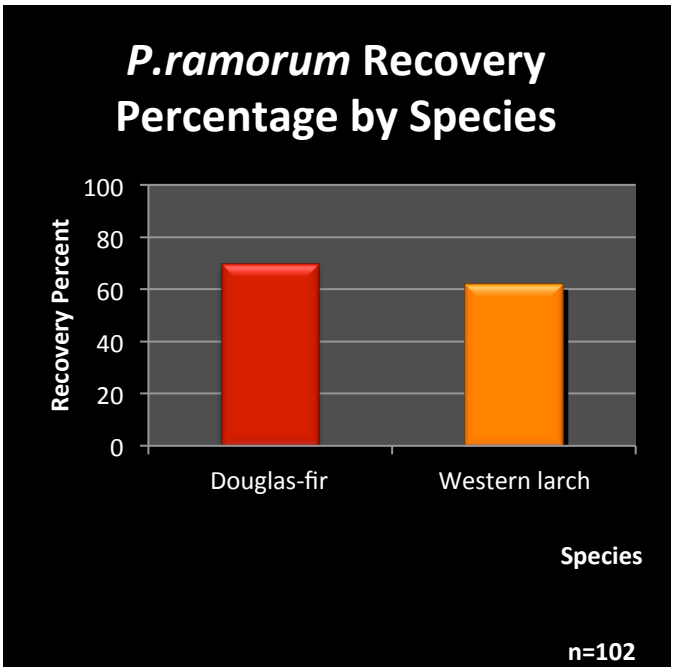

Figure 1: Comparison of *P. ramorum* recovery percentages between Douglas-fir and western Larch.

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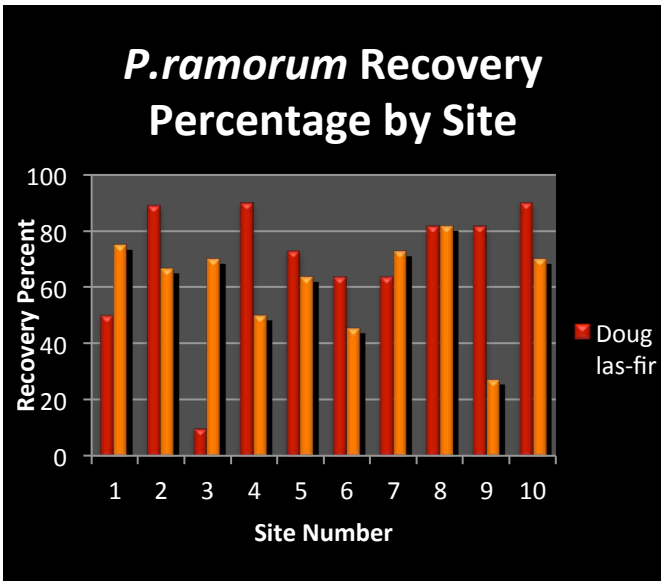

Figure 2: *P. ramorum* recovery percentages of Douglas-fir and western Larch at each site.

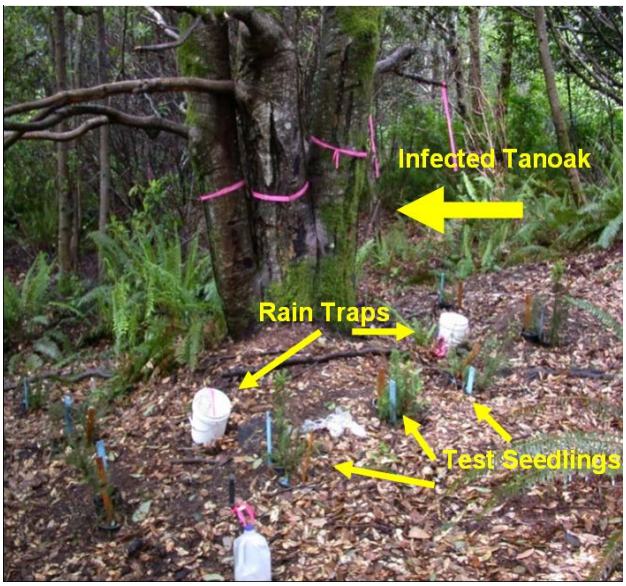

Figure 4: Site # 4 - Pink flagging tied around infected tanoak with rain traps and bait seedlings placed under the canopy.

Figure 3: Symptomatic western larch before pathogen isolation in the lab

Figure 5: Potted seedlings, near the end of the exposure period, at site # 4.

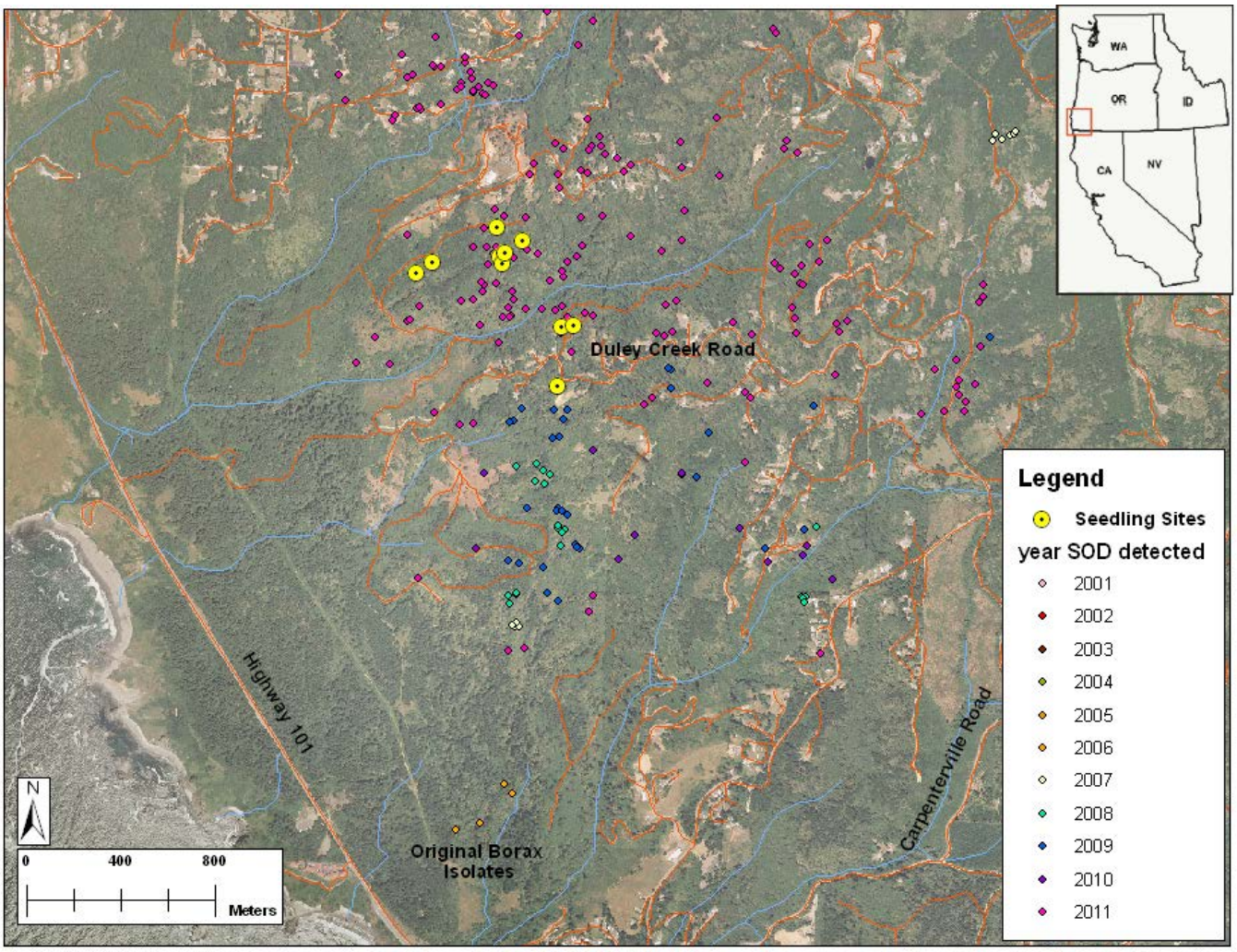

Figure 6: Site distribution in Duley Creek drainage area near Brookings, Oregon.

SYMPTOMATIC ALDER IN THE RIPARIAN ZONE

Laura Sims[1] and Everett Hansen[1]

ABSTRACT

The Forest Service Forest Health Monitoring Program for Region 6 supports a project in western Oregon to survey declining *Alnus* species along waterways to determine species of *Phytophthora* present, to monitor for *Phytophthora alni* and to assess alder health. This is done in part because riparian ecosystems are underrepresented in FIA data due to their linear nature and because the health status of alders in these ecosystems is not well understood. Eighty-eight 100 meter by 10 meter transects running alongside waterways across three subregions of western Oregon were laid out and sampled in 2010. Root and soil samples were collected for baiting. Water samples near declining alders were collected for filtration. Using standardized methods for identification of *Phytophthora* 1189 *Phytophthora* isolates from 17 species groups were collected in 2010. Confirmation of genus was completed with morphological techniques. Confirmation of species was done with Sanger sequencing of the Cox spacer region (Martin & Tooley 2003), by aligning these regions and by using bayesian phylogenetic analysis. Of the 2310 living *Alnus* trees observed, 979 were found to have some level of dieback. Of these 979 trees, the average level of dieback was 24%. *Phytophthora alni* was not found in 2010 in western Oregon. Monitoring continues in 2011 and 2012 with some changes to technique including direct plating of surface sterilized roots.

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REFERENCE

Martin, F.N., Tooley, P.W. 2003. Phylogenetic relationships among *Phytophthora* species inferred from sequence analysis of mitochondrially encoded cytochrome oxidase I and II genes. Mycologia. 95:269-284.

GROWTH RATE OF THE MYCELIA OF *HETEROBASIDION OCCIDENTALE* AT DIFFERENT TEMPERATURES

Robert Edmonds[1] and Mahsa Khorasani[1]

INTRODUCTION

The genus *Heterobasidion* includes some of the most economically important pathogens in northern hemisphere forests. Two species occur in Washington: *Heterobasidion occidentale* (formerly *H. annosum* S strain) on hemlocks, spruces, firs, pines and hardwoods and *H. irregulare* (formerly *H. annosum* P strain) on pines. They cause root and butt rot and mortality. Many forest pathogens, especially foliage diseases and rusts, are expected to respond to global warming (Sturrock et al. 2011), but we are not sure how it will influence root and butt rots. Temperatures have been rising in coastal (Figure 1A), Puget Sound (Figure 1B), and east slopes of the Cascade Mountains (Figure 1C) of Washington since the 1950s.

Thus it is important to know if temperature is a limiting factor for spread of *Heterobasidion* spp. However, we know very little about how temperature influences the growth of *H. occidentale* in Washington. Taubert (2008) investigated the growth rate of *H. annosum* sensu stricto and *H. parviporum* in Europe. She found the optimum growth rate of *H. parviporum* was 27 C while it was 22 C in *H. annosum* sensu stricto. However, she didn't examine temperatures between 22 and 27 C. Brown and Webber (2009) reported 25 C as the optimum growth rate for *H. annosum.*

OBJECTIVE

To determine the growth rates in culture of *Heterobasidion occidentale* isolates from coastal areas, Puget Sound and east slopes of the Cascades at different temperatures ranging from 20 to 29 C.

METHODS

Fruiting bodies of *H. occidentale* were obtained from different hosts in coastal, Puget Sound and east slopes of the Cascades. Cultures from fruiting bodies were grown on maltose agar media on Petri dishes for 7 days in different temperatures between 20 C to 29 C and radial growth was measured after 3 and 7 days.

RESULTS

Figure 2 shows the growth rate in culture of 9 isolates of *Heterobasidion occidentale* from western hemlock in the Puget Sound area at different temperatures between 20 C and 29 C.

The optimum growth rate is between 23 to 24 C varying from 6.9 to 9 mm/day. Table 1 shows growth at 24 C for isolates from the coast, Puget Sound and east slope of the Cascades at 24 C. East slope Cascade isolates from grand fir had a slower growth at 24 C. There was no difference in growth rates of isolates from western hemlock, red alder, and cottonwood at 24 C.

Table 1: Radial growth rates (mm/day) of mycelia at 24 C for isolates from the coast, Puget Sound and east slope Cascades of Washington.

Location	Radial growth rate (mm/day)
Coast	6.6±0.9
Puget Sound	6.3±1.2
East slope Cascades	3.3±1.4

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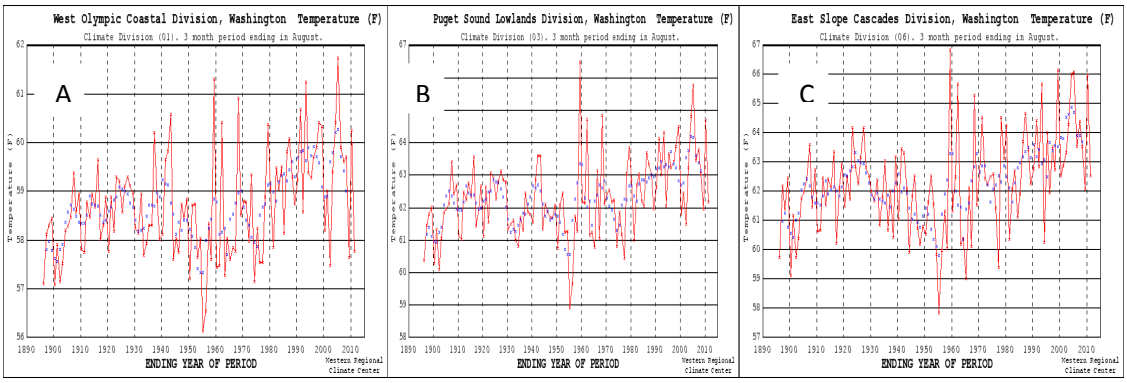

Figure 1: Average June-August air temperatures have increased since the 1950s in (A) coastal, (B) Puget Sound and (C) Cascade east slopes of Washington.

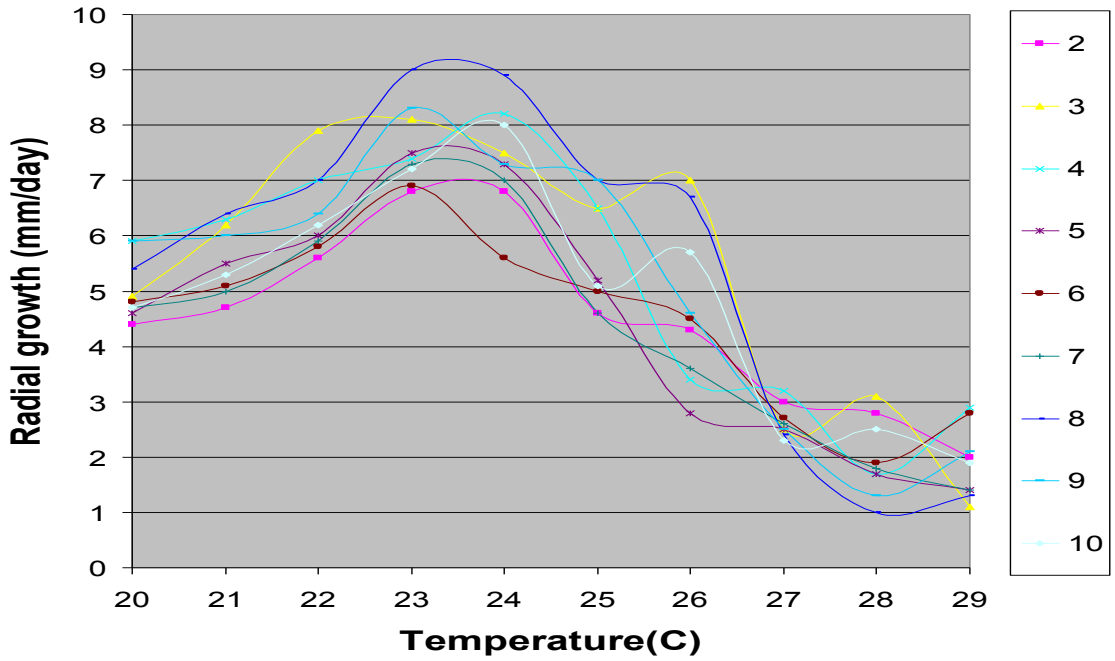

Figure 2: Radial growth rates (mm/day) of nine isolates of *H. occidentale* from western hemlock in the Puget Sound area in relation to temperature.

DISCUSSION

There was some variation in growth rates in hemlock isolates from the Puget Sound area. Optimum temperatures were 23-24 C. Puget Sound and coastal isolates had similar growth rates at 24 C. However, east slope Cascade isolates from grand fir had a slower growth rate, perhaps reflecting adaption to that environment which is colder and drier.

Global warming could potentially increase the growth rates of *H. occidentale* in Washington since temperatures in roots and boles are still below growth optima most of the year. However, growth increases may have occurred already since temperatures have been warming for some time in the Pacific Northwest.

REFERENCES

Brown, A.V., Webber, J.F. 2009. Biocontrol of decay in seasoning utility poles. I. Growth rate and colonizing ability of blue stain and decay fungi in vivo and in vitro. Forest Pathology. 39:145-156.

Sturrock, R.N., Frankel, S.J., Brown, A.V., and others. 2011. Climate change and forest diseases. Forest Pathology. 60:133-149.

Taubert, J. 2008. Temperature requirements for germination of conidiospores and growth of mycelia of *Heterobasidion annosum* s.s. and *Heterobasidion parviporum*. Swedish University of Agriculture, Southern Swedish Forest Research Center Sciences, Alnarp, Sweden.

DISCOVERY OF CRYPTIC *ARMILLARIA SOLIDIPES* GENOTYPES WITHIN THE COLORADO PLATEAU

J.W. Hanna[1], N.B. Klopfenstein[1], M.-S. Kim[2], S.M. Ashiglar[1], A.L. Ross-Davis[1,3], and G.I. McDonald[1]

ABSTRACT

Armillaria solidipes (= *A. ostoyae*) is a root-disease pathogen that causes severe losses in growth and productivity of forest trees throughout the Northern Hemisphere. This species is genetically diverse with variable disease activities across different regions of the world. In North America, *A. solidipes* in the Colorado Plateau exists in drier habitats and causes more disease on hardwoods in comparison with *A. solidipes* in the northwestern USA (McDonald 1999). Here, we address 1) the discovery of cryptic genotypes of *A. solidipes* located within or near the Colorado Plateau region, 2) the analytical issues associated with rDNA sequences containing high levels of heterogeneity, and 3) the significance of these cryptic *A. solidipes* genotypes.

Methods Used To Discover Cryptic *A. solidipes* Genotypes

The intergenic spacer 1 (IGS-1) rDNA region of a single *A. solidipes* [pending vote to conserve *A. ostoyae* (Redhead et al. 2011)] isolate (U5) was cloned and sequenced to examine sequence heterogeneity. The resulting IGS-1 rDNA sequences (8 sequence types derived from 100 clones) demonstrated high heterogeneity within the IGS-1 rDNA region (Table 1). The sequence types of the cloned isolate (U5) are representative of other *A. solidipes* isolates from the Colorado Plateau (Figure 1).

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We then compared the IGS-1 rDNA sequence types from this study with sequences of *A. solidipes* and closely related species from Asia, Europe, and North America. All sequences were edited and aligned by hand/eye and analyzed using the neighbor-joining method for reconstructing phylogenetic trees in MEGA 5.05 (Figure 2) (Saitou and Nei 1987, Tamura et al. 2011).

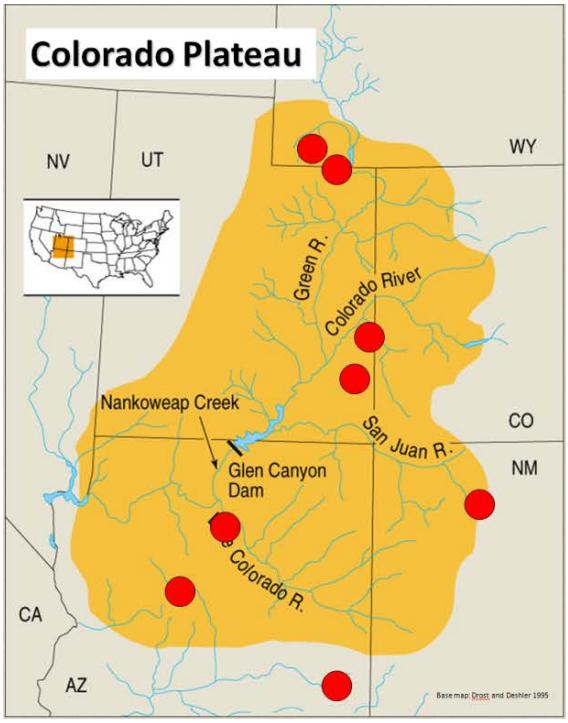

Figure 1: Approximate known locations of isolates putatively regarded as belonging to the "Colorado Plateau" *A. solidipes* group. These isolates contain numerous single nucleotide polymorphisms (SNPs) within the intergenic spacer 1 (IGS-1) rDNA region (Table 1). This indicates that each individual has heterogeneous (hybrid) intraspecific genotypes that include sequence types of both Colorado Plateau and Circumboreal (formerly Rockies group) *A. solidipes* clades (Figure 2).

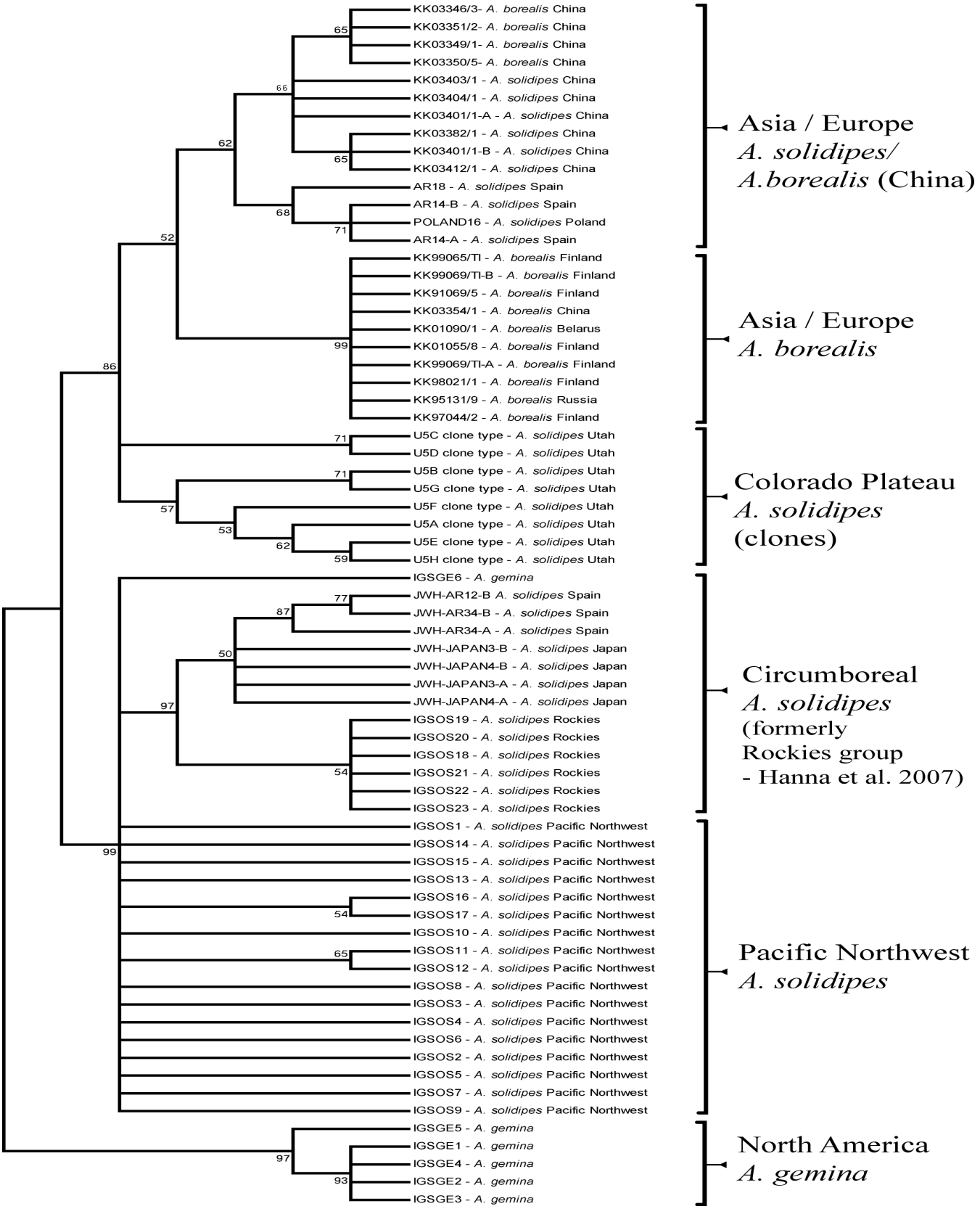

Figure 2: Neighbor-joining 50 percent majority-rule, consensus tree based on 591 basepairs within the IGS-1 rDNA region. The number at each node represents percent bootstrap values out of 500 replicates.

Table 1: SNPs (single nucleotide polymorphisms) within a sequence obtained using direct PCR versus eight IGS-1 sequence types derived from 100 clones of the same isolate (U5).

Base Pair	25	48	195	226	270	300	311	361	366	372	403	411	450	457	483	517	529	542	545
Isolate U5 Direct PCR	Y	Y	Y	Y	Y	R	R	Y	W	Y	R	K	R	Y	S	K	W	Y	R
U5 - Clone Type A	T	C	C	T	T	G	G	T	T	C	A	G	G	T	C	G	T	T	G
U5 - Clone Type B	T	C	C	T	T	G	G	T	T	C	A	G	G	T	C	G	T	C	A
U5 - Clone Type C	C	T	T	C	C	G	G	T	T	C	A	G	G	T	C	G	T	T	G
U5 - Clone Type D	C	T	T	C	C	A	G	T	T	C	A	G	G	T	C	G	T	T	G
U5 - Clone Type E	T	C	C	T	T	G	G	T	T	C	A	G	G	T	C	G	T	T	G
U5 - Clone Type F	C	C	C	T	T	G	G	T	T	C	A	G	G	T	C	G	T	T	G
U5 - Clone Type G	T	C	C	T	T	G	G	T	T	C	A	G	G	T	C	G	A	C	A
U5 - Clone Type H	T	C	C	T	T	G	G	T	T	C	A	G	G	T	C	G	T	T	G

SNP codes:
K = G or T
R = A or G
Y = C or T
S = C or G
W = A or T

Significance Of Cryptic *A. solidipes* Genotypes From The Colorado Plateau

Previous studies of genetic diversity within *A. solidipes* have shown that using heterogeneous sequence types can potentially reduce phylogenetic signal by collapsing clades between that of its ancestral/parental origins (Hanna et al. 2007). For this reason, we examined clones of the IGS-1 region from one of several highly heterogeneous isolates identified from the Colorado Plateau (Figure 1).

Surprisingly, the resulting sequence types derived from the U5 IGS-1 clones are more closely related to *A. borealis* sequence types than with other North American *A. solidipes* groups (Figure 2). Furthermore, a high level of apparent recombination within the clones was revealed (Table 1), and moreover, the eight sequence types from the 100 clones did not account for all the sequence variability observed by direct-PCR sequencing methods (Table 1). The genetic differences between this cryptic genotype and other North American *A. solidipes* isolates may correspond with the differences in disease activity/potential vegetation groups from that of *A. solidipes* in the Northwestern United States (Table 2) (McDonald 1999).

These cryptic *A. solidipes* genotypes from the Colorado Plateau could potentially represent ancient genotypes of *Armillari*a that have occupied the Colorado Plateau for over 200 million years, dating back to an ancient time when present-day geographic regions of North America, Europe, and Asia were contiguous. More studies are needed to determine the ecological significance of these cryptic *A. solidipes* genotypes from the Colorado Plateau.

ACKNOWLEDGEMENTS

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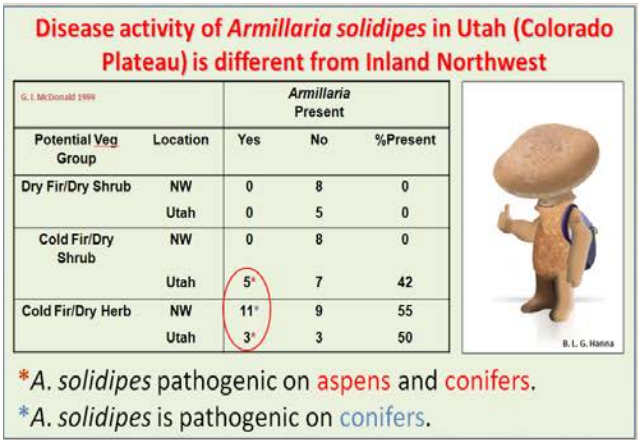
Table 2: Table from McDonald et al. 1999 showing that disease activity of *Armillaria solidipes* differs in Utah from that of *A. solidipes* from the Pacific Northwest.



REFERENCES

Drost, C., Deshler E. 1995. Amphibian and Reptile Diversity on the Colorado Plateau. Pages 326-328 In: LaRoe, E.T. et al. eds. Our Living Resources: A report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Geological Survey, Washingto DC.

Hanna, J.W., Klopfenstein, N.B., Kim, M.-S., McDonald, G.I., Moore, J.A. 2007. Phylogeographic patterns of *Armillaria ostoyae* in the western United States. Forest Pathology. 37: 192-216.

McDonald, G.I. 1999. Preliminary report on the ecology of *Armillaria* in Utah and the Inland West. Pages 85-92 In: Trummer, L., Comp. Proceedings of the 46th Western International Forest Disease Work Conference. Reno, Nevada, Sept 28- Oct 2, 1998. Anchorage, AK. U.S.A.: USDA Forest Service R-10.

Redhead, S.A., Bérubé, J., Cleary, M.R., Holdenrieder, O., and others. 2011. (2033) Proposal to conserve *Armillariella ostoyae* (*Armillaria ostoyae*) against *Agaricus obscurus*, *Agaricus occultans*, and *Armillaria solidipes* (Basidiomycota). Taxon. 60(6):1770-1771.

Saitou, N., Nei, M. 1987. The neighbor-joining method: a new method for reconstructing phylogenetic trees. Molecular Biology and Evolution. 4:406-425.

Tamura, K., Peterson, D., Peterson, N., Stecher, G., Nei, M., Kumar, S. 2011. MEGA5: Molecular Evolutionary Genetics Analysis using Maximum Likelihood, Evolutionary Distance, and Maximum Parsimony Methods. Molecular Biology and Evolution. 28:2731-2739.

DEVELOPING A PREDICTION MODEL FOR *ARMILLARIA SOLIDIPES* IN ARIZONA

N.B. Klopfenstein[1], J.W. Hanna[1], M.L. Fairweather[2], J.D. Shaw[3], R. Mathiasen[4], C. Hoffman[4], E. Nelson[4], M. -S. Kim[5], and A.L. Ross-Davis[1,6]

ABSTRACT

In 2010, a collaborative project was started to determine the distribution of *Armillaria solidipes* (= *A. ostoyae*) in Arizona. The methods and preliminary accomplishments of the 2010 and 2011 (ongoing) field surveys/collections are summarized. During the next phase of this project, surveys will be completed and remaining *Armillaria* isolates will be identified using DNA-based methods. In addition, a preliminary prediction map based on 25 locations positive for *A. solidipes* is presented. Sites positive for *A. solidipes* are associated with climate data to predict the potential current distribution (and disease activity) of *A. solidipes* within the region. Data from this region can be added to global *A. solidipes* datasets that will help develop bioclimatic models for predicting the future distribution and disease activity of *Armillaria solidipes* under various climate-change scenarios.

INTRODUCTION

Severity of Armillaria root disease appears to increase with increased forest management (McDonald et al. 1987). This causes a dilemma for forest managers because it can be difficult to determine if a site is at risk for Armillaria root disease unless symptoms are prominent. The pathogenic species *A. solidipes* [pending vote to conserve A. *ostoyae* (Redhead et al. 2011)] is often difficult to identify from other saprophytic *Armillaria* spp., and *A. solidipes* seems to exist in a non-pathogenic state in some areas (unpublished data). Recently, DNA-based methods became available to identify North American *Armillaria* species (Kim et al. 2006, Ross-Davis et al. 2011). In this study, we are using DNA-based identification and location-specific climate data for bioclimatic modeling to predict where *A. solidipes* is likely to occur and cause disease under different forest management regimes.

OBJECTIVES

(1) Determine suitable climate space for *Armillaria solidipes* in Arizona.

(2) Predict which Arizona forest areas are at risk to disease caused by *Armillaria solidipes.*

(3) Develop habitat-specific management strategies to reduce impacts of Armillaria root disease.

(4) Incorporate information into a bioclimatic model to predict the potential future (e.g., 2030, 2060, and 2090) distribution (and disease activity) of *Armillaria solidipes*.

METHODS

Currently, 115 0.04-ha plots in Arizona have been surveyed for *Armillaria* spp. (Figures 1 and 2). Actual plot locations were selected on the basis of plot availability within a given section, accessibility by road, and maximizing the variation in elevation, slope, and vegetation types encountered on the landscape. Three live or dead trees and shrubs of each species present were inspected for the presence of *Armillaria* spp. “Large”, “Medium”, and “Small” trees (relative to tree sizes found on plot) of each species were selected for inspection and sampling. Tree inspection and sampling was

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conducted by excavating at least one main root and the nearest portion of root collar to a radial distance of approximately 0.5 m from the bole. Shrubs were sampled by extracting the root system. If present, rhizomorphs were collected from trees and shrubs. If resinosis or other symptoms of disease were apparent, the root collar was cut to inspect and collect bark-fan samples, if present. *Armillaria* samples were kept cool and mailed to the Forestry Sciences Laboratory in Moscow, where isolates were established in culture in preparation for DNA-based species identification.

Figure 1: Field crew establishing a plot (left); Tree showing possible symptoms of Armillaria root disease (center); *Armillaria* spp. basidiocarps at the base of a tree (right).

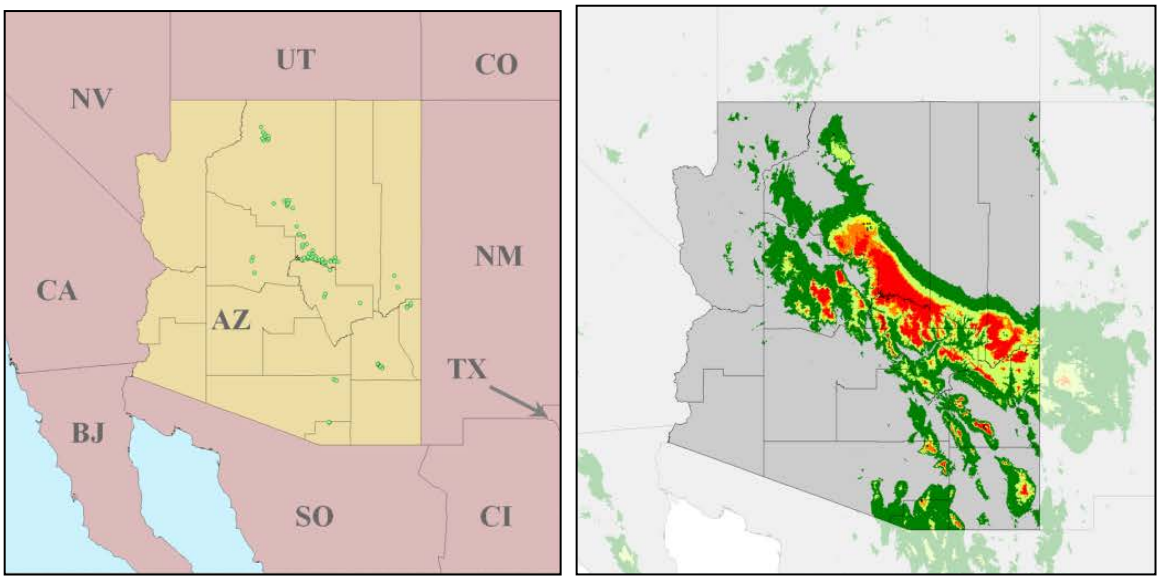


Figure 2: Map of Arizona showing collection sites during the 2010 and 2011 field seasons (left); Preliminary example of predicted suitable climate space for *Armillaria solidipes* in Arizona based on 25 locations positive for *A. solidipes* collected in 2010. Dark green represents predicted suitable climate space for *A. solidipes* with light green, yellow, orange, and red indicating increased likelihood of suitable climate space, respectively (right).

PROJECT OVERVIEW

Phase One - Data Collection

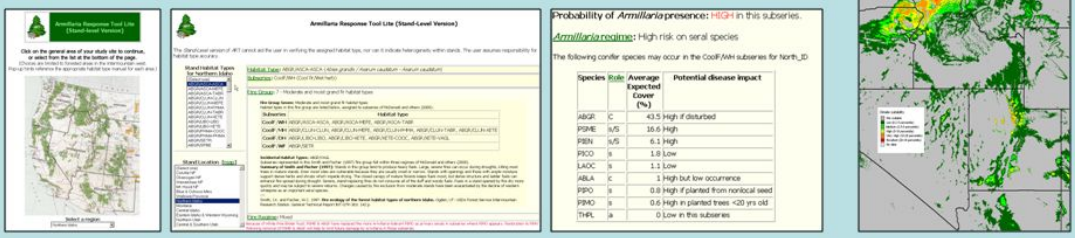
Digging for rhizomorphs (left) Mycelial bark fan colonizing the root collar of a tree (middle) Slope, aspect, location, elevation, plant association, and site description are some of the data recorded at each plot (right).

Phase Two - Identification

After pure fungal cultures of *Armillaria* sp. are obtained scrapings of mycelium are taken from the isolates (left) and added to PCR reagents. The samples then undergo PCR reactions in a thermocycler (middle). Successfully amplified DNA fragments are then sequenced and unique genetic code (right) can be analyzed to identify *Armillaria* spp. (Kim et al. 2006).

Phase Three - Analysis and Products

http://forest.moscowfsl.wsu.edu/fuels/art



Probability of *Armillaria* presence: HIGH in this subseries.

Armillaria regime: High risk on seral species

The following conifer species may occur in the CoolF/WH subseries for North_ID

Species	Role	Average Expected Cover (%)	Potential disease impact
ABGR	C	43.5	High if disturbed
PSME	s/S	16.6	High
PIEN	s/S	6.1	High
PICO	s	1.8	Low
LIDE	s	1.1	Low
ABLA	C	1	High but low occurrence
PIPO	s	0.8	High if planted from nonlocal seed
PIMO	s	0.6	High in planted trees <20 yrs old
THPL	a	0	Low in this subseries

Data from this project can be incorporated into tools for land managers such as the *Armillaria* Response Tool (ART – shown to the left and center) (McDonald et al. 2005) or be used to model climate profiles (Rehfeldt et al. 2006) for *Armillaria ostoyae* and predict the affects of climate change on *Armillaria ostoyae* (example map -right).

FUTURE WORK

We will continue to conduct DNA-based identification of *Armillaria* species at the RMRS Forestry Sciences Lab in Moscow, ID. After all *Armillaria* isolates are identified, locations confirmed to have *A. solidipes* will be used to predict suitable climate spaces for this pathogen (Figure 2). This climate window can also be used to examine how various climate-change scenarios may affect *A. solidipes* in Arizona. The methods developed from this project can also be used to model other important forest pathogens and examine the potential for invasive species to occupy new areas under a changing climate.

ACKNOWLEDGEMENTS

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REFERENCES

Kim, M.-S., Klopfenstein, N.B., Hanna, J.W., McDonald, G.I. 2006. Characterization of North American *Armillaria* species: Genetic relationships determined by ribosomal DNA sequences and AFLP markers. Forest Pathology. 36:145-164.

McDonald, G.I., Martin, N.E., Harvey, A.E. 1987. *Armillaria* in the northern Rockies: Pathology and host susceptibility on pristine and disturbed sites. USDA Forest Service, Research Note INT-371. Intermountain Research Station, Ogden, UT. 5 p.

McDonald, G.I., Tanimoto, P.D., Rice, T.M., Hall, D.E., and others. 2005. Fuels planning: science synthesis and integration; environmental consequences fact sheet 13: Root Disease Analyzer-*Armillaria* Response Tool (*A*RT). USDA Forest Service, RMRS-RN-23-13WWW. Rocky Mountain Research Station, Fort Collins, CO. 2 p.

Redhead, S.A., Bérubé, J., Cleary, M.R., Holdenrieder, O., and others. (2033) Proposal to conserve *Armillariella ostoyae* (*Armillaria ostoyae*) against *Agaricus obscurus*, *Agaricus occultans*, and *Armillaria solidipes* (Basidiomycota). Taxon. 60(6):1770-1771.

Rehfeldt, G.E., Crookston, N.L., Warwell, M.V., Evans, J.S. 2006. Empirical Analyses of plant-climate relationships for the western United States. International Jouranl of Plant Science. 167:1123-1150.

Ross-Davis, A.L., Hanna, J.W., Kim, M.-S., Klopfenstein, N.B. 2011. Advances toward DNA-based identification and phylogeny of North American Armillaria species using elongation factor-1 alpha gene. Mycoscience. Published online first 8/13/11, DOI 10.1007/s10267-011-0148-x.

EARLY EFFECTS OF WHITE PINE BLISTER RUST IN ARIZONA AND NEW MEXICO

Christopher Looney[1], Kristen Waring[1], and Mary Lou Fairweather[2]

White pine blister rust (*Cronartium ribicola*; WPBR) is an invasive fungal pathogen causing widespread mortality in North American five-needled white pines. WPBR was first detected in New Mexico in 1990, and was not found in Arizona until 2009. These Southwestern populations provide a unique opportunity to document the initial impacts of the disease on understudied white pine species (*Pinus strobiformis*, *P. flexilis*, and *P. artistata*). We documented WPBR distribution and white pine forest structure throughout Arizona and New Mexico. In addition, we investigated the effects of WPBR on white pine growth rate and vigor. We randomly installed 50x20 m permanent plots in stands containing white pine basal area exceeding 6.9 m^2 ha^{-1}. We recorded species, diameter at breast height (DBH) for all species and WPBR presence and severity, height, and crown dimensions for all white pine trees. We also documented other damaging agents occurring in white pine. Regeneration of white pine and other tree species was measured on nested 10x10 and 5x5m subplots, respectively. Other measurements included fuel loading, site topography, and overstory canopy cover. White pine faces heavy competition and shows limited regeneration, while forest fuel loading was high. White pine blister rust was not detected outside of known 2009 infection centers in either state, but we describe its occurrence and effects in more detail within those areas. The presence of WPBR was sporadic and light in our surveyed areas, while the incidence of other damaging agents was also low.

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ELEVATED ETHANOL CONCENTRATIONS IN THE CANKER REGION OF *PHYTOPHTHORA RAMORUM*–INFECTED COAST LIVE OAKS

Maia Beh[1], Rick G. Kelsey[2], Dave Shaw[1], and Daniel K. Manter[3]

Phytophthora ramorum, causal agent of sudden oak death, produces lethal cankers in the inner bark and sapwood of certain oak species (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) in coastal California and southwestern Oregon. Ambrosia beetle attacks concentrated in the canker region of *P. ramorum*-infected trees are frequently observed and can greatly hasten mortality in coast live oaks (*Q. agrifolia*). Factors influencing the beetles' preference for the canker tissues have never been explained. Studies have shown changes in concentrations for several phenolic compounds in diseased inner bark of coast live oak that are speculated to serve a role in chemical defense, but with unknown influence on ambrosia beetles.

Ethanol is produced in the vascular cambium of trees when there is insufficient oxygen to carry out normal aerobic respiration and the tissues must resort to fermentative metabolism. Situations known to result in elevated levels of ethanol in conifers include flooding, severe water deficit, or pathogen infection, and this accumulation of ethanol can increase the risk of attacks by bark and ambrosia beetles (Coleoptera: Scolytidae). If ethanol concentrations are similarly elevated in the *P. ramorum*-infected tissues of oaks, then the release of this volatile compound outside of the bark covering the canker region may be responsible for the initial attraction of ambrosia beetles to these diseased trees.

To quantify and compare ethanol levels in the sapwood of *P. ramorum*-cankered versus healthy *Q. agrifolia*, fourteen pairs of healthy and cankered oaks were sampled at 3 locations in Marin and Sonoma Counties, California in September, 2010. Within each pair of trees, cores of sapwood were obtained with an increment borer at: 1) specific locations within and outside the canker margin in the diseased tree, and 2) analogous locations along the bole of the healthy tree. Sapwood samples were sealed in headspace vials and immediately frozen with dry ice. Vials were later heated to 100 ^{o}C for 60 min. to kill any microorganisms. Ethanol concentrations were measured with static headspace gas chromatography. The instrument was calibrated with standard ethanol solutions and ethanol concentrations calculated as µg/gram fresh mass.

The median sapwood ethanol concentration inside cankers was 4.3 times higher than sapwood sampled 1 cm outside the canker edge, and 15.5 times the amount than in uninfected sapwood 15 and 30 cm outside the cankers edge. Sapwood 1.0 cm outside the canker edge had 3.6 times more ethanol than uninfected sapwood at 15 and 30 cm outside the edge. Sapwood samples from paired healthy check trees had low ethanol concentrations at all sample points, further confirming that high levels of ethanol, like those inside the cankers, are not normally found in healthy, uninfected *Q. agrifolia* sapwood. These results provide evidence that the *P. ramorum* infected tissues on coast live oak have the potential to release greater quantities of ethanol to the atmosphere than surrounding healthy tissues and could be responsible for the initial attraction and focused attacks of ambrosia beetles.

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BIOLOGICAL CONTROL OF TANOAK RESPROUTS USING THE FUNGUS *CHONDROSTEREUM PURPUREUM*

Marianne Elliott[1], Simon Shamoun[2], Grace Sumampong[2], Ellen Goheen[3], Alan Kanaskie[4], and Gary Chastagner[1]

In southwest Oregon, an aggressive program of cutting and burning host plants in an effort to eradicate *Phytophthora ramorum* was begun in 2001. It was soon apparent that tanoak (*Lithocarpus densiflorus*) resprouts were highly susceptible to *P. ramorum* and that infected sprouts hamper eradication efforts by maintaining inoculum on site. In Fall 2010, our research team established field trials near Brookings, Oregon to assess the bioherbicidal efficacy of the sap-rotting fungus *Chondrostereum purpureum* on tanoak to inhibit resprouts which can harbor *P. ramorum* and serve as a source of inoculum. Early results showed that *C. purpureum* was able to colonize the stumps of tanoak following treatment (Figure 1).

The basidiomycete fungus *Chondrostereum purpureum* causes a white rot of mostly broadleaf trees and has a wide host range. It invades through fresh wounds in the xylem or cut stumps and is a weak pathogen that can survive as a saprophyte. After the host tree is weakened or killed, *C. purpureum* is quickly replaced by other, more competitive decay fungi that are naturally occurring in the environment. This fungus is used as a biological control agent for woody vegetation all over the world. A preparation of mycelium of the fungus *C. purpureum* is registered under the trade name "Chontrol™ Paste" in the US and Canada for use as a biological control agent and has been tested as a stump treatment on many hardwood species (EPA Registration No. 74200-1, 2004 ; and PMRA Registration No. REG. 2004-09, 2004). Treatment of stumps with *C. purpureum* has been shown to be effective for suppression of resprouting on several species, most notably red alder (*Alnus rubra*).

METHODS

Tanoak trees with a range of diameters from 5 to 45 cm (mean 20 cm) were felled in November 2009. Seven treatments were applied to three blocks of between 18 and 21 trees per treatment. The treatments were assessed approximately one and two years after application in September 2010 and 2011. Number of live sprout clumps, number of sprout clumps dead or with dieback, height of the tallest sprout, sprout clump width, and stump diameter were measured. In addition, presence of *C. purpureum* or other decay fungi was noted.

Figure 1: *Chondrostereum purpureum* fruiting in 2010 on stump treated with Chontrol™ liquid + inoculum.

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[1]Washington State University, Puyallup Research and Extension Center, Puyallup, WA. [2]Natural Resources Canada, Pacific Forestry Centre, Victoria, BC. [3]USDA Forest Service, Forest Health Protection, Central Point, OR. [4]Oregon Department of Forestry, Salem, OR.

RESULTS

There was a positive correlation between stump diameter and number of live sprout clumps (R^2 = 0.685 in 2010, 0.553 in 2011). There was no significant difference in stump diameter among the treatments. Mean stump diameter was 20 cm (range 5 – 45 cm). Fewer live sprout clumps were found on tanoak stumps that received the inoculum treatments in 2011 but these differences were not significant. The two herbicide treatments had the fewest live sprout clumps. Stumps with the hack and squirt herbicide treatment had no live sprouts in both 2010 and 2011. Stumps treated with Chontrol™ formulations with and without inoculum had more dead sprouts than in the Garlon spray treatment, where there were more live sprouts in 2011.

Fruiting bodies of fungi observed on decaying tanoak logs and stumps were collected and taken to WSU-Puyallup. These fungi were cultured on basidiomycete selective media. PCR of the ITS rDNA region was done on cultures and fruiting bodies and the PCR product was sequenced. A BLAST search was done on each sequence and the fungi were identified based on these results and observations of the fruiting body morphology. We will use markers developed for the strain PFC 2139 to determine if *C. purpureum* isolated from treated stumps is naturally occurring or is identical to the isolate originally applied during treatment.

Basidiomycete fungi from other sites collected were *Chondrostereum purpureum, Lenzites betulinum, Stereum hirsutum*, and *Trametes versicolor.* Of these fungi, *C. purpureum, L. betulinum*, and *T. versicolor* are not reported on tanoak in the SMML Fungus-Host database (http://nt.ars-grin.gov/fungal databases/fungushost/fungushost.cfm).

CONCLUSIONS

Chontrol™ formulations appear to have some effect on reducing resprouting in tanoak, but the most effective treatment is the hack and squirt method of applying the herbicide imazapyr. Over time, applications of Chontrol™ may be a more permanent solution as the stumps become decayed. Monitoring these field trials for a third year will give us better results for the bioherbicidal efficacy of Chontrol™ on tanoak resprouts.

If a formulated product of *C. purpureum* and/or its mixture with other stem and wood decay fungi applied to tanoak stems and stumps does inhibit the trees from growing new sprouts, this *P. ramorum* inoculum reservoir would be reduced or eliminated in the ecosystem. In areas where the application of herbicides is not prudent or not permitted, this biocontrol treatment would be an indispensable alternative to chemical herbicides.

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ASSESSING THE ROLE OF VERTICILLIUM WILT IN BIGLEAF MAPLE (*ACER MACROPHYLLUM*) DIEBACK IN WESTERN WASHINGTON

Daniel Omdal[1] and Amy Ramsey-Kroll[1]

INTRODUCTION

Bigleaf maples are an important ecosystem component in the forests of western Washington, providing shade, food, habitat and structural diversity in riparian and upland areas. Over the past several years, concerned landowners, the general public and forest land managers have contacted University of Washington, WA Dept. of Natural Resources (DNR) and US Forest Service (USFS) forest health specialists about increased levels of bigleaf maple decline and dieback observed in western Washington. Symptoms included yellow flagging of large branches, small leaf size, and partial or entire crown dieback. A limited number of site visits and drive-by observations have been made without any conclusive evidence about what is causing the dieback.

Verticillium albo-atrum and *Verticillium dahliae*, causal agents of Verticillium wilt, are reported as damaging agents of bigleaf maple (Minore and Zasada 1990). *V. albo-atrum* and *V. dahliae* are soilborne fungi that invade the xylem of host trees and can cause leaf drying, leaf curling, defoliation, wilting, dieback and tree death (Sinclair et al. 1987). This project investigated whether or not Verticillium wilt was the primary cause of bigleaf maple decline and dieback in western Washington.

METHODS

Sixty-one sites were surveyed in July and August, 2011. A symptomatic bigleaf maple, or a bigleaf maple with yellow flagging of large branches, small leaf size and partial or entire crown dieback, was the site center. At each site center, GPS coordinates, elevation, slope position, aspect, basal area and soil type were recorded. Individual tree characteristics were also recorded, along with up to five symptomatic branch samples from each tree. Branch samples were submitted to the Washington State University Puyallup Plant and Insect Diagnostic lab for processing and microscopic examination for *V. albo-atrum* and *V. dahliae*.

RESULTS

All submitted symptomatic bigleaf maple samples were negative for *V. albo-atrum* and *V. dahliae*. 15% of the symptomatic trees had signs of other root diseases, such as *Armillaria* spp. and *Ganoderma* spp.

DISCUSSION

Based on our 2011 sampling and results, we conclude that the dieback occurring in bigleaf maple in western Washington is being caused by something other than Verticillium wilt. None of the samples we submitted for analysis were positive for *Verticillium* spp. Signs of other root diseases were found in a portion of the trees surveyed, but these results do not suggest that either Armillaria or Ganoderma root diseases are the primary causal agents of bigleaf maple dieback in western Washington.

Dialogue among DNR and USFS forest health specialists in California, Oregon and Washington about the bigleaf maple dieback issue, has raised awareness of another pathogen affecting bigleaf maple in California. *Xylella fastidiosa,* a bacteria*,* is causing maple leaf scorch in northeast California (Woodruff 2011). Xyllela fastidiosa is transmitted from diseased to healthy plants by insects with piercing and sucking mouthparts. The bacteria is

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limited to the xylem and can cause blockage in the water conducting system of the plant. Samples from Washington were sent for *Xyllela* analysis and results are still pending.

REFERENCES

Minore, D., Zasada, J.C. 1990. *Acer macrophyllum*. Pages 33-40 in R.M. Burns and B.H. Honkala (technical coordinators) Silvics of North America, Vol. 2. Agricilture Handbook 654, USDA Forest Servvice. Washington, D.C.

Sinclair, W.A., Lyon, H.H., Johnson, W.T. 1987. Diseases of trees and shrubs. Cornell. University Press, Ithaca, NY. 574 p.

Woodruff, B. 2011. *Xylella fastidiosa* confirmed on bigleaf maple in Northern California. USDA Forest Health Protection, Report # NE11-16.

DNA-BASED IDENTIFICATION OF *ARMILLARIA* ISOLATES FROM PEACH ORCHARDS IN MÉXICO STATE

Rubén Damian Elias Roman[1,5], Ned B. Klopfenstein[2], Dionicio Alvarado Rosales[1], Mee-Sook Kim[3], Anna E. Case[2], Sara M. Ashiglar[2], John W. Hanna[2], Amy L. Ross-Davis[2,4], and Remigio A. Guzmán Plazola[1]

ABSTRACT

A collaborative project between the Programa de Fitopatología, Colegio de Postgraduados, Texcoco, Estado de México and the USDA Forest Service - RMRS, Moscow Forest Pathology Laboratory has begun this year (2011) to assess which species of *Armillaria* are causing widespread and severe damage to the peach orchards from México state, México. We are employing a DNA-based approach in which several genes will be sequenced and compared to known *Armillaria* species.

BACKGROUND

Peach cultivation was first introduced to México by Spanish explorers in the sixteenth century and now constitutes an important crop with approximately 40,500 ha currently devoted to peach production (Anonymous 2011). Most production consists of the clingstone type of peach, which has been grown in the highlands of central México for decades (around 60% of national production). In México state, approximately 3,000 ha of peach orchards are typically established in areas where native forests are cleared. These orchards are relatively short-lived, with a production life span of about 10 years. *Armillaria* is one of the most damaging pathogens of peach trees in México, where it causes significant annual mortality of orchard-grown peach trees.

OBJECTIVES

The objective of this research is to use DNA–based methods to identify *Armillaria* isolates collected from peach trees growing in orchards throughout México state for comparisons with *Armillaria* spp. from native forests. This information will be used to help develop species-specific, *Armillaria*-resistant peach rootstocks, and evaluate potential invasiveness of these *Armillaria* species.

METHODS

Armillaria isolates (n = 62) were collected from 15 peach orchards located throughout México state (Fig. 1). For each of these isolates, up to four loci are being sequenced (ITS, IGS-1, LSU-D, and EF1-α).

RESULTS AND DISCUSSION

Based on IGS-1 sequence alignments, three species of *Armillaria* have been isolated from infected peach trees: *A. mellea*, *A. gallica*, and an undescribed species. These *Armillaria* species are not the same as the species typically associated with Armillaria root disease in the southeastern USA (*A. tabescens*) and may require the development of specialized root stock for resistance (Beckman 2007).

Understanding the distribution of *Armillaria* spp. in México is critical toward predicting potentially invasive *Armillaria* pathogens for the USA and México. This information will also lay a foundation for predicting potential influences of climate change on *Armillaria* root disease in the USA and México.

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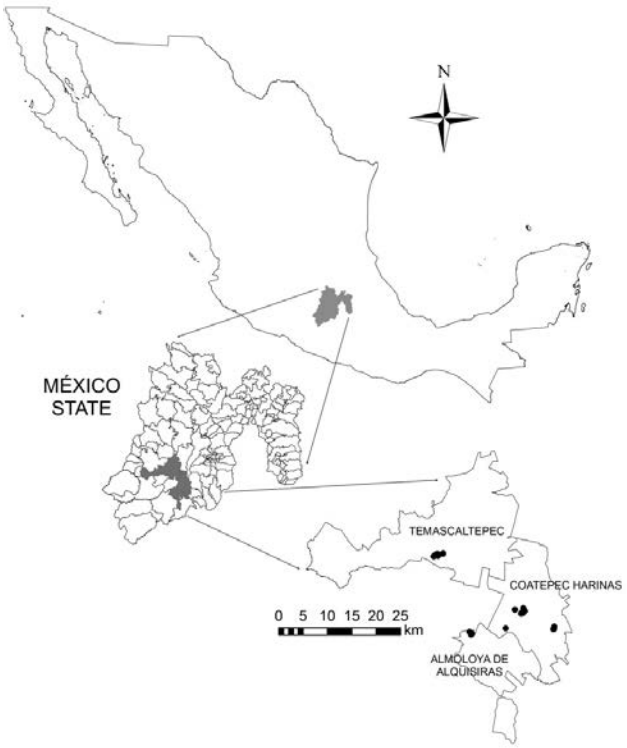

Figure 1: Map showing the location of *Armillaria* collection sites within México state, México (top); Peach trees infected with *Armillaria* spp. (bottom).

REFERENCES

Anonymous. 2011. SAGARPA Database-avance de siembras y cosechas. http://www.siap.sagarpa.gob.mx/.

Beckman, T.G. 2007. Managing peach tree short life and Armillaria root rot in peach orchards. Pages 28-41 In: Proceedings of 3rd National Peach Congress, December 6-8, 2007, Ixtapan de la Sal, Mexico.

OCCURRENCE OF THE ROOT ROT PATHOGEN, *FUSARIUM COMMUNE*, IN MIDWESTERN AND WESTERN UNITED STATES

J.E. Stewart[1*], R.K. Dumroese[2], N.B. Klopfenstein[2] and M.-S. Kim[3]

ABSTRACT

Fusarium commune can cause damping-off and root rot of conifer seedlings in forest nurseries. The pathogen is only reported in Oregon, Idaho, and Washington within United States. *Fusarium* isolates were collected from midwestern and western United States to determine occurrence of this pathogen. DNA sequences of mitochondrial small subunit gene were used to identify *F. commune*. In addition to the aforementioned states, *F. commune* was found from nurseries in Nevada, Montana, Nebraska, and Michigan, USA.

BACKGROUND

The genus *Fusarium* is ubiquitous in most container and bareroot nurseries on healthy and diseased conifer seedlings, in nursery soils, and on conifer seeds of several species, especially Douglas-fir (*Pseudotsuga menziesii*), western white pine (*Pinus monticola*), and ponderosa pine (*Pinus ponderosa*) (James et al. 1990). Since the first report of this Fusarium root rot in forest nurseries, the major pathogen was previously identified as *Fusarium oxysporum* based on morphology (Bloomberg 1981). However, selected *Fusarium* spp. isolates that had previously been characterized as pathogenic on Douglas-fir seedlings displayed a range of high, moderate, and low virulence (Stewart et al. 2006). Stewart et al. (2006) showed that all the highly virulent isolates were identified as *F. commune*, a recently named species (Skovgaard et al. 2003), based on DNA sequences. DNA sequences from the mitochondrial small subunit (mtSSU) and elongation factor-1α (EF-1α) regions are useful for distinguishing *F. commune* from *F. oxysporum*.

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MATERIALS AND METHODS

A total of 260 isolates of *Fusarium* spp. were collected in forest nurseries throughout midwestern and western United States (Table 1). Isolates from each state were collected from one to five forest nurseries. *Fusarium* isolates were collected from diverse sources of host/substrate: diseased or healthy seedlings of Douglas-fir, western larch (*Larix occidentalis*), western redcedar (*Thuja plicata*), Pacific yew (*Taxus brevifolia*), lodgepole pine (*Pinus contorta*), western hemlock (*Tsuga heterophylla*), western white pine, ponderosa pine, grand fir (*Abies grandis*), rabbitbrush (*Chrysothamnus* sp.), sagebrush (*Artemisia* sp.), Austrian pine (*Pinus nigra*), blue spruce (*Picea pungens*), bitterbrush (*Purshia tridentata*), and eastern redcedar (*Juniperus virginiana*), containers of various conifer seedlings, and soil/growing medium.

Table 1: Number of *Fusarium* spp. isolates collected from midwestern and western United States.

State	Total # of isolates
Washington	31
Oregon	79
California	43
Idaho	56
Nevada	13
Montana	14
Utah	12
Nebraska	7
Michigan	5

Molecular species identification. All 260 isolates were characterized using mtSSU sequences. Template DNA was derived from scrapings of actively growing mycelial cultures (3-5 days old), so

no DNA extractions were performed. The PCR products were sequenced with an ABI 3700 DNA Sequencer (Applied Biosystems, Inc.) at the University of Wisconsin – Biotechnology Center (Madison, WI, USA) and the sequences of mtSSU region were blasted to GenBank database.

Confirmation of species identification. Phylogenetic analyses were performed on the 29 *F. commune* isolates from this study, along with previously identified *F. oxysporum* and *F. commune* isolates (Figure 1; Stewart et al. 2006). Isolates of *F. subglutinans* (NRRL 22016: M1431/AF160289), *F. proliferatum* (NRRL22057: M1431/M1432) were included in the analyses as an outgroup. Sequences of *F. proliferatum* and *F. subglutinans* were retrieved from TreeBASE matrices (M1431, M1432) (www.treebase.org). Phylogenetic analyses were conducted using PAUP*4.0b10 (Swofford 2003) and MrBayes v.3.0b4 (Huelsenbeck and Ronquist 2001.)

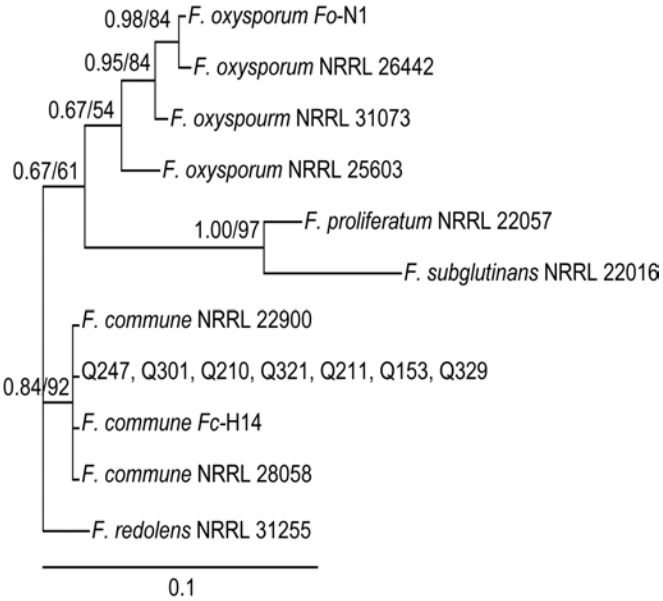

Figure 1: Phylogenetic analyses based on mtSSU region showed that the 29 *F. commune* from this study grouped with the previously identified *F. commune* (Stewart et al. 2006), which is genetically distinct from *F. oxysporum*. Isolates Q247 (WA), Q301 (OR), Q210 (ID), Q321 (NV), Q211 (MT), Q153 (NE), and Q329 (MI) represent strains from each state. Node confidence levels are shown by posterior probability (left) and bootstrap (right) support.

RESULTS AND DISCUSSION

A total of 29 *F. commune* isolates was identified based on GenBank BLAST search using mtSSU sequences from Washington, Oregon, Nevada, Montana, Michigan, and Nebraska (Figure 2). Previous studies also identified *F. commune* in Idaho, Oregon, and Washington (Leon 2009; Skovgaard et al. 2003; Stewart et al. 2006). No *F. commune* was found in California or Utah. A total of 43 isolates was collected from California representing three nurseries, but all are *F. oxysporum*. *Fusarium* isolates representing one nursery in Utah also did not contain any *F. commune*. The majority of *Fusarium* isolates derived from midwestern and western United States were identified as *F. oxysporum* based on mtSSU sequences. Several isolates of *F. redolens* and a couple of *F. solani* were also identified (data not shown). Results from this study indicate that *F. commune* occurs widely throughout the midwestern and western United States. More intensive studies are needed to better characterize the distribution and host range of *F. commune* in tree nursery settings.

ACKNOWLEDGEMENTS

The project was supported by the USDA Forest Service-RMRS. We greatly appreciate Drs. Robert L. James and Jeffrey K. Stone for providing *Fusarium* isolates and John W. Hanna for technical assistance.

REFERENCES

Bloomberg, W.J. 1981. Disease caused by *Fusarium* in forest nurseries. Pages 178--187 In: PE Nelson, TA Toussoun and RJ Cook, eds., *Fusarium*: Diseases, Biology, and Taxonomy. Pennsylvania State University Press, University Park, PA.

Huelsenbeck J.P., Ronquist F. 2001. MrBayes: Bayesian inference of phylogenetic trees. Bioinformatics. 17:754-755.

Leon A.L. 2009. Chemical alternatives to methyl bromide for control of *Fusarium oxysporum* and *Fusarium commune* in Douglas-fir nurseries in the Pacific Northwest. MS Thesis, University of Washington, Seattle, WA.

James R.L., Dumroese R.K., Wenny D.L. 1990. Approaches to integrated pest management of Fusarium root disease in container-grown seedlings. Pages 240-246 In: Rose SJ, TD Landis, eds., Proceedings of the Western Forest Nursery Association and Intermountain Nursery Association: Target seedling symposium, USDA Forest Service, GTR RM-200. Fort Collins, CO.

Skovgaard K., Rosendahl S., O'Donnell K., Hirenberh H.I. 2003. *Fusarium commune* is a new species identified by morphological and molecular phylogenetic data. Mycologia. 95:630-636.

Stewart J.E., Kim M-S., James R.L., Dumroese R.K., Klopfenstein N.B. 2006. Molecular characterization of *Fusarium oxysporum* and *F. commune* isolates from a conifer nursery. Phytopathology. 96:1124-1133.

Swofford D.L. 2003. PAUP*: phylogengentic analyses using parismony*and other methods version 4.0. Sinauer Association, Sunderland, MA.

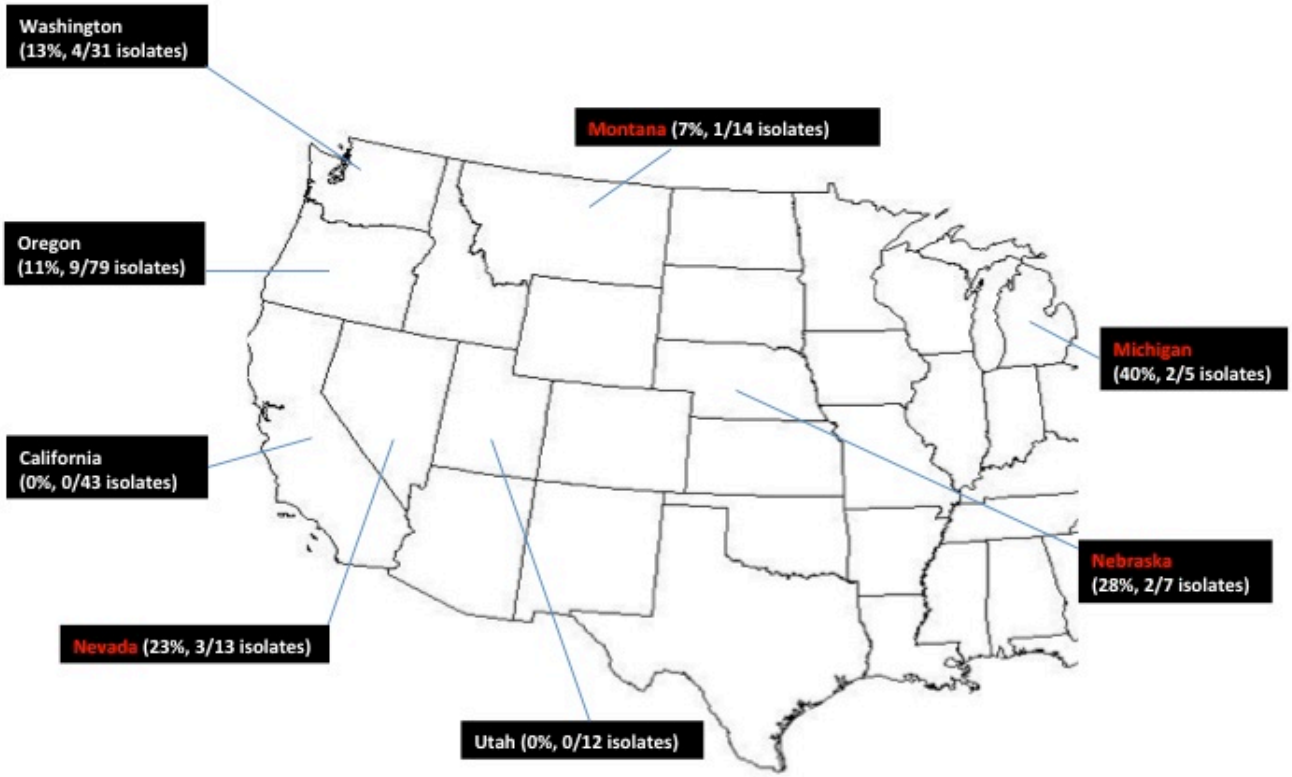

Figure 2: New reports of *F. commune* in four states: Nevada, Montana, Nebraska, and Michigan, USA.

DE NOVO ASSEMBLY AND TRANSCRIPTOME CHARACTERIZATION OF AN *ARMILLARIA SOLIDIPES* MYCELIAL FAN

Amy L. Ross-Davis[1], Jane E. Stewart[2], John W. Hanna[1], Mee-Sook Kim[3], Rich C. Cronn[4], Hardeep S. Rai[5], Bryce A. Richardson[6], Geral I. McDonald[1], and Ned B. Klopfenstein[1]

INTRODUCTION

Armillaria (Fr.) Staude is a widely distributed fungal genus comprising approximately 40 species (Volk and Burdsall 1995) that display diverse ecological behaviors ranging from beneficial saprobe to virulent pathogen. *Armillaria solidipes* (formerly *A. ostoyae*; Burdsall and Volk 2008; pending vote to conserve *A. ostoyae*; Redhead et al. 2011), one of the causal agents of Armillaria root disease, is a virulent primary pathogen with a broad host range in northern temperate latitudes (Kile et al. 1991). This fungal pathogen attacks sapwood as mycelial fans under the bark, and grows between trees as rhizomorphs. The pathogen causes a white rot of infected wood and is responsible for reduced forest yields as a result of direct tree mortality and non-lethal cryptic infections (Cruickshank et al. 2011).

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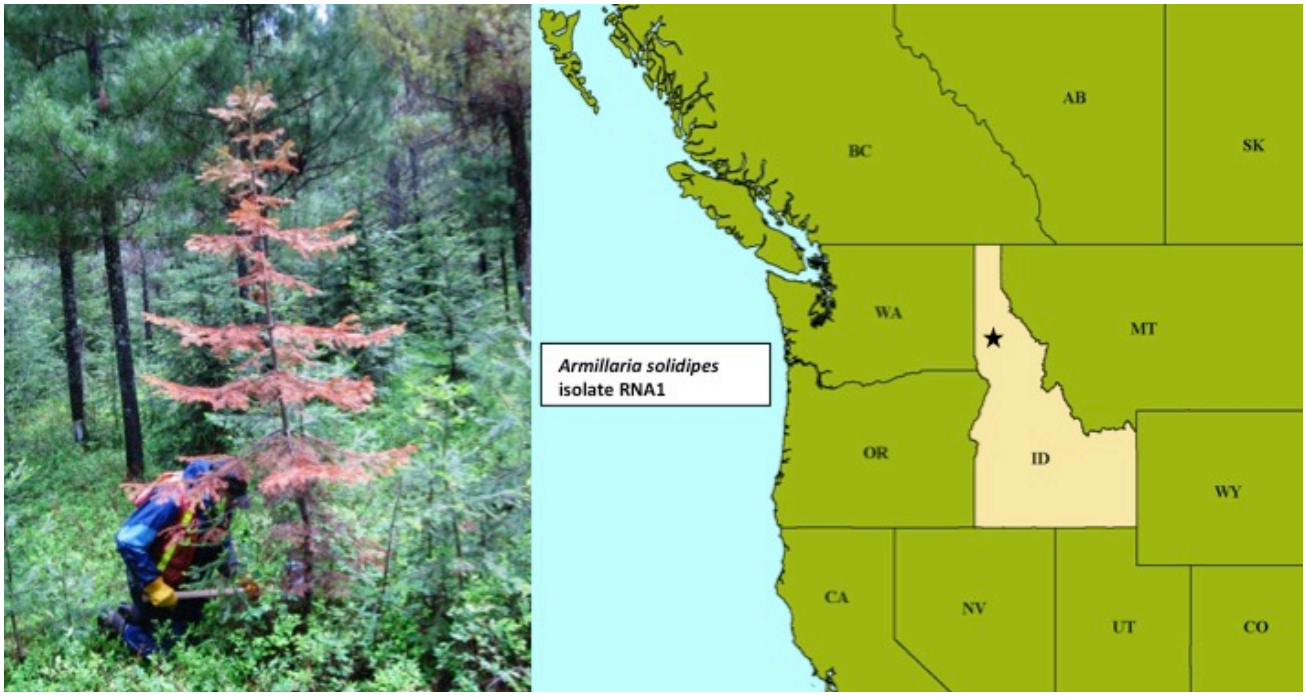

Figure 1: Diseased grand fir (*Abies grandis*) infected with *Armillaria solidipes* (left) collected near Elk River, Idaho (right).

The purpose of this research is to present the first assembly and characterization of a transcriptome from a root disease pathogen during pathogenesis. Specifically, our objective is to identify a large number of genes expressed by an active mycelial fan of *A. solidipes*, focusing on genes that may be associated with pathogenicity (i.e., those that result in a loss or reduction in disease symptoms when disrupted). Detection of putative genes that show homology to annotated genes involved in infection, cuticle and cell wall degradation, response to host environment, production of fungal toxins, and signaling will ultimately help inform forest management decisions.

METHODS

We assembled and characterized a transcriptome of an active mycelial fan of *Armillaria solidipes* infecting *Abies grandis* near Elk River, Idaho (Figure 1; Table 1). The stand from which this isolate was collected has been well-characterized in that genets have been mapped and collected from several different hosts over several years.

Table 1: Assembly statistics.

	Count	Average length	Total bases
Reads	24,166,534	76.77	1,855,146,290
Matched	20,281,443	76.77	1,556,994,617
Not matched	3,885,091	76.74	298,151,673
Contigs	39,943	551	22,027,774

cDNA was generated from polyA$^+$ purified total RNA and then sequenced using a paired-end read approach on the Illumina GAII platform. A total of 24,166,534 reads was generated and assembled *de novo* into 39,943 contigs using the CLC Genomics Workbench 4.7.2. Significant alignments were identified using the NCBI NR database using a BLASTx search with a threshold expectation or e-value of 1e^{-5}. Hits were coded by taxon (Figure 2) and functional annotations relating to pathogenicity were assigned if known (Table 2). Signal peptides were identified using SignalP 4.0.

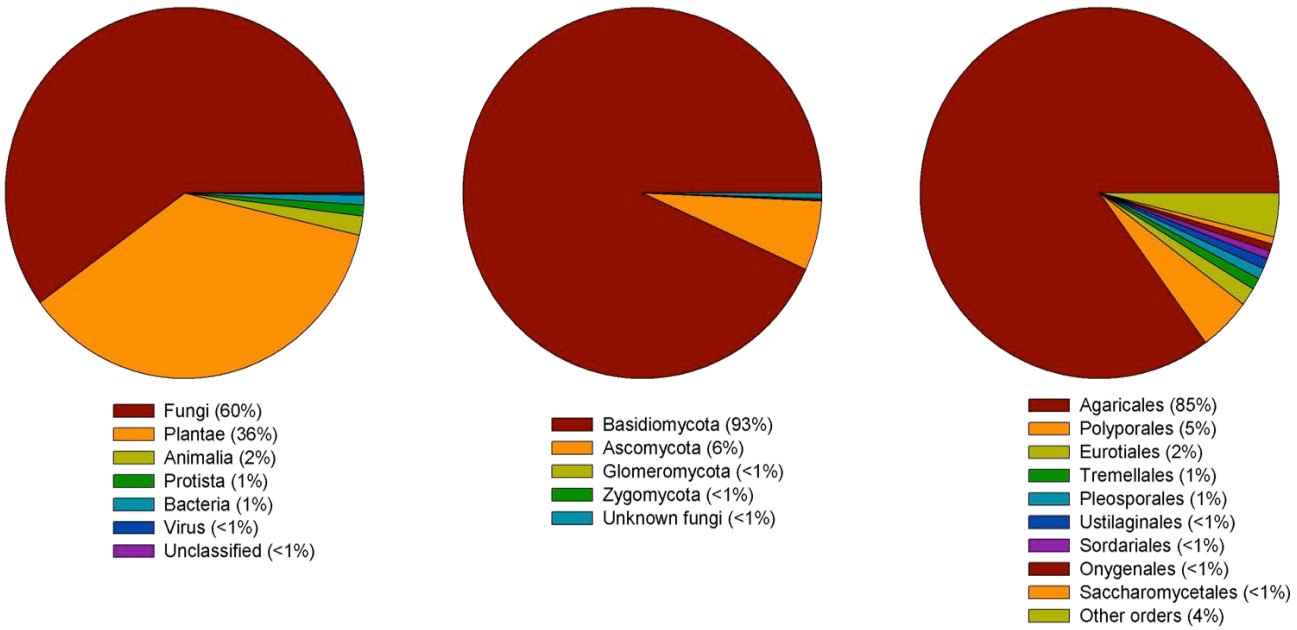

Figure 2: Distribution of significant alignments across kingdoms (A) fungal phyla (B) and fungal orders (C).

Table 2: Alignments with annotated pathogenicity genes (Oliver and Osbourn 1995; Idnurm and Howlett 2001; Tudzynski and Sharon 2003).

Class	Function	Hits
Infection	Host-surface recognition	1 (FUN34 transmembrane protein)
Cell-wall degrading enzymes	Cellulose and hemicellulose degradation	11 (glucanase and cellobiohydrolase) 3 (xylanase) 58 (glycoside hydrolase)
	Lignin degradation	26 (peroxidase and laccase)
	Pectin degradation	1 (pectate lyase)
Response to host	Toxin efflux	11 (ABC transporters)
Fungal toxins	Toxin biosynthesis	3 (branched-chain-amino-acid transaminase)
Signal cascade components	Change gene expression in response to environment	2 (MAP kinase) 5 (G proteins)

RESULTS AND DISCUSSION

***De Novo* Assembly and Transcriptome Characterization.** Of the 39,943 assembled contig sequences, 8,747 had significant alignments when compared against the NCBI NR database using BLASTx ($<1e^{-5}$). Of these, most contigs aligned best with gene sequences characterized from fungi (60%) or from plants (36%; Figure 2a). Of those sequences that best aligned with fungal sequences, 93% of hits fell within the Basidiomycota, 6% within Ascomycota and < 1% within the Glomeromycota and Zygomycota, respectively (Figure 2b). Most of the hits to Basidiomycota were to Agaricales, the order into which *A. solidipes* is placed (Figure 2c). Only 233 of the 8,747 contigs best matched sequences from *Armillaria* species; however, these results largely reflect sequence availability in GenBank.

Identification of Genes Associated with Pathogenicity. A total of 19,792 signal peptides were identified from the 39,943 assembled contig sequences. These short peptide chains direct the transport of proteins across membranes; thus, many are likely involved in pathogenicity. Several significant alignments with annotated genes involved in pathogenicity were identified from the transcriptome (Table 2).

Next, we intend to determine the extent (i.e., geographic coverage and infected host species) of this genet through pairing tests with other isolates collected from this well-mapped stand and examine the distribution of genes in the transcriptome assembly assigned to broad gene ontology categories.

ACKNOWLEDGEMENTS

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REFERENCES

Burdsall, H.H. Jr, Volk, T..J. 2008. *Armillaria solidipes*, an older name for the fungus called *Armillaria ostoyae*. North American Fungi. 3:261–267.

Cruickshank, M.G., Morrison, D.J., Lalumière, A. 2011. Site, plot, and individual tree yield reduction of interior Douglas-fir associated with non-lethal infection by Armillaria root disease in southern British Columbia. Forest Ecology and Management. 261:297–307.

Idnurm, A., Howlett, B.J. 2001. Pathogenicity genes of phytopathogenic fungi. Molecular Plant Pathology. 2:241-255.

Kile, G.A., McDonald, G.I., Byler, J.W. 1991. Ecology and Disease in Natural Forests. Chapter 8 in: Shaw CG III and Kile GA. *Armillaria* root disease. USDA Forest Service, Agriculture Handbook No. 691. Washington, DC. 233 pp.

Oliver, R., Osbourn, A.E. 1995. Molecular dissection of fungal phytopathogenicity. Microbiology. 141:1-9.

Redhead, S.A., Bérubé, J., Cleary, M.R., and others. 2011. (2033) Proposal to conserve *Armillariella ostoyae* (*Armillaria ostoyae*) against *Agaricus obscurus*, *Agaricus occultans*, and *Armillaria solidipes* (Basidiomycota). Taxon. 60(6):1770-1771.

Tudzynski, P., Sharon, A. 2003. Fungal pathogenicity genes. Applied Mycology and Biotechnology. 3:187-212.

Volk, T.J., Burdsall, H.H. Jr. 1995. A nomenclatural study of *Armillaria* and *Armillariella* species. Synopsis Fungorum. 8:1–121.

COMMITTEE REPORTS

FOLIAGE AND TWIG DISEASE COMMITTEE REPORT

Committee Chair – Harry Kope (Alex Woods filled in for Harry)

PRESENTATIONS

TOP DIEBACK OF LODGEPOLE PINE IN CENTRAL, BC
Alex Woods, BC Forest Service, Smithers BC.

In mid-summer of 2011, BC Forest Service in Burns Lake BC was preparing to initiate an aerial fertilization program. The field staff noticed that the stands they were planning to fertilize were suffering leader dieback. The damage appeared to progress quickly over several weeks. The tops of 25-35 year old lodgepole pine were suffering as much as 2-3 m top dieback. At first it was thought that the damage was the result of scleroderris canker but samples sent to the lab came back with another weak pathogen, *Cenangium ferruginosum*. According to Sinclair and Lyon (2nd ed.), *Cenangium ferruginosum* is common after drought or after an unusually cold winter, especially if a severe winter is preceded by an unusually mild autumn. The summer of 2010 was particularly dry throughout BC and in November of 2010 temperature in Burns Lake BC went from a high of +11C to a low of -29C over a period of 4 days. This looks like another unpredictable forest disease condition linked to an erratic weather pattern.

EUROPEAN ASH DECLINE
Michelle Cleary, Swedish University of Agricultural Sciences (SLU), Uppsala, Sweden.

Extensive dieback of European ash (*Fraxinus excelsior*) has been increasingly observed across Europe during the last decade. In Sweden, the disease was first noted in 2001. Since 2004 it has spread throughout the natural range of ash in the country. To date 24 countries have reported damage from the pathogen. Trees of all age classes are affected. Symptoms include necrotic lesions (cankers) on stems and branches, wilting and premature shedding of leaves, dieback of branches, shoots, and twigs, crown dieback, grey-brown discoloration in the wood, epicormic branching as the tree attempts to recover its crown, and eventual mortality. The causal agent *Chalara fraxinea* has been identified quite recently (Kowalski 2006). The primary infection court appears to be petioles of leaves. The emergence of this disease is difficult to explain, but its stepwise pattern of geographic spread suggests the behaviour to be similar to an alien or invasive fungus spreading through Europe at an alarming rate and threatening the existence of ash across the continent. European ash has recently (2010) been red-listed as an endangered species largely due to this pathogen. There is a threat to North American ash species from the accidental importation of European Ash decline and researchers do not know how far this pathogen could spread.

MADRONE LEAF BLIGHT
Marianne Elliot, Washington State University, Puyallup, WA.

This foliar disease was particularly bad in 2011 following a pattern first seen in 2009, the first year severe decline damage was observed. Of the leaf spots that were assessed using standard isolation techniques, 41% could be identified as *Phacidiopycnis washingtonensis*. After further sampling it was found that the same fungus could be isolated from all plant parts including old leaves, new leaves and bark which suggests that the fungus could be an endophyte. The same fungus also causes rot on apples in cold storage so it is thought that cool temperatures favour its development. Based on that potential environmental trigger for this pathogen an examination of recent weather was conducted. Two periods of extreme cold temperatures in November and February of the years previous to the most significant decline suggest that these cold snaps may be the cue to initiate the decline like symptoms in madrone. Further work is being conducted on identifying the most important foliar disease fungi in madrone.

SWISS NEEDLE CAST UPDATE

Alan Kanaskie and Mike McWilliams, Oregon Dept. of Forestry, Salem, OR and Dave Shaw, Oregon State University, Corvallis, OR.

Aerial surveys with accompanying ground checks are being continued in Oregon and expanded into Washington. The 2011 aerial survey has been conducted in Oregon where over 440,000 acres were detected with visible symptoms of disease, the most since the aerial survey began in 1996. The disease continues to intensify on the coast. There is a need for a more consistent survey approach in Washington. Cool wet spring weather has made it difficult to get the surveys done and has also probably exacerbated the disease condition. The Swiss Needle Cast Cooperative continues to maintain a website with lots of information and a complete bibliography of *P. gaeumannii* papers: http://sncc.forestry.oregonstate.edu/.

ROUND ROBIN OF TABLES AND NOTABLE NOTES

MAPLE LEAF SCORCH

Bill Woodruff, USDA, Susanville, CA.

Damage caused by this disease was first noticed in a significant amount in 1998 but it has been tracked since 1984. The symptoms include very small leaves, some progressive dieback and in some cases mortality. As of 2008/2009 the causal organism has been identified as a bacterium, *Ziella pestilosa*.

BOTRYOSPHAERIA CANKER IN OAKS

Paul Zambino, USDA, San Bernadino, CA.

Botryosphaeria canker in coastal live oak has been found to be widespread with up to 40-50% mortality occurring in some areas of California.

DOTHISTROMA NEEDLE BLIGHT IN THE UNITED STATES

Jim Walla, North Dakota State University, Fargo, ND.

Dothistroma needle blight has been reported from 35 states but recent molecular analysis shows that traits previously used to identify species are not valid, so a reassessment of the geographic and host ranges are needed. A request has been made to people throughout the USA to collect samples of needles with fruiting bodies. Genetic analysis work to determine which species of Dothistroma is present and where is being conducted in conjunction with Irene Barnes. A similar analysis in Europe has shown that both species, *D. septosporum* and *D. pini,* are present there.

NEW RELATIONSHIP BETWEEN SCALE INSECT AND PATHOGENIC BACTERIA

Bill Jacobi, Colorado State University, Fort Collins, CO.

A new relationship between a scale insect and a pathogenic bacterium has been found in red oaks. *Brenneria quercina* has been identified as the causal bacterial agent. The symptoms include oozing wounds on the bark of the tree trunk.

***SEPTORIA MUSIVA* IN BC**

Stefan Zeglen, BC Forest Service, Nanaimo, BC.

Septoria musiva canker of poplars was first identified in BC in 2007 in a forest nursery in the Fraser Valley. There was concern that this introduced pathogen could make the leap from hybrid poplar plantations to wild cottonwood populations. This leap appears to have taken place and the pathogen has been found on native black cottonwood. We are now trying to determine the extent to which the pathogen has spread. The symptoms of *S. musiva*, which can cause severe bark cankers and weakening of the trunk, are masked by those of the native pathogen *S. populicola* which causes very minor damage on foliage. Work is being done to try to identify how this non-native species of *Septoria* arrived in BC and to develop genetic tests for more rapid and accurate identification.

RUST COMMITTEE MEETING REPORT

Committee Chair - Holly Kearns

Forty members attended the Rust Committee lunch on Tuesday, October 11, 2011 in Leavenworth, WA. The agenda consisted of individual reports on findings and discussions of on-going rust-related projects, research updates, collaborative needs, etc. The following are brief summaries:

Alex Woods (BC Ministry of Forests; Smithers, BC) reported on a landscape level hard pine rust survey conducted in the Lakes Timber Supply Area of central BC in 2011. A randomly selected sample of 70 lodgepole pine leading stands aged 10-25 was assessed. Virtually all of the stands (99%) contained at least some rust infected trees but more importantly 43% of the stands had combined rusts incidence (western gall rust, comandra blister rust and stalactiform blister rust) greater than 20%. The highest incidence recorded for any stand was 59% comandra blister rust infection. Most of the stands still have adequate healthy stocking despite the high rust incidence. This survey was done as part of a larger examination of the potential to develop hazard and risk ratings for rusts in the central interior of BC. There are mixed opinions as to the viability of a hazard and risk rating for a group of pathogens that are so climatically driven. The landscape level incidence of hard pine rusts in the central interior of BC has changed significantly since the 1980s. This change in incidence levels has coincided with an increasing trend of warmer overnight minimum summer temperatures and more summer days with rain. Any hazard and risk rating developed under current conditions could soon become invalid as the climate continues to change.

John Schwandt (Forest Health Protection; Coeur d'Alene, ID) described several monitoring projects related to white pine blister rust. Projects in Western white pine include*:* analyzing data from pruning and thinning plots 40 years following treatment; installing pruning and thinning monitoring plots in new F_2 plantations; completing a 15 year remeasurement in 22 F2 plantations without pruning or thinning to evaluate long term survival, mortality, and rust infection levels. Projects in Whitebark pine include*:* analyzing 15 years of data from whitebark pine regeneration plots in 5 areas; 400 plots in day-lighting area to monitor tree and rust response to two different day-lighting regimes on three height classes of regeneration; continuing to monitor direct seeding trials. The whitebark pine restoration program funded 35 projects in 2011 with nearly $500,000.

Dan Omdal and **Amy Ramsey-Kroll** (Washington Department of Natural Resources; Olympia, WA) installed six long-term genetically enhanced western white pine field trials in 2006 in western Washington. Each of the six sites has 7 replications and up to 36 planted western white pine families. 2011 was the 2nd year of white pine blister rust assessments. Results to date show that the control families have high levels of blister rust infection, but there is currently little to no mortality from white pine blister rust, depending on the site.

Robin Mulvey (Forest Health Protection; Juneau, AK) has been working with the Hansen Lab at OSU to sequence a variety of rust species from Alaska in an effort to develop a rust reference collection. Several pathogens that could become introduced/invasive in Alaska are rust species, and the goal of this project is to develop a database of the rust pathogens present and presumably native to Alaska. The need for this work was highlighted in August 2011, when large quantities of rust spores washed up in the remote NW Alaska village of Kivalina, an outbreak that was not detected by aerial surveys in July. Cooperative work is underway with NOAA and Agathe Vialle at Natural Resources Canada to identify the rust to species using scanning-electron micrographs of the spores and genetic sequence information; samples were highly degraded and ITS sequencing has been unsuccessful. The morphology of the rust spores are consistent with the genus *Chrysomyxa*, and two likely candidate species are *Chrysomyxa ledicola* (spruce needle rust, alternates on Labrador tea) and *C. pyrolata* (spruce cone rust, alternates on plants in the Pyrolaceae).

Michael Murray (BC Ministry of Forests; Nelson, BC) is conducting the first-ever formal blister rust screenings for whitebark pine in BC. A total of about 45 putatively resistant parent trees were selected from the Kootenay region of British Columbia (near the WA, ID, MT borders) during the 2011 field season. The Kalamalka Research Center (BC Forest Service) will propagate and rear the seedlings (ca. 150/parent). An inoculation plan is not yet finished, but may include two field trials plus an indoor trial. A subset of seeds from 10 parents is undergoing a separate screening at the Dorena Tree Improvement Center (US Forest Service). This trial will provide a comparison (for the 10 parents) with the parallel screenings conducted in BC.

Ned Klopfenstein (Rocky Mountain Research Station; Moscow, ID) reported that the research group in Moscow is involved in an international collaboration among Brazil, Paraguay, Uruguay, Argentina, Colombia, Costa Rica, Mexico, Australia, and the USA investigating the worldwide population structure of the invasive guava-eucalypt-'ohi'a-myrtle rust pathogen (*Puccinia psidii*). Studies are directed toward assessing pathways of spread and identifying global areas at risk.

International studies are continuing to assess evolutionary relationships among international sources of *Cronartium ribicola*, and the studies on range-wide genetic diversity of western white pine were recently published.

Recent publications:

Graça, R.N.; Ross-Davis, A.L.; Klopfenstein, N.B.; Kim, M.-S.; Peever, T.L.; Cannon, P.G.; Uchida, J.Y.; Acelino Couto Alfenas, A.C. 2011. Tracking down worldwide *Puccinia psidii* dispersal. p.14 in Proceedings of the IUFRO working group 2.04.06 Tree Biotechnology Conference 2011: From Genomes to Integration and Delivery. 26 June – 2 July 2011, Arrail d'Ajuda, Bahia, Brazil. BMC Proceedings 2011 5 (Suppl 7): P14 (http://www.biomedcentral.com/content/pdf/1753-6561-5-S7-P14.pdf)

Klopfenstein, N.B.; Hanna, J.W.; Graça, R.N.; Ross-Davis, A.L.; Cannon, P.G.; Alfenas, A.C.; Kim, M.-S. 2011. Approaches to predicting current and future distributions of *Puccinia psidii* in South America under climate-change scenarios. pp. 450-454 in: Proceedings of the IUFRO working group 2.08.03 Improvement and Culture of Eucalypts. 14-18 November 2011, Porto Seguro, Bahia, Brazil. Escola Superior de Agricultura "Luis de Queiroz"

Klopfenstein, N.B.; Geils, BW. 2011. II. Pathogens. pp. 13-26 in: Pearson, D.E.; Kim, M.-S.; Butler, J., eds. Rocky Mountain Research Station Invasive Species Visionary White Paper. Gen. Tech. Rep. RMRS-GTR-265. Fort Collins, CO: USDA, Forest Service, Rocky Mountain Research Station. 38 p.

Kim, M.-S.; Richardson, B.A.; McDonald, G.I.; Klopfenstein, N.B. 2011. Genetic diversity and structure of western white pine (*Pinus monticola*) in North America: a baseline study for conservation, restoration and addressing impacts of climate change. Tree Genetics & Genomes 7: 11-21.

Bill Jacobi (Colorado State University; Fort Collins, CO) presented results on a pruning project in which they found sanitation pruning resulted in limber pine having significantly less crown dieback than unpruned control trees at two high value sites in a National Park and a campground. Pruning up 7-8 feet did not improve crown health but should reduce lower stem infections. They also found that infections were happening every year at the two sites, so pruning is something that would have to occur every 4-5 years.

Crump, A.; Jacobi, W.R.; Burns, K.S.; Howell, B.E. 2011. Pruning to manage white pine blister rust in the southern Rocky Mountains. Res. Note. RMRS-RN-44. Fort Collins, CO: USDA, Forest Service, Rocky Mountain Research Station. 10 p.

The limber pine seedling planting trials along the Front Range of CO/WY had over 70% survival after three years. Results indicate that survival was better when seedlings were planted next to an object and when planted under tree canopy. Planting with hydrogels did not affect survival. Most death occurred in the first year. They plan to revisit the sites after 5 years.

Mary Lou Fairweather (Forest Health Protection, Flagstaff, AZ) continues to monitor the budding white pine blister rust infestation in Arizona, which

was first identified in 2009. Forest Health Protection in Flagstaff is working with Dr. Kristen Waring and graduate student, Chris Looney, of Northern Arizona University, to establish permanent monitoring plots in forests with southwestern white pine, limber pine, or rocky mountain bristlecone pine in Arizona and New Mexico. Some plots established in 2010 and 2011 were burned by large wildfires in 2011. These plots will be revisited in 2012 to record effects of fire on white pine and blister rust.

Rust Committee Chair Holly Kearns announced that this would be her last meeting as committee chair. She has held the position since the 2005 WIFDWC in Jackson, WY. Helen Maffei volunteered to serve as Rust Committee Chair and the members in attendance approved her appointment.

CLIMATE CHANGE COMMITTEE REPORT

Committee Co-chairs - Susan Frankel, David Shaw, and Charles G. (Terry) Shaw

The WIFDWC Climate Change Committee met on Tuesday, October 11 from 5:15 pm to 6:30 pm with over 50 in attendance. The program featured "Trees, forests, and forest diseases: How do we prepare for change?" by Rona Sturrock and Elisa Becker, Canadian Forest Service, Pacific Forestry Centre, Victoria, BC. The paper provided a lively overview and supplement to a recently published review paper: Sturrock, R. N.; Frankel, S. J. ; Brown, A. V. ; Hennon, P. E. ; Kliejunas, J. T.; Lewis, K. J.; Worrall, J. J. and Woods, A. J. 2011. Climate change and forest diseases. Plant Pathology. 60: 133–149.

Sturrock and Becker recommended several outstanding recent references on science and society including:

Stenlid, J.; Oliva, J., Boberg, J.B. & Hopkins, A.J.M. (2011). Emerging diseases in European forest ecosystems and responses in society. Forests 2(2), 486-504.

Mery, G.; Katila, P.; Galloway, G.; Alfaro, R.I.; Kanninen, M.; Lobovikov, M. and Varjo, J. (eds.). 2010. Forests and Society – Responding to Global Drivers of Change. IUFRO World Series Volume 25. Vienna. 509 p.

"Forests and how to save them: The world's lungs". The Economist. September 23, 2010. http://www.economist.com/node/17093495.

Also mentioned was a European Commission project (ISEFOR FP7) **"**Increasing Sustainability of European Forests: Modelling for Security Against Invasive Pests and Pathogens under Climate Change". The project is coordinated by Prof. Stephen Woodward (University of Aberdeen) and comprises seventeen partners in the EU, Russia, China and the US.

There is considerable activity among members concerning climate change and diseases. It appears to be a major issue for the folks at northern latitudes (Canada and Alaska) and less of a pressing issue for those in the southern latitudes. We are considering a panel for the 2012 WIFDWC Lake Tahoe meeting concerning strategies for dealing with climate change, in particular, assisted migration of forest trees, and the pros and cons of this strategy.

WIFDWC Climate Committee Co-chairs: Susan Frankel, USDA Forest Service, Pacific Southwest Research Station; Albany, CA. David Shaw, College of Forestry, Oregon State University; Corvallis. Charles G. (Terry) Shaw, Emeritus, USDA Forest Service, Western Wildland Environmental Threat Assessment Center, Prineville, OR.

NURSERY DISEASE COMMITTEE MEETING REPORT

Interim Committee Chair - Will Littke

THEME: Advances made in nursery pathology through PCR based methodology

Fourteen members attended the Nursery Disease Committee meeting Our first order of business was to thank Katrina Mallams (USFS J Herbert Stone Nursery, OR.) who has served as the moderator for the past few years. Will Littke (Weyerhaeuser Company, Federal Way, WA.) will act as the interim Nursery Disease Committee chairperson.

Anna Leon (Washington State University) began with an overview of her PCR work to quantify soil *Fusarium commune* populations. This study seeks to understand the utility of performing real time PCR on soil samples to determine pathogen risk from this pathogen in Douglas-fir nurseries. Traditional soil assays on selective media such as Komada's reflect total soil *Fusarium* populations. Nathan Johnson (University of Washington) presented some observations on *Fusarium commune* and *Cylindrocarpon spp*. interactions with various biocontrol organisms (*Bacillus, Trichoderma, Gliocladium, Streptomyces*). These agents are being used as part of a larger treatment interaction with various alternatives to methyl bromide study. Mahsa Khoransani (University of Washington) discussed new developments in genetic markers to delineate species of *Cylindrocarpon* on Douglas-fir seedlings. Ned Klopfenstein, John Hanna, and Amy Ross-Davis (USFS RMRS, Moscow, ID.) did some of the initial PCR and isolate identification work and showed that *C. destructans* and *C. liriodendri*, are the predominant isolates found on PNW Douglas-fir seedlings; *C. pauciseptatum* was also found.

Gary Chastagner presented an overview of IR-4 fungicide chemistry testing for control of *Pythium* root disease. Jerry Weiland (ARS Corvallis, OR.) recently found at least ten species of *Pythium* on Douglas-fir from three PNW nurseries. *Pythium irregulare* was found in all three nurseries, but species were found in only one nursery. The IR-4 data and PCR data suggests a minimum of three chemical families might be needed to control "*Pythium*" root disease, unless specific species data is present based on the tolerance data published.

Alternatives to methyl bromide efforts were discussed as part of a joint study by Weyerhaeuser (Will Littke, John Browning), University of Washington (N. Johnson, B. Edmonds, M. Khoransani) and USDA ARS (J. Weiland). Efforts are being placed on 25-foot buffer fumigant rates of various chloropicrin-telone and chloropicrin-metam sodium products in combination with totally impermeable tarps. Results so far appear favorable. Data from mid-summer showed that high populations of fluorescent pseudomonad bacteria in non-fumigated soils were detrimental to applied biocontrol agents.

A discussion on the need for additional collaborative work on PCR development in disease control efforts was contributed to by Susan Frankel (USDA, Albany, CA.), Paul Zambino (USFS, San Bernadino, CA.) Simon Shamoun (CFS-PFC, Victoria, B.C.), and Karen Hutten (University of Washington). PCR results help to explain the great variation seen in fungicide response and will most definitely aid in developing new control regimes.

HAZARD TREE COMMITTEE MEETING REPORT

Committee Chair - Pete Angwin

Approximately 40 people attended the WIFDWC Hazard Tree Committee Breakfast. The meeting was chaired by Pete Angwin and Mary Lou Fairweather took notes. Three items were on the formal agenda:

1) Pete Angwin opened the breakfast meeting with a powerpoint presentation of photos from the 2010 Western Hazard Tree Workshop. Pete opened the formal part of the meeting with a discussion of the 2013 Hazard Tree Workshop. Suggestions are being solicited for the Workshop location. A planning committee meeting will be held in the spring of 2012 to finalize the location and discuss field trip and presentation/panel topics. Because of the current travel issues, the meeting will be held by conference and/or video conference. **Suggestions for the Workshop location and participation in the planning committee are encouraged- call or e-mail Pete if you have suggestions or would like to be part of the committee (phone 530-226-2436 or e-mail pangwin@fs.fed.us).**

2) Judy Adams gave an update on the status of the International Tree Failure Database (ITFD). Following the request last year by Frank Sapio of FHTET to find an outside party to house and maintain the database, the International Society of Arboriculture (ISA) was asked to perform a needs/use review of the ITFD. However, because of high turnover in the grants division of the ISA due to the recession, the ISA could not perform the review. Larry Costello was requested to perform the review and work with ISA. He is working with Sharon Lilly, chair of the ISA Arborist News Editorial Board. They will generate a survey with the ITFD steering committee, which will be used to assess the views of ITFD users and cooperators. Hopefully, the review will be completed by the next WIFDWC Hazard Tree Committee meeting. WIFDWC members are encouraged to contact Larry at Larryoracleoak@me.com with their feelings and opinions regarding the ITFD, and to take part in the survey. The database is still being maintained by FHTET and the database and reports are still available for download from FHTET at http://svinetfc2.fs.fed.us/natfdb/index.aspx. User requests are still up, but tree failure report inputs are still minimal.

3) Forrest Oliveria next led a discussion on developing a National Hazard Tree Strategy/Program for the US Forest Service ("Fools rush in where angels fear to tread!"). Two integral parts of the Strategy/Program would be the development of flexible, but reasonably consistent general protocols for the assessment of hazard trees (Regions would continue to use their existing hazard rating systems), and the development of a national database for hazard tree data (possibly using FACTS or FAD). Forest Health Protection would provide assistance, ground support and training to local National Forest land managers and other cooperators. A three page handout on the subject was distributed. It suggested that the database currently in use for southern pine beetle could be used as a model, in which specific core data items are collected and the resulting data and reports are made available to all cooperators. A lively discussion ensued:

Mary Lou Fairweather asked what purpose and intent of the hazard tree database would be. She expressed concern regarding the potential to supply proprietary or incriminating evidence to lawyers. However, it would be helpful for Districts to have a database to keep track of when hazard tree evaluations and mitigations are done.

Dave Shaw asked if the intent is it to make hazard tree forms and evaluations uniform. Forrest said that this was not the case, that different cooperators would collect different things. However, there would be specific core data elements that would be collected.

Judy Adams stated that this would be a good way to record basic information on where and when trainings are given, both as a way to let people know when and where upcoming training sessions are being offered and to document completed sessions.

Her boss is uncomfortable with loading actual hazard tree information.

Pete Angwin suggested that this would be an excellent way to keep consistent records of what areas were assessed, where, when and by whom.

Greg Filip added that Region 6 is moving toward developing standard assessment methods for hazard trees in developed recreation areas, as well as a set of certification protocols for the assessors. This is similar to the methods and certification protocols currently in effect for roadside danger tree assessment, in which assessors are recertified every three years.

Bill Jones added that the ISA is likewise in the process of developing certification requirements and best management practices. He stated that the Forest Service will be held to the highest possible standard, which is that of the ISA. Because of that, he suggested that training and certification be done in cooperation with the ISA. Bill also stated that for legal reasons, we could not list injuries, fatalities or property damage in a corporate database. (Note: On Dec 15, 2011, Bill reported that the ISA BMP's are finished and are available from their website at http://secure.isa-arbor.com/webstore/BMPs-C59.aspx).

Borys Tkacz reminded everyone that the WO FHP staff is currently revising the Forest Service Handbook sections on Forest Health Protection. If there is a need to include information on hazard tree program (or anything else), that should be forwarded to the WO for consideration.

Forrest stated that in Region 8 they want to equip employees with knowledge and a GPS so they can start dealing with some of their hazards. They would also like to coordinate with other Regions and have a central point for hazard tree data.

As the time for discussion drew to a close, Pete asked for input on how the WIFDWC Hazard Tree Committee should proceed. Forrest said that Bruce Moltzan (who could not be present at the Committee meeting) had suggested that the Regional Hazard Tree coordinators come up with a concept paper that can be used to start a national dialog on hazard tree programs. Forrest agreed to lead this effort. Current Regional coordinators are Marcus Jackson (R1), Jim Worrall (R2), Mary Lou Fairweather (R3), John Guyon (R4), Pete Angwin (R5), Greg Filip (R6), Bill Jones/Roger Menard (R8), Lori Winton (R10), Joe O'Brien/Jill Pokorny (NE) and Judy Adams (FHTET).

Forrest stated that this would be part of a national hazard tree website, similar to the southern pine beetle website. However, the main task at this point is to determine what information would be useful to include in a national hazard tree database.

After the discussions on the three formal agenda items were complete, the remaining time was dedicated to a round robin discussion. 2 items were discussed:

1) Bill Woodruff reported on the removal of a tree with annosus root disease in a campground, and how severe the defect turned out to be.

2) Bill Jones reported on how in Region 8 and the Northeast Area, Class C faller recertification training will take place in April 2012 on the George Washington-Jefferson National Forest, and that this year's focus will be on hazard tree management on Forest Service lands and the new roadside hazard tree policy.

ROOT DISEASE COMMITTEE MEETING REPORT

Committee Chair – Michelle Cleary

This year we had a short, yet lively, committee luncheon meeting that started with some announcements regarding the 13th IUFRO Root and Butt Rot Meeting recently held in Firenze and San Martino di Castrozza, Italy, September 4-8, 2011. The next IUFRO Root and Butt Rot meeting will be hosted by Turkey in 2015. Another upcoming meeting of the 6th IUFRO Working Party on Phytophthora in Forests and Natural Ecosystems will be held to be held in Córdoba, Spain, September 9-14, 2012.

Presentation by Rich Hunt and Duncan Morrison on the use of the name *A. solidipes* versus *A. ostoyae*

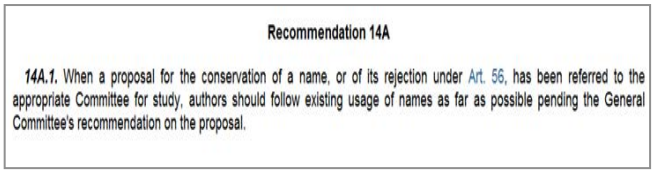
At the 58th WIFDWC meeting in Valemount, many members voiced concerns and voted unanimously on tasking Rich Hunt and Duncan Morrison to investigate the *A. solidipes* vs. *A. ostoyae* issue. During the Valemount meeting, Rich Hunt suggested that in striving for nomenclatural stability the International botanical code would follow what was known as the Tokyo code. The Tokyo code favoured using names in current usage rather than digging-up old names. If this was so, it should be easy to conserve *A. ostoyae*. However, after the meeting Rich examined the code and could not find any such rules within the code. Consequently, he sent an email to several individuals indicating that he thought it would be difficult to conserve the name *A. ostoyae*. One way to eliminate *A. solidipes* is to show that its type is dubious as to what it stands for. This has already been done with several other old *Armillaria* spp. names. One cannot conserve a name by itself. A name must be conserved over some other name. So an argument must be made to conserve *A. ostoyae* over the name *A. solidipes*. This could be successful if *A. solidipes* is dubious. The description of the *A. solidipes* type in relation to other *Armillaria* spp. is summarized in Hunt et al. (2011) - letter to the editor in the August 2011 edition of *Forest Pathology* entitled "*Armillaria solidipes* is not a replacement name for *A. ostoyae*". Copies of this publication and other pertinent email correspondence including that to Burdsall were made available to members before and during the root disease committee meeting.

To conserve *A. ostoyae* over *A. solidipes* one has to make it very clear about the deposition of the two type specimens. Through the investigations, it was learned that Jean Berube had requested Ottawa to conserve *A. ostoyae*. Jean was then asked for help because he would be a logical person to sort out the French language used to describe the deposition of the *A. ostoyae* type. The information about the location *A. solidipes* as supplied by Burdsall and Volk includes the original label of E. Bartholomew herbarium specimen 2690, and information indicating deposition in herbarium FH. It would seem that 'FH' would mean the Farlow Herbarium, because Peck worked in New York. However, over the internet, one can find documentation that Bartholomew specimens are deposited in herbarium 'FH' with 'FH' being short for 'Fort Hays'. It was Rich's understanding that international codes for herbaria were to be four or more letters long, and Fort Hays can be FHKSC. Since it was very confusing as to how to cite the *A. solidipes* type, Rich and Duncan decided to seek help from one of Canada's taxonomic big guns, Scott Redhead (Curator, National Mycological Herbarium, Ottawa, ON.). To have a Proposal considered for conservation it must first be published in *Taxon*. Unfortunately, Scott was too busy with other issues to help put together a proposal published before the vote on conservation proposals at the Botanical Congress in July this year.

In the meantime, Rich discovered he was wrong about the Tokyo code. It is still valid, as found in the publication "Guidelines for proposals to conserve or reject names". There are three quotes from this *Taxon* paper. First "avoid displacing well-established names for purely nomenclatural reasons"; second "Successive Nomenclature Sections have made it quite clear that indulging in name changes for purely nomenclatural reasons is now reprehensible…"; and third "the fact that a name is widely used by non-taxonomists is a cogent reason for its preservation,…". Based on this, it seems rather

obvious that Burdsall & Volk, and the editors of North American Fungi have been reprehensible in trying to replace *A. ostoyae* with *A. solidipes*.

As discussed and supported by these findings, a proposal to conserve the name *A. ostoyae* was being prepared. Two weeks after the WIFDWC meeting, this proposal to conserve *A. ostoyae* was submitted to the journal Taxon. As of November 16, 2011, the proposal was electronically published. It will now be considered in the hands of the appropriate International Committee that will consider the merits of the proposal. This committee will present a recommendation concerning the proposal to the general assembly. The general assembly will formally vote on the proposal at the next International Code of Botanical Nomenclature meeting. Consequently, we should follow Recommendation 14A1 of the Vienna Code and 'follow existing usage' i.e., use only the name *Armillaria ostoyae*". If anyone is challenged for using that name, they can cite the electronic publication and the code article as it is quoted below. One can also cite Hunt *et al.* (2011).



Recommendation 14A

14A.1. When a proposal for the conservation of a name, or of its rejection under Art. 56, has been referred to the appropriate Committee for study, authors should follow existing usage of names as far as possible pending the General Committee's recommendation on the proposal.

On behalf of WIFDWC members, I'd like to thank Rich and Duncan for all their efforts and for spending many of their 'retired' hours to help resolve this issue. It is much appreciated!

Since we did not have time for a round-robin, a request was sent out after the meeting for members to report on any new/current root disease work and publications. The following submissions were provided:

National Root Disease Paper
Blakey Lockman

Root diseases are the most important disease agents in Region One and impact management on millions of acres. In order for root diseases to get the national attention they deserve, we need to make the case that root diseases impact more than just Region 1. So, Holly Kearns and I have formed a small team of Regional pathologists to take on the task of creating a white paper on Root Diseases, similar to what Dick Smith did in 1984 ("Root Disease-Caused Losses in the Commercial Coniferous Forests of the Western United States).

Purpose of the white paper:

- Provide a short, concise document for Rob Mangold, USFS Forest Health Protection Washington Office Director, to share with other Directors in order to heighten awareness of the importance of root diseases at a National level.
- Provide a consistent description of root diseases spanning the affected regions.

Objectives of the white paper:

- Describe the current root disease conditions in each of the affected Regions
- Describe the issues each Region faces relative to managing acreages infested with root diseases
- Describe possible solutions to deal with the above issues

We hope to complete this paper over the winter and have a final out for distribution by spring 2012.

Alpine Larch decay sample found to be a first generation *Heterobasidion* hybrid

A sample collected from alpine larch (*Larix lyallii*) from the Bitterroot Mountains south of Missoula, Montana in September 2010 has proven to be a first generation *Heterobasidion* hybrid. The site is at approximately 8300 ft. elevation, along the shores of Gem Lake, a high alpine lake in the Bitterroot Mountains south of Darby, Montana. Stand is composed of alpine larch, whitebark pine and a few subalpine fir. The down tree was located near several other alpine larch with thin and narrow crowns. There were also several dead standing alpine larch, indicating a possible root disease pocket. An older alpine larch root ball was found nearby with very old annosus-like fruiting bodies in the root system. A sample was collected from heavily decayed roots from a downed tree that was still alive, so it likely fell over or was blown over within the previous year. Roots were nearly fully decayed; decay was white pockets with black flecks,

fairly diagnostic of annosus decay. Isolations were placed on 2% malt agar plates and grown for 7 days. The resulting isolate was then sent to M. Garbelotto and S. Mascheretti , Department of ESPM, University of California, Berkeley, for identification. Garbelotto and Mascheretti used DNA analysis for 3 specific loci to positively identify the isolate as a first generation hybrid between *H. irregulare* and *H. occidentale*. This new find was presented at the IUFRO 13th International Conference Root and Butt Rots of Forest Trees in Italy in September, 2011 by M. Garbelotto (Lockman B., Mascheretti S., and Garbelotto M., in publication).

Greg Filip

We finished taking 30-year data on our Armillaria precommercial-thinning plots in Oregon and Washington. The plots were established in coastal Douglas-fir, hemlock, and Abies. We are analyzing the data this fall and winter and hope to publish a paper on the results.

Moscow lab

The plant pathology staff (Ned Klopfenstein John Hanna, Amy Ross-Davis, and Sara Ashiglar) and collaborators (Geral I. McDonald – emeritus, Mee-Sook Kim – Kookmin University, Seoul, Korea, and Jane Stewart – postdoc. U of Idaho) in RMRS Moscow, ID are continuing a collaborative STDP project with Region 6 (Helen Maffei) and Region 3 (Mary Lou Fairweather) to develop predictive models for *Armillaria* spp. in the inland western U.S. Surveys in Oregon are completed and surveys in Arizona are nearly completed. These predictive models will attempt to predict potential *Armillaria* spp. distributions under various climate-change scenarios. Rubén Damian Elias Roman (graduate student from Colegio de Postgraduados, Montecillo, Mexico) performed DNA-based diagnostics in our laboratory for his Ph.D. research to identify *Armillaria* species that cause damage in peach orchards. Other studies are ongoing to examine the transcriptome (expressed genome determined from mRNA) from a mycelial fan of *Armillaria solidipes* (=*A. ostoyae*) on grand fir. A goal of this project is to identify genes associated with pathogenicity and adaptation. International collaborations are continuing on a long-term project to examine the evolutionary relationships among *A. solidipes* and related species from world-wide sources.

New Publications:

Cleary, M., van der Kamp, B.J., and D.J. Morrison. Effects of wounding and fungal infection with *Armillaria ostoyae* in three conifer species. II. Host response to the pathogen. Forest Pathology [*In press*]

Cleary, M., van der Kamp, B.J., and D.J. Morrison. Effects of wounding and fungal infection with *Armillaria ostoyae* in three conifer species. I. Host response to abiotic wounding in non-infected roots. Forest Pathology [*In press*]

Rönnberg, J. and M. Cleary. Presence of *Heterobasidion* infections in Norway spruce stumps after treatment six years earlier with *Phlebiopsis gigantea*. Forest Pathology [*In press*]

Cleary, M. and T. Holmes. 2011. Traumatic resin duct formation in the phloem of western red cedar (*Thuja plicata*) following abiotic wounding and infection by *Armillaria ostoyae*. International Association of Wood Anatomists Journal. 32: 351-359

Cleary, M., Sturrock, R. and J. Hodge. 2011. British Columbia's southern interior forests: Phellinus root disease stand establishment decision aid. British Columbia Journal of Ecosystems and Management. 12:17–20.

Cruickshank, M.G., Morrison, D.J. and Lalumiere, A. 2011. Site, plot, and individual tree yield reduction of interior Douglas-fir associated with non-lethal infection by Armillaria root disease in southern British Columbia. For. Ecol. Manag. 261: 297-307.

Hunt, R.S, Morrison, D.J. and Bérubé, J. and. 2011. *Armillaria solidipes* is not a replacement name for *A. ostoyae*. Forest Pathology. 41: 253-254.

Kim, M-S.; Hanna, J.W.; Klopfenstein. 2010. First report of an Armillaria root disease pathogen, *Armillaria gallica*, associated with several new hosts in Hawaii. Plant Disease 94: 1510.

Kim, M.-S.; Klopfenstein, N.B. 2011. Molecular identification of *Armillaria gallica* from the Niobrara Valley Preserve in Nebraska. Journal of Phytopathology 159: 69-71.

Morrison, D.J. 2011. Epidemiology of Armillaria root disease in Douglas-fir plantations in the cedar-hemlock zone of the southern interior of British Columbia. For. Pathol. 41: 31-40.

Ota, Y.; Kim, M.-S.; Neda, H.; Klopfenstein, N.B.; Hasegawa, E. 2010. The phylogenetic position of an *Armillaria* species from Amami-Oshima, a subtropical island of Japan, based on elongation factor and ITS sequences. Mycoscience 52: 53-58.

Ross-Davis, A.L.; Hanna, J.W.; Kim, M.-S.; Klopfenstein, N.B. Advances toward DNA-based identification and phylogeny of North American *Armillaria* species using elongation factor-1 alpha gene. Mycoscience [*In press*]

Redhead, S.A, J. Bérubé, M. R. Cleary, O. Holdenrieder, R. S. Hunt, K. Korhonen, H. Marxmüller and D. J. Morrison. 2011. Proposal to conserve *Armillariella ostoyae* (*Armillaria ostoyae*) against *Agaricus obscures*, *Agaricus occultans*, and *Armillaria solidipes* (*Basidiomycota*). Taxon 60: 1770-1771.

DWARF MISTLETOE COMMITTEE REPORT

Committee Chair - F.A. Baker

Bob Mathiesen updated progress on a letter to the editor of Flora of North America regarding Dan Nickrendt's lumping of *Arceuthobium* based only on molecular data. Last year the dwarf mistletoe committee drafted a letter which was signed by the chair; the editor agreed to allow a brief counterpoint at the end of the chapter indicating that pathologists and foresters use the current taxonomy which is based on morphology and host characteristics. Bob will follow to see where things stand.

Bob also mentioned that he has conned Del Wiens into working on taxonomy of *Phoradendron*, and requested photos.

Fred Baker reported finding useful regressions for predicting DMR from % infection on FIA plots.

Katy Mallams, former chair of this committee published a comparison of broom volume rating vs DMR for dwarf mistletoe infected Douglas-fir. It was also announced that Katy will be retiring.

Paul Hennon mentioned a study using inventory data to model hemlock and dwarf mistletoe distributions using climate envelopes.

Leif Mortensen reported that 25% of red firs in California had mistletoe, while about 10% of Jeffrey pine, lodgepole pine and white fir were infected.

The following was submitted for further information:

Taxonomy, Hosts, and Distribution

A detailed morphometric analyses of 17 populations each of red fir dwarf mistletoe (*A. abietinum* f. sp. *magnificae*) and white fir dwarf mistletoe (*A. abietinum* f. sp. *concoloris*) in the Sierra Nevada Mountains and southern Cascade Range found no major morphological differences between these special forms of fir dwarf mistletoe. This supports the earlier findings of Hawksworth and Wiens. Additional field observations also support the extreme host specialization these dwarf mistletoes exhibit in stands of red and white fir. Red fir dwarf mistletoe did not infect white fir and white fir dwarf mistletoes did not infect red fir. The results of this study will be published in Madroño. R. Mathiasen, Northern Arizona University, Flagstaff, AZ.

We are continuing our systematic study of dwarf mistletoes that primarily parasitize white pines in western North America and Mexico. We are using molecular techniques (AFLP analysis) and have made additional morphological measurements for *A. apachecum, A. blumeri, A. californicum, A. cyanocarpum,* and *A. monticola* over the last three years. We are now analyzing AFLP and morphological data. B. Reif and R. Mathiasen, Northern Arizona University, Flagstaff, AZ.

We completed a study of the dwarf mistletoes affecting Brewer's spruce (*Picea breweriana*) in northwestern California and southwestern Oregon and the results were published in Northwest Science: Susceptibility of Brewer spruce (*Picea breweriana*) to dwarf mistletoes (*Arceuthobium* spp., Viscaceae). Northwest Science 84: 295-301. R. Mathiasen and C. Daugherty, Northern Arizona University, Flagstaff, AZ.

The Klamath-Siskiyou Mountains Region has the greatest diversity of dwarf mistletoes in the United States. We recently published the results of our findings on the host ranges of three dwarf mistletoes found in the Klamath-Siskiyou Mountain: Susceptibility of conifers to three dwarf mistletoes in the Klamath-Siskiyou Mountains. Western Journal of Applied Forestry 26: 13-18. R. Mathiasen and C. Daugherty, Northern Arizona University, Flagstaff, AZ.

We estimated the current incidence of infection and extent of mortality in a stand of bristlecone pine infested with bristlecone pine dwarf mistletoe (*A. microcarpum* subsp. *aristate*) on Schultz Peak, a southeast subsidiary ridge of the San Francisco Peaks, Arizona. Dendroecological techniques were used to compare radial growth between lightly-,

moderately-, severely-, and non-infected bristlecone pines. We also paired limber pine (*Pinus flexilis*) with non-infected and infected bristlecone pines as another method of estimating the effects of the mistletoe on radial growth. Results suggested that the incidence of infection has increased by approximately 10% and mortality has increased by approximately 20% since the mid 1970s. Severely-infected bristlecone pines had significant growth losses and less annual variation in growth than limber pine or lightly- and non-infected bristlecone pines. Cross-dating cores from dead bristlecone pines indicated that mortality had rapidly increased starting in 1996. Years of high mortality were related with both high mountain pine beetle (*Dendroctonus ponderosae*) activity and drought conditions. A publication reporting our work is in press with Forest Science. J. Scott, and R. Mathiasen, Northern Arizona University, Flagstaff, AZ.

I have begun an extensive morphometric study of *Arceuthobium campylopodum*, *A. occidentale*, *A. siskiyouense*, and *A. littorum* in California, Oregon, and Washington. Some populations of *A. campylopodum* from Idaho may be included also. This study will take several years to complete because of the extensive distribution of *A. campylopodum* from northern Baja, Mexico to northern Washington. R. Mathiasen, Northern Arizona University, Flagstaff, AZ.

We have started the initial work on producing a publication on how to identify mistletoes in the United States, both species of *Arceuthobium* and *Phoradendron*. We plan to use many color photographs to illustrate important mistletoe characteristics, but the publication will also include keys, non-technical descriptions (and maybe technical ones?), geographic and host range information, and maybe a comparison of similar species. We welcome input from WIFDWC members on how to format the publication and we will be soliciting color, digital photographs of mistletoes from members at a later date. So please start taking good pictures of mistletoes in your area. The publication will follow the Hawksworth and Wiens classification system for *Arceuthobium* (with a few minor additions and changes) and the Wiens classification for *Phoradendron*. While the publication will not include detailed information on the management of mistletoes, it will include a listing of relevant publications on management and ecology. We anticipate this work may take several years. R. Mathiasen, Northern Arizona University, Flagstaff, AZ.; D. Wiens, University of Utah, Salt Lake City, UT. (retired).

Ecology

We have been studying the biomass relationships for un-infected and dwarf mistletoe-infected branches of Douglas-fir in northern Arizona. We are also examining branch architecture of Douglas-fir dwarf mistletoe-induced witches' brooms. The broom biomass study is about to be published in the Arizona/Nevada Academy of Science. Data on broom architecture is still being analyzed for Douglas-fir dwarf mistletoe. L. Smith, R. Mathiasen, and R. Hofstetter, Northern Arizona University, Flagstaff, AZ; C. Hoffman, Colorado State University, Ft. Collins, CO.

2011 STUDENT AWARDS COMMITTEE REPORT

Committee - Bill Jacobi, Holly Kearns, Blakey Lockman, John Schwandt

With the proceeds from last year's Silent Auction in Valemount, BC we provided $1,000 for three Student Travel Awards. The 2011 Student Travel Award recipients were Christy Cleaver ($300, Colorado State University), Karen Hutton ($350, University of Washington), and Nathan Johnson ($350, University of Washington).

Our second annual Silent Auction was an even bigger success. We auctioned off 76 items and received over $130 in donations for Walt Thies' book collection, in addition to several generous cash donations raising a total of $1,675.00 for future travel awards. We want to sincerely thank the many people who brought historic documents and books, homemade food items, amazing wood sculptures, and handmade cloth items, jewelry, etc. to be auctioned off and also thank everyone that participated so generously in the auction.

At the 2011 Business Meeting, the Student Awards Committee was formally established as a standing committee consisting of four rotating members. The current committee developed detailed criteria for applying for the travel award and how selections would be made. Please see the minutes of the Business Meeting for more information.

2011 WIFDWC BUSINESS MEETING MINUTES

Compiled by Stefan Zeglen, WIFDWC Secretary

Conference Chairperson Pete Angwin called the business meeting of the 59th Annual Western International Forest Disease Work Conference to order at 8:35 am on Thursday, October 13, 2011. Fifty-three people were in attendance.

OLD BUSINESS

A motion to accept the minutes from the 2010 WIFDWC business meeting (Valemount, BC) included in the published proceedings was made, seconded and passed.

The 2011 Organizing Committee is recognized for arranging and running an excellent meeting: Angel Saavedra, Greg Filip, Dan Omhal and Amy Ramsey-Kroll (local arrangements), Alan Kanaskie (program chair), Judy Adams (web co-ordinator), Holly Kearns (treasurer), John Schwandt (treasurer emeritus), Bob Edmonds (grad presentations and poster co-ordinator), and Stefan Zeglen (secretary).

TREASURER'S REPORT (HOLLY KEARNS)

Attendance at this year's meeting was very good with 90 attendees including 58 regular members, 17 students, 7 retirees and 8 guests. [treasurer's financial report table following]

Jim Cook, representing the Washington State Academy of Sciences, addressed the group briefly to recap his presentation and discussion at an open session held the night before. A copy of his letter can be found elsewhere in these proceedings.

COMMITTEE REPORTS

All committee chairs, or designates, gave a brief verbal update on their meetings. Items of note include Holly Kearns stepping down as the Rust Committee chair and being replaced by Helen Maffei. Also, Katy Mallams retires as the Nursery Pathology Committee chair and is replaced by Will Littke. Both are thanked for their service.

HONORARY LIFE MEMBERS

HLM status is bestowed upon recent retirees Terry Shaw, Don Goheen and Sue Hagle.

NOMINATING COMMITTEE

This year's Railroad Committee, consisting of Greg Filip, Alan Kanaskie and Ellen Goheen, nominated the following candidates for the next meetings Executive Committee:

1. Conference Chair: Alex Woods.
2. Program Chair: Paul Hennon.
3. Secretary: John Browning.

A motion to adopt the nomination committee's selection was made (Theis), and seconded (Jacobi) and passed unanimously.

OAA COMMITTEE

Pete Angwin retires as chair of the committee this year and Harry Kope assumes the chair. Bill Jacobi is on deck. Paul Hennon is nominated to fill the vacant position. A motion is made (Jacobi), and seconded (McWilliams) to accept Paul's nomination. The motion passes.

FUTURE MEETINGS

The 2012 WIFDWC will be held in Lake Tahoe, CA during the week of October 7-12. Phil Cannon is spearheading the local arrangements team and has booked the Granlibakken Resort and Conference Centre just south of Tahoe City as the venue. Phil was not present to sing the praises of Lake Tahoe but he did send brochures and a link to a website (www.granlibakken.com).

As directed by a motion from the 2010 WIFDWC, Conference Chair Pete Angwin contacted the other five North American forest disease-related working groups to see if they were interested in holding a joint workshop with WIFDWC in 2013 or 2014. These groups were asked to poll their members and report back their interest in holding a joint meeting at a mutually agreeable location. Pete reported their responses as follows:

1. North Central Forest Pest Workshop – maybe, either year was possible;
2. Northeast Forest Health Workshop – lukewarm, perhaps 2014;
3. Northeast Forest Pest Council – no;

4. Southeast Forest Disease Workshop – favourable;
5. Great Plains Forest Health Council – possible.

The common theme seemed to be that money was tight and it would be better to hold the meeting in their backyard rather than ours. No one made a concrete offer for a joint meeting date or location. After some discussion of these responses, a motion to table the idea of a joint meeting was made (Fairweather), and seconded (Shaw) but failed to pass. A second motion to table the idea until the 2013 business meeting was made (Shaw), and seconded (Fairweather). The motion passed.

Nominations for locations for the 2013 WIFDWC were opened and two locations proposed – Alberta and Utah. Some discussion around the merits of both ensued until the pertinent point was raised that the IUFRO World Congress would be held in Salt Lake City, UT from October 5-11, 2014. The Utah proposal was withdrawn. A motion to select Alberta for 2013 was made (Ramsfield), and seconded (Shamoun). The motion passed. Tod Ramsfield volunteered to handle local arrangements for the meeting.

Nominations for locations for the 2014 WIFDWC were opened and Utah was the lone proposal. A motion to select Utah for the 2014 meeting was made (Baker), and seconded (Goheen). The motion passed.

BYLAW AMENDMENTS (STEFAN ZEGLEN)

The last consolidation of the WIFDWC bylaws was printed in the 2003 proceedings. Numerous amendments have been made during the seven business meetings since without the revisions being reflected in print. Also, there are numerous inconsistencies, errors and omissions in the bylaws that need to be addressed in order to provide clarity and better guidance to future committee members.

In order to effect these changes, the Secretary proposed a series of motions to amend the bylaws. These motions were prepared in advance and included in the registration package for members to inspect prior to the business meeting. The motions and member vote results are listed below. All accepted changes are listed in the new consolidated bylaws published elsewhere in these proceedings.

1. Motion #1 was made (Zeglen), and seconded (Shaw) to make housekeeping amendments to the bylaws to bring them up to date. The motion passed.
2. Motion #2 was made (Zeglen), and seconded (Goheen) to amend deficiencies in the bylaws including defining duties of officers, clarifying electronic balloting procedures and committee structure, and incorporating the Outstanding Achievement Award criteria. The motion passed.
3. Motion #3b was made (Frankel), and seconded (Kearns) to convert the ad-hoc Student Scholarship Committee into a standing committee. The motion passed.
4. Motion #3c was made (Jacobi), and seconded (Schwandt) to establish Student Travel Awards. The motion passed.

In addition, motions to amend the bylaws were submitted in advance and presented at the business meeting for a vote by the members.

1. Motion #4 was made (Angwin), and seconded (Edmonds) to enable committee chairs to authorize expenditure of funds generated by their committees. The motion passed.
2. Motion #5 was made (Baker) to annually rotate subject matter standing committee meeting dates and times. The motion failed for lack of a second.

Finally, one motion from the floor was entertained.

1. Motion #6 was made (Hunt), and seconded (Morrison) to amend the wording around the qualifications for Honorary Life Membership. The motion passed.

ARMILLARIA NOMENCLATURE

Following up on the direction they were given by members at the 2010 business meeting, Rich Hunt and Duncan Morrison presented the results of their investigation into the recent proposal to change the name of *Armillaria ostoyae* to *Armillaria solidipes*. A published letter to the editor of *Forest Pathology*

was circulated that outlines the case against the change. More detail on this item can be found in the Root Disease Committee report published in these proceedings.

NEW BUSINESS

Ned Klopfenstein reminded members to try and become more involved in collaborations with our Mexican colleagues.

A motion was made (Angwin), and seconded (Goheen) to appoint to the Student Scholarship Committee the former members of the previous ad-hoc committee – Holly Kearns, Blakey Lockman, Bill Jacobi and John Schwandt. The motion passed.

Upon completion of business a motion to adjourn the meeting was made, seconded and passed.

TREASURER'S REPORT

Holly Kearns

We had a large turnout in Leavenworth with 82 total registrants including 58 regular members, 17 students, and 7 retirees. The following is a summary of transactions for the WIFDWC account from 1/1/2011 through 12/16/2011. Please note that our Federal Tax Id # is **35-2307554.**

Transaction	**Income**	**Expenses**	**Balance**
WIFDWC balance as of 12/31/2010:			***$15,771.99***
2011 WIFDWC Meeting –Leavenworth, WA Oct. 10-14			
Total registration	$25,200.67		
Hotel meeting rooms, meals, breaks (excludes $250 deposit paid in 2009)		$11,398.54	
Field trip transportation		1,743.45	
Field trip supplies and lunches		1,578.41	
Pre-meeting field trips		994.58	
Speaker expenses		1,812.04	
Trinkets and awards		1,140.05	
Supplies		55.78	
Estimate for Leavenworth proceedings		(3,500.00)	
Estimated Net Proceeds ($2,977.82)			
Other Account Activity			
2010 Proceedings (printing and mailing)		4,520.45	
2012 Meeting deposit		3,500.00	
Bank interest	45.01		
Ending Balance			**$14,274.37**
Student Award Fund balance as of 12/31/2010			**$1,364.00**
2011 Silent Auction Proceeds	1,675.00		
2011 Student Travel Awards		1,000.00	
Ending Balance			**$2,039.00**
Hazard Tree Committee Balance			**$5,401.96**
Total Balance as of 12/16/2011 (WIFDWC + Hazard Tree + Student Award)			**$21,715.33**

BYLAWS OF THE WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE

Passed by a vote of the Membership at the Business Meeting of October 13, 2011

Article I
Objectives_____

The Western International Forest Disease Work Conference (WIFDWC) was formed in 1953 to provide a forum for information exchange among forest pathologists in western North America. The primary objectives of the organization are:

To exchange information on forest pests and related matters through periodic meetings and other appropriate means,

To promote education, research and extension activities in forest pathology, and

To sustain and improve the health of western North America's forests.

Article 2
Membership_____

Membership is open to individuals who are engaged in forest pathology related endeavors in western North America. These include but are not limited to: research, survey, management, teaching or extension activities pertaining to tree diseases, forest health, or deterioration of forest products.

Western North America is defined as Canada: British Columbia, Yukon, Alberta, Manitoba, Saskatchewan; United States: Washington, Oregon, California, Idaho, Nevada, Utah, Arizona, Montana, Wyoming, Colorado, New Mexico, North Dakota, South Dakota, Nebraska, Kansas, Alaska, Hawaii, Guam, the Commonwealth of the Northern Marianna Islands and other Pacific Islands in Micronesia; and all of Mexico.

Membership is established after attending one Western International Forest Disease Work Conference. Members must attend another Western International Forest Disease Work Conference within 5 years or their membership is no longer valid.

Honorary Life membership will be automatically awarded to those members of WIFDWC (as defined above) who have attend at least 5 previous meetings of WIFDWC and have retired. Newly retired members who meet these criteria should notify the current WIFDWC Secretary of their status. Other members who have retired but do not meet the attendance criteria or other outstanding contributors to the field of Forest Pathology may request, or be proposed for, Honorary Life Membership by members present at an annual business meeting.

A list of Honorary Life Members will be published in the Proceedings of each meeting.

A 50% or more reduction in the registration fees for Honorary Life Members, to include a copy of the Proceedings, should be considered by the Executive Committee, as per Article 7.

Article 3
Officers_____

WIFDWC officers will include a Conference Chairperson, Secretary, Treasurer, Program Chairperson, Historian and Web Coordinator. The Conference Chairperson and Secretary will be elected by majority vote of the membership at the annual business meeting. If there is no majority, an acting Chairperson will be appointed by the current Conference Chairperson. The tenure of the Conference Chairperson and Secretary begins at the conclusion of the WIFDWC meeting where they were elected and ends when all business from the next WIFDWC is completed. The Treasurer, Historian and Webmaster will be elected every five years, to serve for the following 5 years.

Duties of the Conference Chairperson

At each WIFDWC, the Conference Chairperson will run the general and business meetings. The Conference Chairperson will appoint an interim Program Chairperson at the start of each WIFDWC to gather suggestions and opinions to guide the conference in the planning of next year's conference. The Conference Chairperson will also appoint three members to serve as the "railroad committee" to nominate candidates for next year's Conference Chairperson and Secretary (and every fifth year, Treasurer, Historian and Web Coordinator). The Conference Chairperson may appoint members to assist in conducting the affairs of the Conference including, but not limited, to Local Arrangements representative(s) and Program Chairperson. The Conference Chairperson may also appoint ad hoc committees and their chairpersonss as deemed necessary to assist in carrying out the mission of WIFDWC.

In the event that a new Conference Chairperson cannot carry out their duties, the previous Chairperson will carry them out. If another member of the Executive Committee cannot or will not carry out their duties the Conference Chairperson may appoint a replacement.

Duties of the Secretary

The Secretary shall maintain the membership and mailing lists. The Secretary shall send out meeting notices to the membership, take minutes at the business meeting, and compile and distribute the Conference proceedings. The secretary will query all Honorary Life Members to determine if they want to receive a free copy of the proceedings and only those responding in the affirmative will receive a copy.

Duties of the Treasurer

The Treasurer shall receive all payments, be custodian of WIFDWC funds, keep an account of all moneys received and expended, and make commitments and disbursements authorized by the Conference Chairperson. At the annual business meeting the Treasurer shall make a report covering the financial affairs of WIFDWC. All funds, records and vouchers in the Treasurer's control should be subject to inspection by the Executive Committee.

Duties of the Program Chairperson

The Program Chairperson is appointed by the Conference Chairperson. The Program Chairperson is responsible for all aspects of the conference agenda including arranging the format and timing of the meeting, selecting panel chairpersons or moderators, selecting the poster session coordinator, assigning subject matter committee meeting times, and arranging keynote, contributing paper and other speakers.

Duties of the Historian

The Historian will keep a complete set of WIFDWC proceedings and answer any inquires as needed. The Historian will contact the WIFDWC Secretary and provide the address for mailing the archival copy of the proceedings.

Duties of the Web Coordinator

The Web Coordinator will create and manage the WIFDWC website. The Web Coordinator will supervise the hosting, security and access of the website. Content for the website will be provided by the Executive Committee for each meeting. The Web Coordinator will ensure that previous WIFDWC meeting websites and their proceedings are archived and linked to the current website.

Compensation

Officers will not be compensated for their services.

Non-liability of Officers

The officers shall not be personally liable for the debts, liabilities or other obligations of the WIFDWC.

Article 4
Decision Making Process_____

The business meeting will be run under Roberts Rules of Order. Meetings are open to the public and non-members may participate in meetings. Only members may vote.

Decisions will be made by majority, with each member granted one vote. Votes may be called for at the annual business meeting or via electronic ballot (i.e., e-mail ballot, web poll, etc.). A quorum is reached when more than 25 members are present.

Article 5
Finances_____

Expenditures

The Conference Chairperson may authorize expenditures of WIFDWC funds. Standing Committee Chairs may similarly authorize the expenditure of funds that are generated by their standing committees (e.g., Hazard Trees Committee). Checks, orders for payment, etc. may be signed by the Treasurer, or other person designated by the Chairperson. The Executive Committee may determine which and how many outside speakers they want to invite, and travel costs for such speakers can be paid from registration fees.

Contracts

The Conference Chairperson may authorize any officer or agent of WIFDWC to enter into a contract on behalf of WIFDWC. Standing Committee Chairs may similarly authorize any agent of their standing committee to enter into a contract on behalf of their committee. Unless so authorized, no person shall have any authority to bind WIFDWC or any standing committee to any contract.

Gifts

The Conference Chairperson or the Treasurer may accept on behalf of the WIFDWC any contribution, gift, or bequest. Commercial sponsorship of conference special events is not allowed.

Fiscal year

The WIFDWC fiscal year shall begin on the first of January and end on the last day of December.

Article 6
Bylaws_____

Amendments
Changes to bylaws shall be made available to all WIFDWC members for review at least one month prior to the next business meeting. A two-thirds majority is required to pass a motion to amend existing bylaws if the vote is held at a business meeting. An affirmative vote from at least 26 members is required to approve a motion voted on by electronic balloting (i.e., e-mail ballot, web poll, etc.).

Article 7
Meetings_____

Frequency

The WIFDWC endorses holding annual meetings but will, on vote of the membership, change the time of any particular meeting when circumstances dictate that such action be taken.

Date

WIFDWC endorses holding meetings in late summer but will change the interval between any two meetings when circumstances dictate that such an action be taken. Meeting dates will be set by the Executive Committee for each meeting.

Registration

Registration will be reduced by half, if possible, for graduate students and Honorary Life Members. It will be at the discretion of the WIFDWC Executive Committee for each meeting to offer a further reduction in fees to graduate students and Honorary Life Members and to offer further reduced fees to others such as retired professionals and visitors.

Article 8
Committees_____

There shall be two types of committees, namely

a) Standing Committees – as designated in the by-laws, and

b) Ad Hoc Committees – as appointed by the Conference Chairperson to serve for a term specified by the Chairperson.

The chair of each standing committee shall prepare a report of the committee activities for the membership. The report will be submitted by the publication deadline to the Secretary for inclusion in the proceedings.

The following are WIFDWC standing committees:

- Executive Committee
    - Composed of the elected Conference Chairperson, Secretary, Treasurer, Historian and Web Coordinator.
    - The Conference Chairperson may appoint a Program Chair, Local Arrangements representative(s) and other persons as necessary to carry out the business of the next WIFDWC meeting.
    - The Executive Committee may invite non-member speakers to the annual meeting and pay their travel expenses from conference registration fees.

- Awards Committee
    - Composed of three members with the longest serving member designated as chair.
    - Committee will be comprised of a representative from each of the following – a university employee, a public agency employee, and one member at large. At least one member should be from Canada.
    - The chair's term will be completed at the end of the annual business meeting and a new junior member will be appointed by the Conference Chairperson. The most senior serving member will assume the chair for the next year.
    - The chair will provide a report of activities at the annual business meeting.
    - Responsible for accepting and evaluating nominations and determining recipients of the WIFDWC Outstanding Achievement Award as outlined in Article 10.

- Student Scholarship Committee
    - Composed of four members with the longest serving member designated as chair.
    - The chair will provide a report of activities at the annual business meeting.
    - The committee will be comprised of at least one representative from a university.
    - Replacement of committee members will be by election at the annual business meeting. The committee is responsible for fundraising to finance any awards given by the committee.
    - The committee is responsible for determining and advertising the award application criteria, receiving and evaluating applications and determining recipients of the WIFDWC Student Travel Awards as outlined in Article 10.
- Hazard Trees Committee,
- Dwarf Mistletoe Committee,
- Root Disease Committee,
- Rust Committee,
- ~~Disease Control Committee~~ [disbanded 2002],
- Nursery Pathology Committee [approved 2002],
- Foliage and Twig Diseases Committee [established 2007, approved 2009],
- Climate Change Committee [established 2007, approved 2010].

Ad hoc committees are established by the Conference Chairperson to carry out various functional needs (e.g., the annual Nominating Committee). Ad hoc committees carry out specific, normally short term, tasks required by the membership. The terms of reference for ad hoc committees will be determined by the Conference Chairperson in consultation with the membership.

Article 9
Proceedings_____

Papers for each year's proceedings must be submitted to the Secretary by the deadline set for each conference by the Secretary.

Distribution of proceedings is made to all paid registrants and honorary members who have indicated a desire to receive them and will be made available to others at cost.

Article 10
Awards_____

Outstanding Achievement Award

Members may recognize outstanding achievement in the field of forest pathology by bestowing the WIFDWC Outstanding Achievement Award. The award will recognize an individual that has, in the opinion of the membership, contributed significantly to the field of forest pathology in western North America.

The award will be presented during the conference by the chair of the Awards Committee or designate. The recipient will receive a framed certificate or plaque. The recipient will present a keynote address at the following year's WIFDWC. A list of recipients will be published in the proceedings.

Members may nominate other current or active members for the award; they may not nominate themselves. A member may only make one nomination each year. A nomination must include: a short introductory letter, a narrative of the nominees qualifications, educational background, work history, etc., letters of support from other members and organizations, and copies of a few of the nominee's published works. Nominations are due no later than three months prior to the start of next year's conference and must be sent to the Awards Committee chair.

The Awards Committee may decide to not make an award if no suitable candidates are nominated.

Student Travel Awards

Members encourage participation in the annual conference by students engaged in studies in the field of forest pathology by bestowing the WIFDWC Student Travel Awards to enable their attendance. The awards are intended for students currently enrolled in a university graduate level program with a thesis or dissertation topic relevant to the field of forest pathology. The awards are intended to assist with conference-related expenses.
Criteria for application and selection of award recipients will be determined by the committee and made public at least four months prior to the early registration date for the meeting or by the first WIFDWC mailing. Completed applications are due by the deadline set by the committee.
The awards will be presented at least four weeks prior to the early registration date for the conference by the chair of the committee or designate. The recipients will receive an award of up to US$500 depending on funding availability. Recipients will be required to make an oral or poster presentation at the meeting for which they received the award. Oral presentations are preferred.

The committee may decide to not make an award if no suitable candidates apply.

Select Motions and Decisions_____

1998

Outstanding Achievement Award—established.

1999

Honorary Life Members—members added and provisions discussed (see 1996 Proceedings for historic retrospective on HLM).

Assisting Outside Speakers—amendment passed.

Website—Committee Reports and Meeting synopsis by the Chairperson would be posted; web committee (Baker, Muir, and Adams) formed.

2000

Outstanding Achievement Award—staggered committee established and recommendations made.

Joint Meetings with WFIWC—motions passed to meet in 2004, have dual program chairs, form a planning committee in 2001 for the joint meeting.

2001

Standing Committees—proposal to reorganize Disease Control Committee tabled.

2002

Standing Committees—motion passed to disband the Disease Control Committee and establish a Nursery Pathology Committee.

2004

Outstanding Achievement Award—changes to the Bylaws for this award were proposed and accepted by the membership.

Executive Committee—motion to make Webmaster an official position on the committee was approved.

2007

Standing Committees—motion passed to create both an ad hoc Foliar and Shoot Diseases Committee and a Climate Change Committee.

2008

Digital Proceedings—motion to make WIFDWC proceedings available on the website was approved.

2009

Standing Committees—motion passed to confirm the Foliage and Twig Diseases Committee as a standing committee.

2010

Standing Committees—motion passed to confirm the Climate Change Committee as a standing committee.

Fund Raising—the first WIFDWC Silent Auction was held to raise funds for graduate student travel awards.

2011

Standing Committees—motion passed to add the Student Scholarship Committee as a standing committee.

Business Meeting—motion passed outlining requirements needed to pass a motion by means of an electronic ballot.

Bylaw items changed during the October 13, 2012 revision:

- Clarification of terminology and titles;
- Inclusion of option for electronic balloting;
- Clarification of WIFDWC officers duties and responsibilities;
- Updating of committees structure;
- Formalization of Outstanding Achievement Award and Student Travel Awards; and
- Allowing stand committee chairs to expend funds and enter into contracts.

WIFDWC OUTSTANDING ACHIEVEMENT AWARD RECIPIENTS

Year	Recepient	Meeting	Comments
2000	Lew Roth	Kailua-Kona, HI	For pioneering work on *Phytophthora lateralis*, *Armillaria* and dwarf mistletoes, and for inspiration and leadership of a generation of plant pathology students and colleagues.
2000	Duncan Morrison	Kailua-Kona, HI	For long-standing contributions to forest pathology research, especially in relation to roots diseases and tree hazards.
2001	Bob Gilbertson	Carmel, CA	For contributions to the taxonomy and identification of wood-inhabiting basidiomycete fungi.
2002	No award given.		
2003	Everett Hansen.	Grants Pass, OR	For strong leadership in forest pathology including research on the biology and management of tree and seedling diseases of western conifers
2004	Bob James	San Diego, CA	For strong leadership in forest pathology especially technology transfer and research on the biology and management of forest nursery diseases for growers and nursery pathologists throughout the West.
2005	Walt Thies	Jackson, WY	For sustained long-term high quality research on laminated root rot and other root diseases of forest trees.
2006	Bart van der Kamp	Smithers, BC	In recognition of outstanding lifetime contribution to tree disease research and for inspiring a generation of students and colleagues in the field of forest pathology.
2006	Alan Kanaskie	Smithers, BC	For outstanding leadership, as a practicing forest pathologist, in the management of Swiss Needle Cast and Sudden Oak Death.
2007	Richard Hunt	Sedona, AZ	In recognition of his valuable research and extension efforts on white pine blister rust, along with many other contributions to forest pathology and biology.
2008	No award given		
2009	Bill Jacobi	Durango, CO	In recognition of your 30-plus years as an educator, researcher, organizer, advocate and practitioner of forest pathology.
2009	Bob Edmonds	Durango, CO	In recognition of your 40-plus years as, an educator, researcher, organizer, advocate and practitioner of forest pathology and ecology.
2010	Paul Hennon	Valemount, BC	For sustained, significant contributions to our knowledge and understanding of forest disease dynamics and ecology.
2011	Susan Frankel Ellen Goheen	Leavenworth, WA	For leadership in the science and practice of forest pathology and for critical contributions to the management of Sudden Oak Death.

OAA COMMITTEE MEMBERS

Year	Chair	Members	
2000	J. Byler	W. Littke	B. van der Kamp
2001	W. Littke	B. van der Kamp	R. Sturrock
2002	B. van der Kamp	R. Sturrock	G. Filip
2003	R. Sturrock	G. Filip	
2004	G. Filip	D. Goheen	S. Zeglen
2005	D. Goheen	S. Zeglen	D. Shaw
2006	S. Zeglen	D. Shaw	B. Ferguson
2007	D. Shaw	B. Ferguson	R. Reich
2008	B. Ferguson	R. Reich	E. Goheen
2009	R. Reich	E. Goheen	P. Angwin
2010	E. Goheen	P. Angwin	H. Kope
2011	P. Angwin	H. Kope	B. Jacobi
2012	H. Kope	B. Jacobi	P. Hennon

STANDING COMMITTEES AND CHAIRS, 1994—2011

Committee	Chairperson	Term
Hazard Trees	J. Pronos	1994—2005
	P. Angwin	2006—present
Dwarf Mistletoe	R. Mathiasen	1994—2000
	K. Marshall	2001—2003
	F. Baker	2004—present
Root Disease	G. Filip	1994—1995
	E. Michaels Goheen	1996—2005
	B. Ferguson	2006—2009
	M. Cleary	2010—2011
	B. Lockman	2012-
Rust	J. Schwandt	1994, 2005
	R. Hunt	1995—2004
	H. Kearns	2006—2011
	H. Maffei	2012
Disease Control[a]	B. James	1995—2002
Nursery Pathology	B. James	2002—2005
	K. Mallams	2007—2010
	W. Littke	2011—present
Foliar and Twig Diseases[b]	H. Kope	2007—present
Climate Change[c]	S. Frankel	2007—2008
	S. Frankel & D. Shaw	2009—present

[a]Disease Control committee was disbanded in 2002.
[b]Foliar and Twig Diseases committee was made full charter member in 2009.
[c]Climate Change committee was made full charter member in 2010.

PAST ANNUAL MEETING LOCATIONS AND OFFICERS

Meetings and Officers, 1953—2011

Annual	Year	Location	Chairperson	Secretary- Treasurer	Program Chair	Local Arrangements
1	1953	Victoria, BC	R. Foster			
2	1954	Berkeley, CA	W. Wagener	P. Lightle		
3	1955	Spokane, WA	V. Nordin	C. Leaphart	G. Thomas	
4	1956	El Paso, TX	L. Gill	R. Davidson	V. Nordin	
5	1957	Salem, OR	G. Thomas	T. Childs	R. Gilbertson	
6	1958	Vancouver, BC	J. Kimmey	H. Offord	A. Parker	
7	1959	Pullman, WA	H. Offord	R. Foster	C. Shaw	
8	1960	Centralia, WA	A. Parker	F. Hawksworth	J. Parmeter	K. Shea
9	1961	Banff, AB	F. Hawksworth	J. Parmeter	A. Molnar	G. Thomas
10	1962	Victoria, BC	J. Parmeter	C. Shaw	K. Shea	R. McMinn
11	1963	Jackson, WY	C. Shaw	J. Bier	R. Scharpf	L. Farmer
12	1964	Berkeley, CA	K. Shea	R. Scharpf	C. Leaphart	H. Offord
13	1965	Kelowna, BC	J. Bier	H. Whitney	R. Bega	A. Molnar
14	1966	Bend, OR	C. Leaphart	D. Graham	G. Pentland	D. Graham
15	1967	Santa Fe, NM	A. Molnar	E. Wicker	L. Weir	P. Lightle
16	1968	Couer D'Alene, ID	S. Andrews	R. McMinn	J. Stewart	C. Leaphart
17	1969	Olympia, WA	G. Wallis	R. Gilbertson	F. Hawksworth	K. Russell
18	1970	Harrison Hot Springs, BC	R. Scharpf	H. Toko	A. Harvey	J. Roff
19	1971	Medford, OR	J. Baranyay	D. Graham	R. Smith	H. Bynum
20	1972	Victoria, BC	P. Lightle	A. McCain	L. Weir	D. Morrison
21	1973	Estes Park, CO	E. Wicker	R. Loomis	R. Gilbertson	J. Laut
22	1974	Monterey, CA	R. Bega	D. Hocking	J. Parmeter	
23	1975	Missoula, MT	H. Whitney	J. Byler	E. Wicker	O. Dooling
24	1976	Coos Bay, OR	L. Roth	K. Russell	L. Weir	J. Hadfield
25	1977	Victoria, BC	D. Graham	J. Laut	E. Nelson	W. Bloomberg
26	1978	Tucson, AZ	R. Smith	D. Drummond	L. Weir	R.Gilbertson
27	1979	Salem, OR	T. Laurent	T. Hinds	B. van der Kamp	L. Weir
28	1980	Pingree Park, CO	R. Gilbertson	O. Dooling	J. Laut	M. Schomaker
29	1981	Vernon, BC	L. Weir	C.G. Shaw III	J. Schwandt	D. Morrison R. Hunt
30	1982	Fallen Leaf Lake, CA	W. Bloomberg	W. Jacobi	E. Hansen	F. Cobb J. Parmeter
31	1983	Coeur d'Alene, ID	J. Laut	S. Dubreuil	D. Johnson	J. Schwandt J. Byler
32	1984	Taos, NM	T. Hinds	R. Hunt	J. Byler	J. Beatty E. Wood
33	1985	Olympia, WA	F. Cobb	W. Thies	R. Edmonds	K. Russell
34	1986	Juneau, AK	K. Russell	S. Cooley	J. Laut	C.G. Shaw III
35	1987	Nanaimo, BC	J. Muir	G. DeNitto	J. Beatty	J. Kumi
36	1988	Park City, UT	J. Byler	B. van der Kamp	J. Pronos	F. Baker
37	1989	Bend, OR	D. Goheen	R. James	E. Hansen	A. Kanaskie

Meetings and Officers, 1953—2011 (cont.)

Annual	Year	Location	Chair-person	Secretary	Treasurer	Program Chair	Local Arrangements	Historian	Web Coordinator
38	1990	Redding, CA	R. Hunt	J. Hoffman	K. Russell	M. Marosy	G. DeNitto		
39	1991	Vernon, BC	A. McCain	J. Muir	K. Russell	R. Hunt	H. Merler		
40	1992	Durango, CO	D. Morrison	S. Frankel	K. Russell	C.G. Shaw III	P. Angwin		
41	1993	Boise, ID	W. Littke	J. Allison	K. Russell	F. Baker	J. Hoffman		
42	1994	Albuquerque, NM	C.G. Shaw III	G. Filip	K. Russell	M. Schultz	D. Conklin T. Rodgers		
43	1995	Whitefish, MT	S. Frankel	R. Mathiasen	K. Russell	R. Mathiasen	J. Taylor J. Schwandt		
44	1996	Hood River, OR	J. Kliejunas	J. Beatty	J. Schwandt	S. Campbell	J. Beatty K. Russel		
45	1997	Prince George, BC	W. Thies	R. Sturrock	J. Schwandt	K. Lewis	R. Reich K. Lewis		
46	1998	Reno, NV	B. Edmonds	L. Trummer	J. Schwandt	G. Filip	J. Hoffman J. Guyon	D. Morrison	
47	1999	Breckenridge, CO	F. Baker	E. Michaels Goheen	J. Schwandt	J. Taylor	D. Johnson		J. Adams
48	2000	Waikoloa, HI	W. Jacobi	P. Angwin	J. Schwandt	S. Hagle	J. Beatty		
49	2001	Carmel, CA	D. Johnson	K. Marshall	J. Schwandt	A. Kanaskie	S. Frankel		
50	2002	Powell River, BC	B. van der Kamp	H. Maffei	J. Schwandt	P. Hennon	S. Zeglen R. Diprose		
51	2003	Grants Pass, OR	E. Hansen	B. Geils	J. Schwandt	H. Merler	E. Michaels Goheen		
52	2004	San Diego, CA	E. Goheen	B. Lockman	J. Schwandt	H. Merler K. Lesiw	J. Pronos J. Kliejunas S. Smith		
53	2005	Jackson, WY	M. Fairweather	H. Merler J. Guyon	J. Schwandt	K. Burns	J. Hoffman F. Baker J. Guyon		
54	2006	Smithers, BC	K. Lewis	M. Jackson	J. Schwandt	B. Lockman	A. Woods		
55	2007	Sedona, AZ	S. Zeglen	M. McWilliams	J. Schwandt	J. Worrall	M. Fairweather B. Geils B. Mathiason		
56	2008	Missoula, MT	G. DeNitto	F. Baker	J. Schwandt	W. Littke	B. Lockman M. Jackson		

Meetings and Officers, 1953—2011 (cont.)

Annual	Year	Location	Chair-person	Secretary	Treasurer	Program Chair	Local Arrangements	Historian	Web Coordinator
57	2009	Durango, CO	G. Filip	J. Adams	J. Schwandt	D. Shaw	K. Burns B. Jacobi J. Worrall R. Mask J. Blodgett	R. Sturrock	J. Adams
58	2010	Valemount, BC	R. Sturrock	M. Fairweather	J. Schwandt	D. Goheen	M. Cleary R. Reich		
59	2011	Leavenworth, WA	P. Angwin	S. Zeglen	H. Kearns	A. Kanaskie	G. Filip A. Saavedra A. Ramsey-Kroll D. Omdal		

Bylaws passed at 1998 WIFDWC Business Meeting identify officers as chairperson and secretary elected at annual business meeting and treasurer and historian, elected every five years.

IN MEMORIAM

John R. Parmeter Jr.

Professor of Environmental Science, Policy and Management, Emeritus
UC Berkeley, 1927 – 2010

John Richard (Dick) Parmeter Jr. was born on September 16, 1927, and died of heart failure on October 27, 2010. Dick grew up in The Dalles, Oregon. At the age of 17 he joined the U.S. Marines, and was preparing for the invasion of Japan when the atomic bomb ended the war.

He attended Eastern Oregon College from January 1947 to June 1948, and then transferred to Oregon State College, where he graduated with honors in 1951, earning a degree in botany with a minor in forestry. He then went east to the University of Wisconsin, where he earned a Ph.D. in plant pathology in 1955 and as a bonus met and married his wife, Anita. His dissertation was entitled "Oak wilt development in bur oaks." Following graduation he worked for the U.S. Forest Service at the Lake States Experiment Station studying "Microclimate in relation to distribution of white pine blister rust." In 1957 he was hired as assistant professor of plant pathology at the University of California, Berkeley, where he stayed until his retirement as professor in 1990.

Since Dick was the first professor in the Department of Plant Pathology to teach and conduct research primarily on forest diseases, the challenges of developing a teaching and research program were daunting. His stated philosophy was a "desire to do good work, to entertain and amuse my curiosity, to maintain a high standard for myself and the department, and to somehow balance these goals in reasonable proportion."

He taught courses on the biology of plant parasitic fungi and forest pest management, and lectured in several classes in the College of Natural Resources. Dick mentored many graduate students and was sought after as a teacher and counselor who had the interest and would take the time to guide students in their studies and research programs.

He loved research and teaching, but he believed that if you earned a Doctor of Philosophy degree you should also have some understanding of classical literature. For example, he might ask a student, "What do you think was the strategy and significance of the Greeks' victory over the Persians at the Battle of Marathon?" Dick did not necessarily expect an answer; he just wanted to see how the student would respond. A favorite comment of his was Klipstein's "Law No. 2: Firmness in delivery dates is inversely proportional to the tightness of the schedule." In a serious world, Dick enjoyed such relaxing moments.

Dick's research achievements were many and varied. He authored and co-authored dozens of scientific and technical papers on various aspects of forest pathology. He became a world authority on the root pathogen Rhizoctonia and contributed a substantial amount of new information on the biology, epidemiology, and control of a serious disease of conifer forests caused by the dwarf mistletoes in the genus Arceuthobium. He was the first to identify air pollution as the cause of the death of pines in southern California and later contributed substantially to a better understanding of the disease and its mitigation. He worked in Yosemite National Park to identify and aid in developing management strategies for diseases that were causing serious tree losses as well as hazardous conditions to the public in this national treasure. Other examples of his interests and achievements are studies on the interactions of weather, insects, and pathogens on death and damage in conifer forests; the lethal effects of components of smoke on forest fungal pathogens; research on "gall rusts" of conifers with regard to their potential impact if introduced on conifers elsewhere in the world; research on the identification of several unknown diseases in California's urban forest environment. Dick was also among the earliest

investigators to embrace the concept of integrated pest management (IPM) (1981), and along with a few others, recognized and published on the effects of weather modification (climate change) on forests and forest diseases and pests.

Dick was an enthusiastic supporter of the College of Natural Resources and departmental functions. Over the years he chaired and served on numerous college and departmental committees, including supervising many Ph.D. candidates in plant pathology. He was also an active member and served in many outside organizations: American Phytopathological Society; Western International Forest Disease Work Conference; California Forest Pest Action Council (Advisor to Governor); and, Mycological Society of America.

Dick's leadership, along with his joy in research and working with others, led to a very productive career. Through these cooperative efforts he was often granted substantial financial research support from the state and the U.S. Department of Agriculture.

On a more personal note, I had the honor and pleasure of being Dick's first graduate student. He was a new professor and I a forestry graduate and recently discharged GI. We seemed to connect with one another immediately. Through patience and understanding he mentored me to completion of my graduate program. By good fortune I was employed by the U.S. Forest Service Experiment Station in Berkeley and had the opportunity to collaborate with Dick on pathology research over the duration of our careers. Over that time we not only became working colleagues, but also close friends. We both loved the out of doors, and spent many days together camping, fishing, and hunting. He was an accomplished artist, but painted mostly for his own enjoyment. He also loved music. We spent many hours together in the lab or traveling to and from field research listening to his taped classical music.

Shortly after retirement, Dick and his wife Anita moved to Florence, Oregon, where they could enjoy the beautiful coastal environment. Unfortunately, I saw less of him after that. In 2008, former students organized a reunion in Idaho, in which many of his students and colleagues paid tribute to their beloved professor and friend.

Dick is survived by his wife Anita, son Jack, and daughter Amy.

"To sit on a rock or log where no one has sat before and to contemplate the sweep of geology or biology unfettered by civilization is close to a religious experience. The only things like it are love and music."

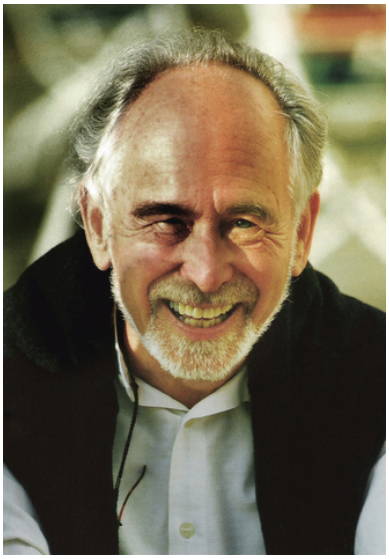
John R. Parmeter (1927-2010)

Robert F. Scharpf
http://universityofcalifornia.edu/senate/johnrparmeterrr.html, downloaded 3/20/12

IN MEMORIAM

Peter Schütt

Former Professor of Forest Botany at the Ludwig-Maximilians University, Munich, dendrologist and forest pathologist, founder of the journal Forest Pathology and editor-in-chief from 1970 until 1990, died on the 9th October 2010.



Peter Schütt was born on 13th September 1926 in Berlin. His scientific career started with a dissertation on dendroclimatology of oaks at the university of Berlin in 1954, followed by an appointment at the Federal Forest and Wood Research Institute in Reinbek, where he worked on genetics and breeding of Scots pine. In 1963, he received his postdoctoral lecturer qualification for his thesis 'Lophodermium needle cast of Scots pine in relation to provenance and planting location' and in 1969 became associate professor at the University of Saarbrücken, Germany. In 1970, he accepted the position of a full professor for Anatomy, Physiology and Pathology of Plants (later renamed Forest Botany) at the Department of Forest Sciences, University of Munich. He kept this position until his retirement in 1994.

Peter Schütt was an impressive personality – extremely knowledgeable, creative, communicative, approachable, hospitable and human. He never avoided controversial discussions, and he convinced his audience by use of the highest intellectual standards and sparkling wit. His central concern was to sustain the natural environment, particularly trees and forests. He translated this idea into science as well as into public policy, long ago before terms like Environmental Sciences, Biodiversity and Sustainability had permeated the common awareness. Already at a very early stage of his career, he studied the impact of air pollutants on forest trees in close collaboration with atmospheric chemists. Later he frequently tackled hot topics, for example side effects of pesticides on forests, the impact of exhaust emissions on trees and finally forest decline in relation to large scale air pollution. His commitment to strict air pollution control strongly influenced European legislation and he thereby contributed significantly to the preservation of forest and human health.

In Forest Pathology, Peter Schütt pursued a holistic approach, considering likewise biotic and abiotic disease causing agents as well as host genetics and environment. He performed, initiated or supervised many research projects which distinctly improved our understanding of the aetiology, epidemiology and control of major tree diseases, e.g. Lophodermium needle cast, Heterobasidion root rot and various tree declines. At a very early stage, he integrated endophytic microorganisms conceptually and experimentally into forest pathology and initiated studies on interactions between pathogens, endophytes and air pollutants. In the year 1970, he founded the European Journal of Forest Pathology (now Forest Pathology) which quickly became an internationally renowned institution for our field.

Peter Schütt was also a very active dendrologist: he authored several dendrology books and, most importantly, he initiated the Encyclopedia of Woody Plants, a comprehensive series of monographs on tree species of the world for which he acted as editor for several years and to which he contributed many species descriptions.

Peter Schütt was an enthusiastic and inspiring university teacher. Many generations of students enjoyed his lectures, which were very spirited, demonstrative, rich in content and intellectually stimulating. He was always very open to new ideas and cooperated intensively with colleagues around the world. His major skill was to motivate his co-workers. He convinced us that science can be real fun. We were allowed to work in great freedom; Peter Schütt guided us using a very long line. 'Motivation by confidence' was his secret formula. His critical, but very constructive style of reviewing scientific manuscripts was highly appreciated by authors all over

the world. Peter Schütt provided a real catalyst to encourage and advance many people – students (including 26 PhD students), colleagues and environmentalists.

After gaining emeritus status in 1994, he focused his energy in the editing of and contributing to the Encyclopedia of Woody Plants. Now, Peter Schütt has passed away peacefully after a few years of continuously declining health. We will remember him as a great scientist, teacher and friend.
Forest Pathology, Volume 41, Issue 1, pages 83–84, February 2011, DOI: 10.1111/j.1439-0329.2010.00706.x

IN MEMORIAM

A tribute to Richard T. "Dick" Bingham (1918–2010)

A founding member of the White Pine Chapter, Idaho Native Plant Society from Richard Bingham's obituary and White Pine Chapter archives, compiled and edited by Dennis Ferguson and Nancy Miller

Richard T. "Dick" Bingham of Juliaetta died Saturday, December 18, 2010 in Moscow at the age of 92. He was born March 1, 1918 in New York City. He attended grade school and high school in New Jersey, leaving there in September of 1936 to attend the University of Idaho in Moscow. He graduated in 1940 with a BS in Forestry and continued in graduate school in 1941-42 to attain the Degree of Master of Sciences in Forest Pathology.

In October of 1942 Dick volunteered for the Marine Corps and was entered in an Officer's Candidate Unit at Quantico, VA. He remained in the Corps through the Mariana Islands Campaign and was honorably discharged in January of 1946.

He commenced Forestry Research in Spokane, WA in 1946, and almost all of his research was concerned with controlling the White Pine Blister Rust Disease. From 1950-75, mostly while stationed at the Forest Service Forestry Science Laboratory at Moscow, he pioneered in white pine tree breeding toward genetic control of the Blister Rust Disease.

Here he worked closely with Forest Service and other scientists throughout the United States and the World. Early research results were promising and he was selected for the US Department of Agriculture Superior Service Award. In 1974 his entire research unit received a similar award. Dick authored or co-authored over 40 scientific publications on blister rust, white pine silviculture, white pine rust resistance, and local flora and plant checklists. Two annotated checklists of relevance still to those who find their recreation in Hells Canyon or the Seven Devils are the Guide to the Common Plants of the Seven Devils Mountains (authored by Richard T. Bingham and Clyde J. Miller) and Guide to the Common Plants of Hells Canyon (Richard T. Bingham and Douglass M. Henderson). Both booklets are out of print, however they have both been digitized and you can access them from the Documents links on the White Pine Chapter's web site: (www.whitepineinps.org/WPCheck.html)

Probably Dick's most cherished honor was his selection as the 1984 Honor Alumnus of the University of Idaho, College of Forestry, Wildlife and Range Sciences. Dick organized a group of people interested in native plants. Following a 1989 visit from Susan Bernartus of the Pahove Chapter, a recent graduate of the University of Idaho, this group became the White Pine Chapter of Idaho Native Plant Society. Dick was the chapter's first president and In January 1989 he gave the first presentation to a chapter meeting at the University of Idaho Life Sciences building. The program was a slide-lecture on the 'Flora of the Seven Devils.'

Dick co-led the chapter's first field trip to Skyline Drive (Mary Minerva McCroskey State Park) with Ray Boyd. He was to co-lead a number of other field trips over the years. Many members still remember the weekend campouts Dick led to Seven Devils in July 1990, August 1993 (with Clyde Miller), and August 1997.

Dick remained active in the White Pine Chapter for many years, and was still a member at the time of his death in December. We shall miss his humor and the sparkle in his eyes when he talked about his favorite topics, especially native plants.

Sage Notes is a publication of the Idaho Native Plant Society Volume 33 (2) May 2011. http://www.idahonativeplants.org/news/SageNotesMay2011.pdf downloaded 3/20/12

IN MEMORIAM

KEITH RAYMOND SHEA 1924-2010

Keith Shea passed away in Olympia with family at his side on September 19, 2010. He was born on December 24, 1924 in Greenfield, IA in a rural farmhouse to Helen and Richard Shea. During the depression he and his mother lived with his grandparents. Keith ran a trap line which he visited twice daily and with the money he made was able to gradually build a flock of sheep which he then sold as needed to help with expenses. His mother then married William Freeze. Keith graduated from Dexter High in 1942 and joined the military 6 months later. Dad was a veteran of WWII and proudly served on the USS Charleston in the Aleutians, China and the Battle of Attu Island as a medic. He attained a BS in Forestry from the University of Minnesota where he married Edith Gasser. He earned a PhD in Forest Pathology from the University of Wisconsin in 1954.

His forestry career began in Centralia with the Weyerhaeuser Company as one of the initial research team. While in Centralia, he was scout leader of Weyerhaeuser Troop 44, and a member of the local Kiwanis. Keith was also on the Centralia School Board and along with other community leaders brought the community college to Centralia. In 1966 he moved to Corvallis, OR to become head of the US Forest Research Station. He married his current wife, Dolores Kinder Wall in 1968. The couple then moved to Washington DC in 1971 and he rose to the level of Associate Deputy Chief, Forest Research, USDA Forest Service. After years of public service, he retired in 1986. Keith's international research and published papers in the areas of acid rain, the gypsy moth, the pine bark beetle, high yield forests and reforestation continue to have merit in managing today's international forests.

Dad loved to travel and to meet people. He and his wife, Dolores, traveled as often as possible during his career and later during retirement. Keith especially loved New Orleans and celebrating Mardi Gras with friends in Apache Junction, AZ, where he and Dolores wintered for 20 years. His love of nature and the great outdoors along with camping, hiking, fishing, woodworking and cooking gourmet food has been passed on to all who knew him. He is survived by his loving wife of 42 years, Dolores. Children include David Shea, Robin Hilleary, Julie DePuydt, Dan Shea (deceased) Paula Shea, Matt Shea, Joe Wall, Peggy Harris, and Penny Schaefer. The family, including 16 grandchildren, 18 great-grandchildren and his cat Sammy will miss him greatly. Remembrances, in lieu of flowers can be made to the Boy Scouts of America, The Nature Conservatory or to a favorite charity. A graveside memorial service will be held at Mountain View Cemetery in Centralia on October 2nd at 2:00PM. Arrangements are under the direction of Newell-Hoerling's Mortuary.

Published in the Chronline on 9/28/2010
http://www.legacy.com/Obituaries.asp?Page=LifeStoryPrint&PersonID=145690994, Downloaded 3/20/12

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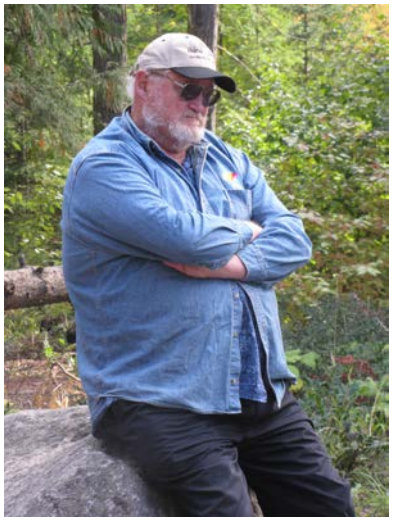


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We have lost track of the following Honorary Life Members. If you know of their whereabouts, please contact Fred Baker at 435-797-2550 or fred.baker@usu.edu

Paul Aho
Ed Andrews
Alvin Funk
Bob Harvey
John Hopkins
Paul Lightle
Don Norris
Art Partridge
Jack Roff
Jim Trappe
Lori Trummer
Bob Tinnin
Mike Sharon
Richard B Smith
Ed Wood
Conrad Wessela

DECEASED MEMBERS

Stuart "Stuie" Andrews

Jesse Bedwell

Robert Bega

Warren Benedict

John Bier

Richard “Dick” Bingham

Bill Bloomberg

Roy Bloomstrom

Thomas "Buck" Buchannan

Don Buckland

Hubert "Hart" Bynum

Elmer Canfield

Ross Davidson

Oscar Dooling

Norm Engelhart

Ray Foster

Dave French

Lake S. Gill

Clarence "Clancy" Gordon

John Gynn

John Hansbrough

Hans Hansen

Homer Hartman

George Harvey

Frank G. Hawksworth

Dwight Hester

Tommy Hinds

Brenton Howard

John Hunt

Paul Keener

James Kimmey

Andrea Koonce

Don Leaphart

Tom McGrath

Neil E. McGregor

Jim Mielke

D. Reed Miller

Vergil Moss

Harrold Offord

Nagy Oshima

Lee Paine

John Palmer

John "Dick" Parmeter Jr.

Clarence Quick

Lew Roth

Dave Schultz

Peter Schutt

Charles G. Shaw

Keith Shea

Albert Slipp

Willhelm Solheim

Albert Stage

Phil Thomas

Willis Wagener

Charles "Doc" Waters

Larry Weir

Ed Wicker

John Woo

Ernest Wright

Wolf Ziller

Back Row: Leif Mortenson, Greg Filip, Dan Omdal, Sasquatch, Alan Kanaskie, John Browning, Bob Edmonds, Elisa Becker

Front Row: Ebba Peterson, Laura Sims, Helen Maffei, Joe Hulbert, Robin Mulvey, Sarah Navarro, Rich Hunt, Danielle Martin

Back Row: Cynthia Ash, Bill Jacobi, Will Littke, Bill Woodruff, Everett Hansen, Mike McWilliams, Paul Hennon
Front Row: Bruce Moltzen, Duncan Morrison, Eric Smith, Ellen Goheen, Gary Chastagner

Back Row: Walter Thies, Susan Frankel, Bob Mathiesen, Alex Woods, Todd Ramsfield, Blakey Lockman
Front Row: Gail Thies, Karen Hutton, Amy Ramsey-Kroll, Lina Farfan, Judy Adams

Back Row: James Hoffman, Mike Cruickshank, Marianne Elliot, Mary Lou Fairweather, Simon Shamoun, Kim Camilli, Michael Murray

Front Row: Brent Oblinger, Rona Sturrock, Anna Leon, Fred Baker, Stefan Zeglen, Tim Owen

Back Row: James Morin, Santa, Yun Wu, Bill Jones, John Schwandt, Holly Kearns, Forrest Oliveria

Front Row: Mahsa Khorasani, Abbey Hudler, Maia Beh, Nathan Johnson, Jim Hadfield

Back Row: Dave Shaw, Ned Klopfenstein, Terry Shaw, Pete Angwin, Richard Reich, Borys Tkacz
Front Row: Danny Norlander, Amy Ross-Davis, Kathy Lewis, Jim Walla, John Hanna, Paul Zambino