Proceedings of the 55th Annual Western International Forest Disease Work Conference

Radisson Poco Diablo Resort
Sedona, Arizona

October 15 to 19, 2007

Compiled by:
Michael McWilliams
Oregon Department of Forestry
Insect and Disease Section
Salem, Oregon

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&
Patsy Palacios
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Utah State University, Logan

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SPECIAL THANKS!

Photos were taken by John Schwandt, Michael McWilliams, Pete Angwin, Rona Sturrock and Walt Thies

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PROGRAM

MONDAY, OCTOBER 15TH

1:00 – 5:00 pm	Workshop: **Forest Diseases and Climate Change in Western Forests – What Do We Know; What Do We Need to Find Out?** Organized by Susan Frankel, Pacific Southwest Research Station, and Terry Shaw, Western Wildland Environmental Threat Center
3:00 – 5:00 pm	Nursery Meeting
3:00 – 6:00 pm	Registration
5:00 – 7:00 pm	Evening Social (snacks, no-host bar)

TUESDAY, OCTOBER 16TH

7:00 – 8:30 am	Dwarf MistletoeCommittee Breakfast Meeting (Fred Baker)
7:00 – 8:30 am	Registration
8:30 – 8:45 am	Opening and Welcome from WIFDWC Chair Stefan Zeglen
8:45 – 10:00 am	Introductions and Regional Reports
10:30 – 11:30 am	Introductions and Regional Reports (continued)
11:30 – 12:00 pm	2006 Distinguished Achievement Award Recipient (Bart van der Kamp)
12:00 – 1:30 pm	Root Disease Committee Lunch Meeting (Brennan Ferguson)
1:30 – 2:15 pm	Student Papers (Betsy Goodrich, moderator):
	Fuel and Stand Characteristics in Ponderosa Pine Infested With Mountain Pine Beetle, Ips Beetle, and Southwestern Dwarf Mistletoe in Colorado's Northern Front Range (Jennifer Klutsch, Colorado State University)
	Changes in Roadside Soils and Vegetation Through the use of Magnesium Chloride Based Dust Suppression Products (Betsy Goodrich, Colorado State University)
2:15 – 3:00 pm	**Review of SOD** (Ellen Goheen, Forest Service, Forest Health Protection, Central Point, OR)
3:30 – 4:30 pm	**Collaboration and Studies in Mexico** (Brian Geils, moderator)
	Evaluating Growth Impacts of *Arceuthobium vaginatum subsp. vaginatum* on *Pinus cooperi* in Durango, Mexico (Brian Howell)
	An Armillaria Collection Trip To Mexico (Phil Cannon and Ned Klopfenstein)
	Research Initiatives in Northern Baja California, Mexico, and Southern California, USA (Patricia Maloney and Det Vogler)
4:30 - 4:50 pm	**Risk of Interstate Movement of Invasive Tree Pests** (Bill Jacobi, Dept. of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins, CO)
7:00 – 9:00 pm	Posters/Ice Cream Social (Michelle Cleary, poster session moderator)

WEDNESDAY, OCTOBER 17TH

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	Introduction (Susan Frankel, Pacific Southwest Research Station, Berkeley, CA)
	Bark Beetle Outbreaks in the West: Influences of Past, Current, and Future Climate Change (Jeffrey Hicke, Department of Geography, University of Idaho, Moscow ID)
	Piñon Ecology and Landscape Change (Craig D. Allen, US Geological Survey, Jemez Mountains Field Station, Los Alamos NM)

Pathology Without Pathogens (Paul Hennon, US Forest Service, State and Private Forestry and Pacific Northwest Research Station, Juneau Forest Health Office Juneau AK)

Pests, Pathogens and Climate Change Interactions in Western Forests: Results of a Recent Concepts Workshop (Terry Shaw, US Forest Service, Pacific Northwest Research Station, Western Wildlands Environmental Threats Assessment Center, Prineville OR)

12:00 – 1:30 pm	Rust Committee Lunch Meeting (Holly Kearns)
1:30 – 3:30 pm	Panel: **Aspen Mortality Trends and Climate** (Jim Worrall & Mary Lou Fairweather, moderators)

Understanding the Causes and Extent of Recent Aspen Decline in the Western Canadian Interior (Ted Hogg, Research Scientist, Forests and Climate Change, Natural Resources Canada, Canadian Forest Service, Northern Forestry Centre, Edmonton, Alberta)

Aspen Decline in Arizona: Abiotic Factors and Sacred Cows (Mary Lou Fairweather, US Forest Service, Southwestern Region, Forest Health Protection, Flagstaff, AZ)

Sudden Aspen Decline in Southwestern Colorado: Site and Stand Factors and a Hypothesis on Etiology (Jim Worrall, US Forest Service, Rocky Mountain Region, Forest Health Management, Gunnison, CO)

Survivor Aspen: Can We Predict Who Will be Voted Off the Island? (Fred Baker, Department of Wildland Resources, Utah State University, Logan, UT, and John Shaw, Forest Inventory and Analysis, Rocky Mountain Research Station, Ogden, UT)

4:00 – 5:00 pm	Business Meeting
6:00 pm	Banquet

THURSDAY, OCTOBER 18TH

8:00 am – 5:00 pm	All-Day Field Trip

FRIDAY, OCTOBER 19TH

8:00 – 10:00 am	**Contributed Papers** (Jim Blodgett, moderator)

Survival and Sanitation of Dwarf Mistletoe-Infected Ponderosa Pine Following Prescribed Underburning (a 10-Year Study in New Mexico) (Dave Conklin, USDA-FS, Forest Health Protection; Brian Geils, USDA-FS, Rocky Mountain Research Station)

Permanent Plots for Measuring Spread and Impact of Douglas-Fir Dwarf Mistletoe in the Southern Oregon Cascades: Results of the Ten Year Remeasurement (Katy M Mallams, USDA-FS, Forest Health Protection)

Fusarium oxysporum **(Koa Wilt) in** *Acacia koa* **– First Resistance Results From Greenhouse Testing** (Nick Dudley, Hawaii Agriculture Research Center; Richard Sniezko, Dorena Genetic Resource Center; Robert James and Phil Cannon, Forest Health Protection)

Cronartium ribicola **in Whitebark Pine and Southwestern White Pine – First Resistance Screening Results at Dorena GRC** (Richard Sniezko, Angelia Kegley, and Robert Danchok, USDA-FS, Dorena Genetic Resource Center)

Resistance to *Phytophthora lateralis* **in Port-Orford-Cedar – Recent Results From Greenhouse Screening** (Richard Sniezko, Angelia Kegley, and Scott Kolpak, Dorena Genetic Resource Center; Everett Hansen & Paul Reeser, Oregon State Univ., Dept. of Bot. and Plant Pathol.)

Interactions Between Western Gall Rust and its Pine Hosts in the Sierra de San Pedro Martir, Baja California Norte, Mexico (Detlev R Vogler, Institute of Forest Genetics, USDA-FS, PSW Research Station)

WIFDWC Outstanding Achievement Award 2006
Dr. Bart van der Kamp
Learning to Teach – Again and Again

Dear friends and colleagues,

I'm greatly honoured by being asked to address this august group once more. I did a quick count and I believe this is the 21st WIFDWC I have attended. I hope there are many more to come. I well remember my first one: Coeur d'Alene in 1968. I had been to scientific meetings before, but this was something else. I had of course been forewarned by the stories Jack Bier, my predecessor at UBC, used to tell, but nothing prepared me for what I found. What a bunch of characters. And all the names I had read about but never met. There was Willis Wagener, Frank Hawksworth, Dick Parmeter, Lew Roth, Don Leapheart, Bob Scharft, Art Partridge, Oscar Dooling, Larry Weir, Keith Shea, Bob Gilbertson, and so on. Some of them are here today. All male of course, although there had been the odd woman at previous meetings, and the social customs of the time in their full glory: the social achievement award was a serious business. I'm afraid never attained that honour.

How did I come to be a forest pathologist? From as early as I can remember, back in Holland in the 1940's, I wanted to be a forester. You can imagine my delight when the family immigrated to British Columbia in 1955 and I started to explore the forests around Vancouver. During my last years as undergraduate I was particularly fascinated by Jack Bier's classes in Forest Pathology, and through his good offices I ended up with a Research Fellow appointment at the University of Aberdeen in Scotland, to do my PhD with Stan Murray on *Peridermium pini*. Just after starting there I married Adriana, the love of my life, and she is here with us today. Our oldest was born shortly after in Aberdeen. Jack Bier met an untimely death while we were in Scotland. A couple of months later I got a phone call out of the blue from then dean Gardner asking if I wanted the job at UBC (no search committees and interviews in those days). He had one condition: I had to be finished before I left Scotland. That gave me seven months, and I had barely started to write. Well I remember the very last night before the deadline when we finally had the five copies lying on the floor of our digs, all done on a giant Underwood typewriter with four carbon copies. And so I ended up as the Forest Pathologist at the UBC at the immaure age of 25.

That brings me to the title of my talk today: 'Learning to teach – again and again'. At this point I would like my former students to step out. They've heard it all before and probably don't believe it anymore, and they might choose to bring up some embarrassing events that I would sooner keep quiet.

We arrived in Vancouver at the end of November, and in January I was to start teaching two courses. Well, I thought I was ready. I had the first five lectures pretty well worked out. But then came the first lecture. I was through my material for the first lecture in 10 minutes, and in desperation carried on with the second and I was well into the third before the bell rescued me. I wouldn't gladly repeat those first three months, and I'm still amazed that the students didn't walk out on me after the first few lectures.

Slowly things improved. I grew a little more at ease in front of the class, and after the first sets of exams I had a better idea of what made sense to students and what didn't. However I had many lessons to learn still. One time Adriana brought several of our kids into class to give them an idea of what I was doing. After ten minutes or so of sitting in the back row they started to get fidgety and so she took them away. As they were leaving, one of the students turned around and said 'boring eh'. The kids were scandalized, but it

set me to thinking. How could such a fascinating subject as tree diseases be boring!

Boring? But how? Was it me? Was it inherent in the subject matter? Or something else? I well remember a boring lecturer in mycology. A full year course MWF at 8:30. I liked the subject well enough, and did alright in the course. But I was there and awake for the full hour only five times in the whole year. My class notes would start off well, quickly deteriorate into some illegible scrawls, and then stop altogether. Was I doing that to these students? Well, maybe, but at least they were mostly awake. The subject matter then? I couldn't see that it was any less exciting than any of the other courses.

I finally hit on what I still think is the main cause: boring because irrelevant. Let me explain. Most of carry around in our minds a set of ideas of how the world operates and how we fit in. Let me call it our 'world-grasp' to coin a new word. True, that world-grasp is not always perfectly integrated or critically examined, but it serves to get us through the day. For most people new ideas are interesting if they speak to and lead to a change in that world-grasp. Academic learning, however doesn't do much of that for many students. You go to school because you have to, you do what is required to pass the courses, but it is all quite irrelevant to the important issues in your life – hobbies, sports and personal relationships, and so you forget it as soon as you've written the final. True, occasionally you run into an inspired teacher, and those who are exposed to several of these are fortunate indeed. No wonder that two years after taking a course students remember only about 5% of the material presented in lecture, and a random 5% at that – not necessarily the important stuff.

It so happened that at this time (mid 70's) there was a revolution going on in the faculty. When I came back in '67, the old guard of professors who had been there since the beginning of time, was still in charge. However the late sixties saw a surge of new hiring – all young guys, (no women yet) and by the mid 70's they were in the majority, (and that cohort remained in charge until about 10 years ago). Forestry was changing from how to get the logs out as cheaply as possible to how to manage the land in a stewardly fashion. Ecology was the in-word. And so the 'new guard' pushed through a major curriculum revision designed in large part to give the students an integrated understanding of how a living forest operates, and how it may be manipulated. That knowledge was to be a major part of their 'world-grasp'. At first it was still all about trees, but in time the whole forest was included. The idea was that we would form a common understanding in the minds of all students. Integration was the key word. Did it work? Only so-so. The trouble was (and still is) that course content at UBC (and most universities that I know) is decided solely by the instructor. After all, she is the expert, and often the only expert around in that particular discipline. The time allotted to subjects changed, as did their sequence, but the content remained largely the same.

The big breakthrough for us came serendipitously in the early 80's with the establishment of a field school in the BC Interior. Because of the remote location (not even phone access) it worked out that we would typically have 10 or so faculty members in attendance for the whole time, both day and evening. The outcome of course was that at each site we visited, almost all of them had something to say, often providing alternative interpretations, and debating them in front of the students. And the debates would carry on in the evening. Finally we were getting some real integration among faculty members! For me it was also an eye opener (literally). I learned to see and diagnose a lot of things that I had been blissfully ignorant of.

Teaching is rewarding. I can honestly say that except for the first 5 years, teaching undergraduates has been one of the most enjoyable parts of my job. I would even go farther, and say the most enjoyable teaching was the many years that I taught the introductory course in Forestry to the new incoming students. When I've given what I think was a good lecture, I can walk all the way back to my office without

touching the ground! Trouble is that what I consider a good lecture and what the students think is often not the same. And there are always surprises. One time one of the ladies in front of the class kept scratching the top of her head in a kind of intentional way. I couldn't figure out for the longest time what was going on. Then at last it dawned on me: every time I did it she did it.

I want to turn next to a narrower topic: How to teach diagnosis. I'm sure you've all seen cases where fully qualified foresters have failed to notice a major disease problem. And we say tut-tut; that's pretty bad. But learning to see is hard work. There is a filter between our eyes and our brain that eliminates things that we don't know or expect. Let me tell you a story. Many years ago we went to visit my brother's place at Christmas time. He was just out, and so we added two decorations to his Christmas tree. One was a little paper airplane, and the other was my shoe. When he came back we asked him about the new decorations. Well, he could see the paper airplane right away, but he could not see the shoe, even though it was in plain view. His filter was saying 'Christmas trees don't have shoes'.

Let me say it again: Learning to see is hard work. Mostly we learn from others; to see something new by yourself takes determined, focused and sustained effort. That is even true for us even if we want to see new disease situations. Let me give you an example from the history of Forest Pathology. All of us would agree, I believe, that *Phellinus weirii* is a major root disease of western conifer forests. We see it everywhere all the time. Now if you pick up the first (1938) edition of John Boyce's standard text 'Forest Pathology', and you turn to the chapter on root diseases, all the old familiar root rots are there: *Fomes annosus*, *Armillaria mellea,* etc. But *Phellinus* is missing. It wasn't until Irene Mounce, Jack Bier and Mildred Nobles (1940) did their work at Cowichan Lake on Vancouver Island that it was recognized that *Poria weirii* was not only a common butt rot of cedar, but also a devastating root rot of many conifers. Up until that time nobody had 'seen' yellow laminated

root rot. The second edition of Boyce's book (1948) makes a mention of the root disease, and by the third edition (1961) it has its proper prominence.

All this has major implications for teaching diagnosis. Modern technology allows us to throw enormous amounts of high quality information at students. PowerPoint is a great tool. But if you think that exposing students to a disease by showing them a good set of high quality ppt's will do the trick, think again. The only way in my experience is to take them out into the real forest and have them do the diagnosis there, again and again. Ppt is great for demonstrating new variations of an old disease to an audience already familiar with the disease, but it just doesn't cut it as a first and only exposure. So teach in the field as much as possible.

But there is more. In diagnosis I use more information than I can say – then I am even explicitly aware of. Take the common case of recognizing an old friend. We can pick him out of thousands without the least difficulty. Yet try to describe in words what makes you recognize him, and what you get is a police sketch, which, as we know, doesn't look like anyone at all! Or: back in Dendrology I learned how to recognize a Douglas-fir. Shiny, pointed brown buds; needles of equal length standing out in all directions; pendant cones with three-pronged bracts extending beyond the cone scales. But now I drive down the highway at 70 miles per hour and recognize Douglas-fir without difficulty, at least in the forest types I'm familiar with. How do I do it? Certainly not by the characteristics I learned in dendrology, and probably something to do with Gestalt, but I'm not really sure. So it is with disease recognition. I watch an old hand like Duncan Morrison. He puts an axe in a stump and pronounces *Armillaria.* He hasn't seen any mycelial fans or basal resinosis, and yet he knows, and he's right. So how do you teach if you can't even say just what you are trying to teach?   Again, go in the field and do it over and over again in front of students, and have them do it, and somehow, magically, they learn. All complex skills have this unspecifiable dimension. To give another example, if you try to follow a

complex technique from a description in a paper, often it doesn't work. You need to go and work for a while in the lab where they do it routinely, and then you learn. Obviously the objective scientific description in the Methods section of a paper doesn't say it all. I would in fact maintain it can't say it all. With lots of work we can decrease this unspecifiable dimension, but I don't believe we can eliminate it completely. Human knowledge cannot be made wholly objective. Teaching is like a Master-Apprentice relationship. It requires a personal relationship and trust and a willingness on the part of the apprentice to imitate the master, for it is in imitating the master that we learn, and that involves more than just listening. And the good Master is careful to expose his own limitations so that the students too can become aware of theirs.

I'm going to end with a difficulty that has become common in the last decade or so. When I went to school, it was generally believed that there was only one truth about the material world, and that science was the way to get at it. Scientists were the priests of western society, and the promised land of plenty for all was just around the corner if only we would listen. Do you remember all the serious talk about what we would do with all our leisure time when the workweek would be reduced to one or two days and all production fully automated? International conferences were brought together to solve this looming problem! Certainly, scientists were regarded as men of authority (still very few women in those days) working for the universal good.

Of course that couldn't last; those promises could not be met. (Though even today such promises abound. Just read the section in almost any research proposal dealing with benefits that will accrue from the proposed work). The result has been skepticism about the benefits of science. And there are good reasons for that skepticism. It's not so much that science doesn't work, but rather that science has promised more than it could possibly deliver, and even more important, that science has become to a great extent the servant of big money industry and the military. Scientists are no longer universally seen as the

disinterested servants of the public good (although many remain just that), but rather as self serving technocrats who have sold their soul for fame and a good income. Should we be surprised? I think not. Just think about how often we hear of 'expert witnesses' at trials contradicting each other. It doesn't seem very difficult for lawyers to find scientifically qualified witnesses to say almost anything they want. Along with the loss of faith in science there is now a widespread skepticism of anyone proclaiming that he has the truth or the way to the truth. Philosophers call this post-modernity. The best description I know is 'a general incredulity of meta-narratives'. (Translated: a general lack of belief that anyone can know the 'whole story', or even that there is a 'whole story').

In the classroom this translates into a situation in which the student simply does not accept what is being taught. It is much the same when you speak to a public audience, as all of us know only too well. Evidence doesn't matter that much. Their opinion is as good as anyone else's. You can see this attitude all around you (particularly here in Sedona). Just look, for example, at the popularity of quack medicine, or the extremes of both the 'greens' and 'browns' in debates about the environment. In the latter especially, the proponents of the extremes have no difficulty in citing any scientific results that support their position and ignoring those that don't. In all these cases, the respect for, and the authority of science (to the extent that it still exists) is co-opted to serve the agenda of some group striving for power and influence. You can draw a close parallel here with the way religious belief has also often been co-opted to serve some political end. In Canada, publicly funded science must now increasingly serve the public needs (translate: the concerns of the party in power). Perhaps this is inevitable with a scientific establishment now largely funded out of the public purse, but I should remind you that for the first several centuries of 'western science' this was not the case. Science was a hobby (or better: a cultural activity) that only the independently wealthy could afford to pursue. (As a 'type specimen' consider Charles Darwin.) Of course it is good to serve the public needs, but they

had better be the needs that the scientist herself recognizes, and, in the best of all possible worlds it is the responsibility of the scientist herself to determine how she can best serve those needs.

How does one deal with this new attitude? I believe that if, as a teacher, you hide behind your expertise, you will increasingly run into this kind of resistance. The best way is to present yourself as a whole person. I spoke earlier of a 'world-grasp'. Of course the teacher has one too. What is now necessary is to reveal that world grasp to the student (or to any audience you wish to convince), to expose your motivations, your doubts, and hopes. Sure that's scary, but strangely it is also liberating. I now start a course by saying that in addition to the official course objectives there is also a secret agenda, one that I had better reveal, namely that I want the students to look at the world the same way I do. And then I speak of my love for and fascination with in the natural world. Of course you can't force this, but it does lead to a greater trust – a necessary condition for true learning. It helps to defuse the suspicion that the real reason for the things I teach is some secret and unsavoury agenda not shared by the student. I believe this has always been the agenda of every great teacher.

This works out in many ways. For instance on field tours, in addition to all the technical stuff, I often make the point that the natural ecosystems and landscapes we are in are not only fascinating to the intellect, but also beautiful places, and I'll point out the ever changing play of light, the sea fogs drifting in and out on a clear, crisp fall day, or the sheer wonder of these giant old organisms reaching for the sky. In fact I go so far as to say that unless you have an appreciation for the beauty of the place you are not fit

to manage it. The effect of such declarations is interesting. Some students will say 'of course, I know that'. Others are surprised that such sentiments have a place in forestry education, and it actually sets them free of supposed constraints. Still others are mystified and a few even angry. Nevertheless it is this kind of opening up as a person that makes you real to the student, and somehow it makes it much easier for them to accept all the rest and to share in your enthusiasm for the subject.

So here are some of the lessons I have learned. Took me a long time, and I'm sure I'm not finished. I thank you for bearing with me for some time, and I commend teaching to you as a most important and rewarding activity.

WIFDWC Outstanding Achievement Award 2006
Alan Kanaskie

Thank you for the honor of your recognition of my leadership and our work on Swiss Needle Cast (SNC) and Sudden Oak Death (SOD). I prefer to immediately deflect your attention to the team of people that I work with in Oregon. Our situation for the last 15 years has allowed us to mount rather remarkable responses to important challenges because of the cadre of extremely capable, collaborative, and enthusiastic people working in our state. We get things done because no one is guarding their sandbox or putting self-interest before the tasks at hand. It seems like everyone does their best to enable the others. In this environment it's easy and usually enjoyable to do good things.

The award was specific to SNC and SOD, so I can mention, without too much fear of omission, the key people whose cooperation, camaraderie, and council I treasure. From Oregon State University: Everett Hansen, Wendy Sutton, Paul Reeser, Jeff Stone, Doug Maguire, Doug Mainwaring, and Dave Shaw. From the USDA Forest Service: Ellen Goheen, Greg Filip, and Dave Bridgewater. From the Oregon Department of Forestry, Michael McWilliams, Dave Overhulser, and Steve Dutton (retired). From the Oregon Department of Agriculture: Nancy Osterbauer and Dan Hilburn. From the private sector: Bill Woosley (retired), Fred Arnold (South Coast Lumber), Mark Gourley (Starker Forests), and John Washburn (Green Diamond Resources, retired). From the USDI-BLM: Walt Kastner and

Rick Shultz. It's a long list and it could be longer, but these are the people that stand out. You might notice that many of these people are not pathologists, and that fact illustrates the importance of involving other disciplines in solutions to disease problems.

To be recognized for achievement as a "practicing forest pathologist" or as Stephan put it, a "Pulaski-carrying pathologist" was indeed a surprise, and was surprisingly meaningful.   When I chose a professional rather than an academic/research path, I did so with the understanding that I probably would not get much recognition, at least not in the ways typical of the research and academic communities. As a youth, peer recognition carried serious weight, positively or negatively.   My parent's comments always were suspect because they were supposed to say that I did well, and sometimes did so when performance clearly didn't warrant it.   My grandmother was more direct and honest, sometimes painfully so, but often she had me confused with another grandkid or an uncle, so I could dismiss her comments if I didn't like them. Peers were different, and even though what they said may have been motivated by self-interest, their comments mattered most, and they still do. So thanks again for the award - it is precious – and I encourage the Award Committee to continue this type of recognition for the Pulaski carriers or other subcultures of Forest Pathologists.

Outstanding Achievement Award 2007
Dr. Richard S. Hunt

Numerous individuals nominated Dr. Rich Hunt this year for the WIFDWC Distinguished Achievement Award. The common thread in all of the nominations attests to Rich being personable and well respected, not just his "exemplary publication record". The nominees talked about the great pleasure they had in nominating Rich for a variety of reasons. They were inspired by his dedication, infected by his passion for his research and extension, and appreciated his mentoring/ role modelling. Some have known him for 20, 30 and even 40 years – always speaking of lasting impressions.

On the serious side, Rich is noted for his "scientific integrity and rigorousness". "His scientific approach has always been to ask the relevant scientific questions and to conduct the appropriate experimentation to answer those questions." Examples of this included his landmark paper documenting that *Cronartium ribicola* was heterothallic, based on carefully conducted series of experiments involving caging cankers to prevent insect spermatization that "demonstrated that aecidia could not be formed without the spermatization from other cankers" (Hunt 1985). Rich had a knack of designing simple and elegant experiments to resolve complex issues.

Rich published on a wide range of topics including operational procedures for blister rust control such as pruning and topping for non-resistant white pine crops, to bringing clarity to complex topics such as the definition of virulence and pathogenicity (Hunt 1994),

to quantitative resistance as the basis for the white pine blister rust breeding program (Hunt 1997). He has contributed ~ 200 journal papers, review articles, and technology transfer publications. He also served for several years as the forest pathology editor for the Canadian Journal of Plant Pathology.

Rich enjoyed working with others. He led a team consisting of a geneticist, and two molecular biologists, and several students. Rich collaborated with forest pathologists, geneticists, foresters in private industry and the BC Forest Service. He generously shared his time and wit with them on countless field trips.

Rich is well known internationally through his active participation in WIFDWC as the Canadian representative on the blister rust committee, and at IUFRO on the Tree Rust Working Party, and as a member of the Canadian Phytopathological Society (CPS). Rich has chaired many local and annual meeting sessions of WIFDWC and CPS. Rich was honoured about 3 years ago by the CPS with the title of Fellow of the Society.

Below is a quote (source not identified) taken from one of the many letters of support for Rich's nomination:

"One of the things I appreciated most about Rich was his wry sense of humour. Rich could bring choruses of laugher for a presentation on the taxonomy of white pine blister rust (WIFDWC Meeting in Bend, OR, 1989). That takes talent and connection to the audience!"

PRE-MEETING SESSION
Forest Diseases and Climate Change in Western Forests: What Do We Know; What Do We Need to Find Out?

Part 1.

Abiotic Diseases and Climate Change
  John Kliejunas, USDA Forest Service, Pacific Southwest Region, Retired

Cankers Diseases and Climate Change
  John Kliejunas, USDA Forest Service, Pacific Southwest Region, Retired

Climate and Forest Declines in Western North America
  Paul Hennon, USDA Forest Service, State and Private Forestry and Pacific Northwest Research Station, Juneau, AK.

Effects of Climate Change on Woody Decay: Heart Rots and Sap Rot
  Jessie Micales Glaeser, USDA Forest Service, Forest Products laboratory, Madison, WI.

Climate and Foliar Diseases
  Jeff Stone, Oregon State University, Corvallis, OR. and Harry Kope, B.C. Ministry of Forests, Victoria; Canada

***Phytophthoras*/Climate/Climate Change**
  Ellen Goheen, USDA Forest Service, Forest Health Protection, Medford, OR.

Root Diseases and Climate Change
  Mee-Sook Kim, USDA Forest Service, Rocky Mountain Research Station, Moscow, ID.

General Cinsiderations, Dwarf Mistletoe and Stem Rusts
  Brian Geils, USDA Forest Service, Rocky Mountain Research Station, Flagstaff, AZ.

Part 2.

Economic and Ecological Impacts of Non-Native Forest Pests and Pathogens in North America
  Juliann Aukema, National Center for Ecological Analysis and Synthesis, Santa Barbara, CA.

Abiotic Diseases and Climate Change
John Kliejunas[1]

General Statements

Proposed climate changes include warmer winter temperatures and more frequent droughts. Environmental extremes are expected to increase (Hopkin and others 2005). Climate change scenarios have predicted an increase in temperature and a decrease in moisture for western forests. One model predicts that the western U.S. gets wetter winters and warmer summers throughout the 21st century (compared to current climate). Under warming climate scenarios, interior western forests would likely have more precipitation, but it would fall mainly in the traditional October-April wet season. Summers would still be hot and dry, and may be even hotter and longer than they are now (USDA Forest Service 2004).

Current Condition

Abiotic, environmental factors affect plant disease in several ways. First, abiotic diseases result from effects of the environment alone directly on the host (disease associated with environmental extremes). Second, factors such as temperature and moisture affect host resistance, the biotic pathogen, and its ability to infect. Third, environmental factors are stress agents in declines. Some examples include:

1) Direct effects of environmental extremes (for example drought, low or high temperatures) are generally negative (Desprez-Loustau and others 2006).

2) Pathogens predisposed by drought can induce very severe damage, as shown for *Sphaeropsis sapinea*; a greater development of symptoms was consistently associated with water stress (Bachi and Peterson 1985, Blodgett and others 1996, Blodgett and others 1997, Paoletti and others 2001).

3) After a healthy tree has been stressed one or more times, its defense systems can become impaired making it vulnerable to attack by insects and diseases (Wargo and Haack, 1991).

Predicted Future

The literature contains numerous predictions of the effect of climate change on abiotic diseases, on host

[1]J. Kliejunas is retired with USDA Forest Service Pacific Southwest Region. kliejunas@comcast.net.

susceptibility and pathogen success, and on declines.

Some examples include:

1) Injury to trees by winter cold may be slightly lower than at present; unseasonable frosts will cause damage; autumn frosts may become more damaging (U.K. Climate Impacts Programme).

2) Abiotic diseases associated with environmental extremes are expected to increase, and interactions between biotic and abiotic agents might represent the most important effects of climate change on plant diseases (Boland and others 2004).

3) Future drought is projected to occur under warmer temperature conditions. Of particular concern is regional-scale mortality of overstory trees, which rapidly alters ecosystem type, associated ecosystem properties, and land surface conditions for decades (Breshears and others 2005).

4) An increased incidence of summer drought would make trees more vulnerable to attack by weak pathogens. Increased incidence of summer drought would favor diseases caused by fungi whose activity is dependent on host stress, particularly root pathogens and latent colonizers of sapwood (Broadmeadow 2002, Lonsdale and Gibbs, 2002).

5) Facultative pathogens, such as *Armillaria* root rot, and wilt diseases, such as *Verticillium* wilt, and those caused by pine wood nematode as well as secondary canker-causing fungi would benefit from the heat and drought stress caused to forest and urban trees (Boland and others 2004, Schoeneweiss 1975)

6) Climate change can be expected to create abnormal stress conditions in forest stands (Columbia Mountains Institute of Applied Ecology 2005).

7) Forests under stress are likely to be further stressed. Forests on marginal sites may deteriorate if climatic changes make conditions less conducive to survival. Forests already stressed by crowding, pathogens, or atmospheric conditions may not survive the additional climatic stress (Winnett 1998).

8) In a changing climate with increased temperatures, evapotranspiration, and extreme weather events, there will be an increase in the frequency and severity of stress factors, which may lead to more frequent forest declines (Sturrock 2007).

9) Climatic extremes accelerate chronic declines. A variety of pests, pathogens and parasites can take advantage of trees stressed by environmental changes (Jurskis 2005).

Canker Diseases and Climate Change
John Kliejunas[1]

General Statements

Climate change could have positive, negative, or no impact on individual plant diseases. (Chakraborty and others 2000). Proposed climate changes include warmer winter temperatures and more frequent droughts. Environmental extremes are expected to increase (Hopkin and others 2005). Climate change scenarios have predicted an increase in temperature and a decrease in moisture for western forests. In scenarios run through one model, the western U.S. gets wetter winters and warmer summers throughout the 21st century (compared to current climate). Under warming climate scenarios, interior western forests would likely have more precipitation, but it would fall mainly in the traditional October-April wet season. Summers would still be hot and dry, and may be even hotter and longer than they are now (USDA Forest Service 2004).

Current Condition

Very few stem canker and dieback pathogens attack vigorous trees. Outbreaks or epidemics of these diseases are usually an indication that the trees have been predisposed by stress, the most common being water stress and freezing stress (Schoeneweiss 1981). Secondary canker-causing fungi are favored by heat and drought stress (Schoeneweiss 1975). Most published studies refer to a positive association between drought and disease, i.e. disease favored by drought, or drought and disease acting synergistically on tree health status, with a predominance of canker/dieback diseases, caused by pathogens like *Botryosphaeria*, *Sphaeropsis, Cytospora* and *Biscognauxia* (*Hypoxylon*) (Desprez-Loustau and others 2006). Some examples include:

1) Although *Botryosphaeria dothidea* is been found on a wide range of host species, it can cause serious damage only to those host plants that are weakened or under environment stress (Ma and others 2001). Drought stress and winter injury have been associated with increased infection and canker expansion of *B.dothidea* (Brown and Hendrix 1981).

2) A greater development of *Sphaeropsis sapinea* symptoms is consistently associated with water stress (Bachi and Peterson 1985, Blodgett and others 1996, Blodgett and others 1997, Paoletti and others 2001). The most severe *Sphaeropsis* shoot blight occurred in the driest year and the least in the wettest year. (Blodgett and others 1997).

3) *Septoria musiva* cankers on inoculated water-stressed trees were significantly larger than those on nonstressed trees (Maxwell and others 1997)

4) The distribution of pitch canker is limited by low temperatures. Inoculation trials during winter yielded low infection rates. In spring and summer trials, wounds inoculated on day zero became infected at a significantly higher rate than those inoculated two days later (Inman and others 2007, unpublished).

5) The disease *Biscogniauxia mediterranea,* a serious problem in oaks in the Mediterranean area, and not previously detected further north than southern Tuscany, is now in Slovenia (Jurc and Ogris 2006).

Predicted Future

Based on published research and predicted changes in climate, the literature contains numerous conclusions concerning the effect of climate change on canker diseases, including:

1) Most secondary canker-causing fungi would benefit from the heat and drought stress caused to forest and urban trees (Boland and others 2004, Schoeneweiss 1975).

2) An increased incidence of summer drought would favor diseases caused by fungi whose activity is dependent on host stress, particularly canker fungi (Broadmeadow 2002, Lonsdale and Gibbs 2002).

3) Incidence of *Sphaeropsis sapinea* on pines would increase as drought increases. Some other canker diseases, such as *Thyronectria* canker of honeylocust and *Cryphonectria cubensis* on Eucalyptus would decrease (Despres-Loustau and others 2006).

4) Climate change could lead to outbreaks of *Biscogniauxia mediterranea* further north than current distribution (Jurc and Ogris 2006).

5) The pitch canker pathogen, now limited by environmental conditions, may find favorable conditions in the Sierra Nevada as milder winter minimum temperatures occur (Battles and others 2006).

Climate and Forest Declines in Western North America
Paul Hennon[1]

Factors	Ohia Decline	Pole Blight	Cedar Decline
Tree species	*Ohia lehua*	Western white pine	Yellow-cedar
Disturbance/ Age structure	Volcanic flow/ even-age	Fire, harvest/ Even-age	Peatland succession/ multi-age
Onset	1954 (also in 1906)	1929	1880-1900
Vulnerable age	Old, "senescence"	Young, "Pole stage"	Older, "overstory"
Soil Factor	Poorly drained	Low moisture holding capacity	Poorly drained
Stand structure	Open canopy	Dense, closed canopy	Open canopy
1° biotic factors	Is *Phytophthora cinnamomi* 1° or 2°?	-	-
2° biotic factors	*Plagithmysus, Armillaria, Pythium, Endothia*	*Leptographium*	*Armillaria, Phloeosinus*
Proximate injury	Rootlet mortality	Root desiccation	Rootlet freezing
Climate	Minimal influence	Drought (1916-1940)	Warmer winters, reduced snow, early spring freezing
Outcome/management	Mortality progressive / No management	Resolved with end of drought, introduction of blister rust	100 y progressive mortality/ salvage, favor cedar in deep soils, deep snow
Lesson for climate:	Climate was a minor factor	Good climate lesson	Good climate lesson

For more information see Hennon, Paul E. "Pathology without Pathogens: Opportunities for Forest Pathologists in a Changing Climate" this volume.

Effect of Climate Change on Wood Decay – Heart Rot and Sap Rot
Jessie A. Micales Glaeser[1]

The decomposition of coarse woody debris has been of interest to carbon cycle and climate change modelers for the past decade, but only a few studies have looked at the possible influence of climate change on the amount of wood decay in both living trees and "dead" woody debris. Both decay processes are intimately dependent on moisture and temperature regimes and thus could be heavily impacted by future climate change.

There are many hundreds of species of sap rot fungi that saprophytically degrade downed wood, recycling nutrients back into the soil. Many of these fungi are not host specific so there is a wide array of decay fungi available to colonize a specific substrate under a given set of conditions. Wood moisture content is the most important factor for decay and is dependent on humidity, precipitation, and uptake from ground contact. Decay fungi cannot degrade wood with moisture contents below 30% (fiber saturation point), and the upper moisture limits generally do not exceed 90%, due to lack of oxygen. Wood temperature is the second most important factor for fungal activity. Fungi can generally begin growth as soon as wood temperature exceeds 0°C, the temperature at which free water in the wood cell wall is available to the fungus. The optimum temperature for growth is highly dependent on the fungal species; most decay fungi have temperature optima between 20 – 35 °C but some can be even higher. For example, *Gloeophyllum sepiarium* and *Phlebia subserialis* can colonize case-hardened logs and the upper portions of slash piles under conditions of high temperatures and relatively low moisture. Modeling studies that have looked at the decomposition of woody debris have

concluded that an increase in regional warming and drying in the west would increase rates of decay, leaving less debris on the forest floor and resulting in increased release of carbon dioxide, except when limited by extremely xeric conditions.

"Heart rot" fungi are pathogens that colonize the central portion of living trees. These fungi are much more specific in their host requirements and often cannot continue growth after the death of the tree. Trees can often limit the amount of fungal colonization by mounting active chemical and physical barriers, but trees under stress, particularly drought stress; have fewer reserves available for host defense. Drought conditions can also result in the formation of radial cracks in conifers, which can serve as infection courts to heart rot fungi. At the same time, low humidity and dry conditions make the wound surface less favorable for spore germination, resulting in fewer established infections. Heart rot fungi can persist in dried wood for many years so colonization can resume under more favorable conditions.

Therefore it appears that the influence of changing climate will have a substantial influence on the development of wood decay, especially in the decomposition of downed wood and slash. More research is needed to determine climatic effects on individual heart rot diseases.

Climate and Foliar Diseases
Jeffrey Stone[1] and Harry Kope[2]

The implications of climate change for specific foliar diseases of forest trees are difficult to predict. This is because there is a fine balance between these disease-causing organisms, their tree hosts and their environment. Few foliage pathogens have a broad host range, so predicting effects of climate change on disease involves individual pathogen-host associations. Yet there are far too many foliar pathogen species to consider each adequately. Furthermore, there is very little detailed epidemiological information on specific climate factors influencing distribution and severity for most foliage pathogens. Although there are numerous anecdotal examples of foliage disease outbreaks related to anomalous weather, there are only a few foliage diseases for which epidemiological models have been produced. Some of the best-studied forest foliage diseases with respect to climate/weather are *Dothistroma* needle blight of lodgepole pine, Swiss needle cast of Douglas-fir, and sudden oak death.

It is possible, however, to make some predictions about relationships between climatic factors and foliage diseases in general. The majority of conifer foliage pathogens sporulate and infect new foliage in the spring and early summer, most have spores that are dispersed in rain/mist, and most require high relative humidity (RH) or free moisture for spore germination and initial infection to occur. Because of this, foliage diseases typically vary in severity from year to year depending on annual weather and the duration of warm, wet periods during the sporulation/infection period in the spring. In much of the Pacific Northwest USA, for example, a period of mild temperatures and relatively constant high RH in spring is typically succeeded by a prolonged summer dry period that is much less favorable to spore dispersal in infection. A general prediction, therefore, is that climate trends that extend the duration or amount of spring precipitation will lead to increased foliage disease severity in western forests, and conversely, trends leading to increased aridity will diminish foliage diseases.

A second general prediction can be inferred from the life cycles of the foliage pathogens. Some species, like *Phaeocryptopus gaeumannii*, the Swiss needle cast pathogen, and *Rhabdocline pseudotsugae*, one of the *Rhabdocline* needle cast pathogens, reproduce only sexually and have a determinate sporulation period. Because they sporulate only once per year, disease severity caused by these pathogens tends to integrate long-term climate trends. Others, like *Mycosphaerella pini*, the red band needle blight pathogen (also called *Dothistroma* needle blight), *Mycosphaerella dearnessii*, the brown spot pathogen, and *Rhabdocline weirii*, have both a sexual and an asexual reproductive state. The asexual reproductive state often can be induced rapidly and continue indefinitely under favorable conditions. These fungi can rapidly amplify under intermittent, periods of precipitation and favorable temperatures at any time during the growing season. Severe disease can appear abruptly in a single growing season. Therefore, climatic change that results in later season precipitation, increased intermittent summer precipitation, and greater variation in annual precipitation are likely to favor an increase in diseases caused by asexually reproducing pathogens.

Finally, novel or emerging pathogens will likely become more damaging as a result of climate change and forest management. There are numerous species of foliage-parasitic fungi, the majority of which have long been considered insignificant with respect to disease impacts. However, under changing climate conditions, the adverse impacts of some of these fungi will probably increase. Two of the better-studied examples of foliage diseases whose impacts have increased substantially during the past decade, at least in part due to climate influences, are red-band needle blight (*Dothistroma* needle blight) in northwestern British Columbia, and Swiss needle cast of Douglas-fir in western Oregon. These two diseases are caused by endemic pathogens that previously had been considered to cause only negligible impacts to forest health. Climatic change alters the balance between host, pathogen, and environment in unpredictable ways. Human management of forests adds another level of complexity to the mix. It is therefore likely that undescribed species, or pathogens currently considered of negligible importance, will find increased opportunities under changing climate conditions.

[1]J. Stone is with Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR.
stonej@science.oregonstate.edu.
[2]H. Kope is with Forest Practices Branch, British Columbia Ministry of Forests and Range, Victoria, British Columbia, Canada. harry.kope@gov.bc.ca.

Phytophthoras/Climate/Climate Change
Ellen Goheen[1]

Phytophthora ("plant killer") is a genus that is parasitic on a wide variety of host plants, with species acting as fruit, foliage, stem, crown, or root pathogens, or some combination thereof. While some *Phytophthora* species are quite host specific, others have rather broad host ranges. *Phytophthora* species have the capacity for a very short regeneration time because of the rapid production of flagellate zoospores. They produce resting spores which can survive for years in leaf litter and organic matter in the soil. They also reproduce sexually facilitating genetic recombination. The mixing of *Phytophthora* species in artificial environments has facilitated the production of hybrid species that are pathogens of very different hosts from those affected by the species contributing to the hybrid.

Phytophthora owes its success to its ability to increase inoculum levels from low, often undetectable levels to high levels within a few days or weeks due to the rapid production of sporangia and zoospores when environmental conditions are favorable. Temperature controls on *Phytophthora* vary widely among *Phytophthora* species, however, most *Phytophthoras* are considered to do best under relatively mild to warm conditions. Moisture, on the other hand, including rainfall, dew deposition, and irrigation, is the main environmental factor that controls the pathogen. The multi-cyclic nature of *Phytophthora*-caused plant diseases can and has resulted in severe epidemics in forest and agricultural systems worldwide.

Two well-studied *Phytophthora* species can be used in the "model system" concept to describe what is known about *Phytophthoras*, climatic conditions conducive to disease, and climate change. *Phytophthora infestans* is an infamous disease of potatoes and tomatoes that is aerially spread, affects leaves stems and tubers, and is favored by moist atmospheric conditions. It has been tracked and modeled extensively arount the world.

[1]Ellen Goheen is with the USDA Forest Service, Forest Health Protection, Medford, OR. egoheen@fs.fed.us.

Disease forecasting models use factors including hours and days of leaf wetness, relative humidity, and temperature to predict risk. Climate change-related modeling of *P. infestans* where potato growing regions are trending towards warmer and wetter growing seasons (based on historical climate data) indicate an increased risk of potato late blight, including extending the time frame for diease risk into earlier summer months than previously observed.

Phytophthora cinnamomi, of great importance in forest and woodland pathology, is well known as a devastating root pathogen now encountered worldwide with over 900 species on its host list. The pathogen is readily moved in free-flowing water, soil water, and organic matter. Risk mapping for diseases caused by *P. cinnamomi* has been based on factors such as winter survival potential of the pathogen and soil type (usually focusing on soil drainage characteristics). Recent climate change modeling efforts in Europe, for example, indicate that increases in winter temperatures of 0.5 to 1.5 ˚C allow for increased survival of *P. cinnamomi* propagules. Investigators suggest that the result would be increased impacts where the disease currently occurs and potential range expansion northward and eastward from the Atlantic coast.

Western Forest Diseases and Climate Relations: Root Diseases and Climate Change

Mee-Sook Kim[1], Bryce A. Richardson[2] and Ned B. Klopfenstein[3]

Climate change could alter patterns of disturbances from pathogens through (1) direct effects on the development, survival, reproduction, dispersal, and distribution of pathogens; (2) physiological changes in tree defenses; (3) indirect effects from changes in the abundance of mutualists and competitors.

In general, any climate change could increase the incidence and spread of root disease if host trees become maladapted and undergo stress due to climate change. In addition, climate change could alter fitness of various mycorrhizal fungi and other beneficial microbes that currently suppress root disease.

Some reports suggested that hot and dry conditions (e.g., prolonged drought) are expected to increase incidence and spread of root diseases in forests. However, it is difficult to specifically predict how this climate change will affect diverse root diseases under various projected climate scenarios. Currently, the distribution of pathogens that cause root disease in the western USA is not well documented. Current disease surveys often overlook non-symptomatic trees that are infected by pathogens.

[1]USDA, Forest Service, Rocky Mountain Research Station, Moscow, ID. mkim@fs.fed.us.
[2]USDA, Forest Service, Rocky Mountain Research Station, Moscow, ID. brichardson02@fs.fed.us.
[3]USDA, Forest Service, Rocky Mountain Research Station, Moscow, ID. nklopfenstein@fs.fed.us.

Furthermore, precise diagnostic tests are seldom performed to accurately identify pathogens, and precise GPS-recorded data about pathogen distribution is frequently lacking. Without reliable and accurate information of pathogen distribution under present climate conditions, it is difficult to assess the relationships among root-disease pathogens and climate variables. Determining the precise distribution of currently existing pathogens and disease is the first step toward understanding impacts of climate change on root pathogens.

An example of predicting *Armillaria ostoyae* based on climate variables will be discussed at the workshop. A short description of this pilot project follows: To develop a climatic envelope for *A. ostoyae*, latitude, longitude and elevation were compiled from 102 confirmed locations where this species was found. These location data were used to develop climate-variable estimates from a spline-climate model at 1 km2 resolution followed by Random Forests multiple-regression tree analyses.

Currently available climate models that predict suitable climate space for forest tree/shrub/forb species based on various climate change scenarios will also provide a basis to determine climate effects on host vigor and pathogen distribution in the western USA. These approaches will be discussed at the workshop.
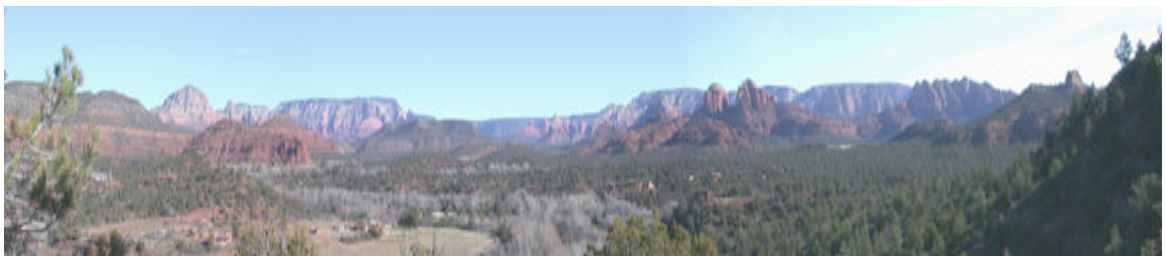
Panoramic view of the immediate Sedona area, covering an angle of view of approximately 90 degrees. The line of bare (deciduous) trees at center left marks the course of Oak Creek. From Wikipedia.org. October 2008.
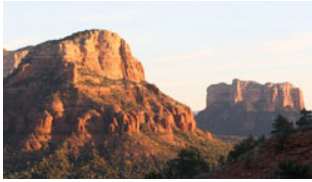
Western Forest Diseases and Climate Relations: General Considerations, Dwarf Mistletoe and Stem Rusts
B. W. Geils[1]

Abstract

This is a preliminary, draft outline for organizing information on the relation of climate to western forest diseases. The question is how to assess the threat of these diseases under a regime of climate change. Although forest diseases are often important, assessment of disease–climate relations is a challenging problem due to the multiple values at risk and the complexity of these systems. Several approaches are described and illustrated with examples for mistletoes and stem rust. Only a sampling of examples is given to illustrate a variety of concepts; many additional examples are to be found in the literature. Knowledge gaps are identified.

I. FOREST DISEASE–CLIMATE RELATIONS ARE IMPORTANT BUT CONFOUNDING

A. At times and places, forest diseases are important forest ecosystem drivers.

B. The hosts, pathogens, and associated organisms have complex relations with the atmospheric environment represented by climate and weather.
1. Climate refers to averages and regimes…
2. Extreme weather events (rare or occasional) are very influential.
3. Primary factors for consideration of forest pathogens are heat and moisture.
4. Secondary factors include carbon dioxide concentration, ozone, radiation, etc.
5. Direct effects influence the epidemiology (dispersal and survival) of the pathogen, e.g., high temperature lethal to pathogen spores during dispersal.
6. Some indirect effects are mediated by host, e.g., on host physiology with changes in balance of growth and defense.
7. Other indirect effects are mediated by associated organism, e.g., changes in the abundance of vectors natural enemies, etc
8. Effects and consequences may be positive or negative and with or without feedback
9. Expression may be reduction in disease or emergence of latent pathogens (shift to pathogenic behavior).

C. Forest diseases are classified as abiotic or biotic.
1. Abiotic diseases are often decline syndromes with multiple and complex predisposition and inciting factors.
2. Biotic diseases involve a host and pathogen in a conducive environment.
   a. Vectors, symbionts, or collateral species usually also associated or affected.
   b. Host and pathogen have each their own genetics and distribution (constraints, metapopulation, interaction networks).

II. VALUES AT RISK ARE ECOLOGICAL, SOCIAL AND ECONOMIC

A. Impacts range from direct, rapid and obvious to indirect, cumulative, subtitle, and cascading.

B. Quantifiable measures of more direct impacts include estimates of disease losses, of management/control costs, and area at risk.

C. Effects of more indirect nature include altered fire regime, nutrient cycling, hydrology, and energy budgets and changes in abundance or survival of native species and their genetic integrity.

III. FOREST DISEASE–CLIMATE RELATIONS ARE EMBEDDED IN COMPLEX SYSTEMS

Paradigm determines what is considered relevant and how study is conducted. Older observations and literature may be useful but need careful re-interpretation and re-phrasing to be relevant for the present question.

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1. 20th Century paradigms pervade much of the older literature.
    a. Climatic, climax vegetation and plant associations are seen as important; disturbance is considered a transient deviation.
    b. The historic range of variability assumed to provide an adequate reference frame.
    c. Evolutionary time is thought to run slower than ecological time.
    d. The forest pathology perspective is that weakened, understory trees and poorly managed forests deteriorate and decline over time because of numerous insults from biotic and abiotic agents.
    e. The defined purpose of forest pathology is to reduce immediate economic losses.
2. 21st Century paradigms appear infrequently in older literature and more commonly in recent literature.
    a. Disturbance and recovery is seen as pervasive; long persisting associations are rare.
    b. Rapid change and regime shifts are expected.
    c. Especially at population levels which become a greater focus genetic/phenotypic change is seen over generational times and as responses to ecological events.
    d. The forest pathology perspective is for declines and disease to function in reference to stabilizing selection—healthy dominant trees in the forest (the survivors) are selectively killed by a combination of specifically ordered factors.
    e. The contribution of forest pathology is for sustaining desirable ecosystems.

B. Complex systems are characterized by nonlinear behavior and hierarchical structure.
    1. Criticality provides a potential link between spatial pattern and phase shift.
    2. Concepts of scale and hierarchy give a framework for describing complex systems in which different processes dominate at different scales.
        a. Scale topics include: translation from regional climate to a topographic mesoclimate, mapping spatial gradients and quantifying temporal rates of change.
        b. Hierarchical topics help explain cross-scale interactions as fast, bottom-up mechanisms and slow, top-down constraints.
    3. Feedbacks refer to connections which amplify or dampen dynamics.
    4. Emergent properties/synergism are not predicted because the whole acts as more than sum of parts.

C. The study of forest disease–climate relations for assessing potential direct and indirect effects on future ecosystems benefits complexity science.
    1. Only broad trends may be foreseeable.
        a. ID of specific, controlling factor and consequent, predictable response is unlikely
        b. Plural approaches and perspectives are needed.
    2. Species respond individually; network analysis may prove useful.
    3. Present communities are transient; a biogeography viewpoint of system states (distribution and abundance) and dynamics processes (migration, extinction, and evolution) is practical.
    4. Adaptive plasticity and systemic-induced resistance may affect host balance between growth and defense.
    5. Novel abiotic–biotic interactions under climate change are to be expected.
    6. Disturbance induces heterogeneity at multiple scales.
    7. Identifying alternative states (desirable or undesirable) and vulnerability to regime shift are goals.
    8. Biodiversity functions in ecosystem persistence.
    9. Slow, higher-level, larger-scale ecosystem processes constrain dynamics at faster, smaller-scale, lower levels.
    10. A focus on re-organization after disturbance/shift should consider:
        a. model action (disease type)
        b. pathogen virulence, aggressiveness, and host range
        c. host importance, uniqueness, and phytosociology.

IV. MULTIPLE APPROACHES ARE AVAILABLE AS OBSERVATION SOURCES AND INSPIRATION FOR HYPOTHESES.

A. Paleoecology provides an alternative, temporal perspective of different climates and communities.
B. Biogeography and invasive biology provide spatial perspectives on ecological and evolutionary processes

with emphasis on population, species, and community levels.

1. Invasive biology is a model for emergent diseases (new climate like a new habitat); both describe series of steps (dispersal, establishment, lag, spread, replacement/assimilation).
   a. There does appear to be a link between life history traits and invasiveness (statistical evidence in support).
   b. The importance of phenotypic plasticity, habitat difference (rate of change), and genetic variation are mostly unknown at this time.
   c. Lag phase processes involve adaptive evolution or sorting of adaptive genotypes.
   d. Dispersal and reproduction determine spread, persistence, and abundance of invasive/emergent.
   e. Community dynamics after emergence (impacts, feedbacks) determine the consequences.
C. Fundamental biology as epidemiology, ecophysiology, phytopathology, genetics, and other disciplines provide a mechanistic perspective with emphasis on life histories and pathogen–host interactions.
   1. Life histories, pathogen–host interactions, and ecology are core subjects.
   2. Information on variability and constraint (e.g., optimal and tolerable temperature ranges) are core data.
D. Synthesis of information involves the organization and presentation of information for addressing specific issues.
   1. Climate mapping is useful but requires proper interpretation.
   2. Modeling is very useful but has limitations. For example, caution in predicting forest dieback from growth models arises from several shortcomings.
      a. Tree growth is based on the realized rather than the fundamental niche.
      b. Life spans of trees in the models are too low.
      c. Vegetative reproduction is not incorporated.
      d. Tolerance of climatic fluctuations is given inadequate attention.
      e. The unique niche of fire-tolerant species is not considered.
      f. The protected position of species growing under unique edaphic conditions is not included.

V. DWARF MISTLETOES (*ARCEUTHOBIUM*) ARE DAMAGING AERIAL PARASITES OF CONIFERS.

A. The paleoecology record of pollen and microfossils of mistletoes is long and relatively rich.
   1. The presence of lodgepole pine dwarf mistletoe is a as mean annual temperature marker.
   2. Mistletoe distributions have changed over eons.
B. The biogeography of mistletoes is relative well documented.
   1. An example from historical biogeography is the speculation that favorable climate in Europe for mistletoe contributed to its increase and destruction of European forest
   2. Mistletoe elevational distribution more restricted than that of host
   3. Ponderosa pine mistletoe is absent in Black Hills but host is susceptible.
   4. On South Rim of Grand Canyon, distribution of southwestern dwarf mistletoe near the rim is due to either climate gradient or recent introduction history.
   5. The distributions and associations with site and stand factors is known for many species.
      a. Inferences are made that direct climate effects, host condition, and mistletoe capability are responsible
      b. More southwestern dwarf mistletoe is observed on ridges than slopes than bottoms but no difference observed in same study for Douglas-fir dwarf mistletoe.
C. There is a large body of science on the fundamental biology of mistletoes.
   1. Information is weakest in genetics but there are some relevant observations.
      a. By inference from contrasts of pinyon-juniper systems on different soils, stressed-environments may provide a reserve of more stress-tolerant genotypes
      b. Beyond species immunity, little known of differential, resistance with a genetic basis (not a gene-for-gene system).
   2. Life history focuses on epidemiology and ecophysiology.
      a. Mistletoes are perennial, long-lived, slow reproducing with moderately abundant seeds and short dispersal distance.

      b. Response to temperature varies by species; examples are
        i. extreme cold lethal, ice-nucleation/undercooling
        ii. dormancy and chemical inhibitor on germination
        iii. shoot growth related to temperature (+).
      c. Rain/snow effect seeds in multiple ways, such as
        i. rain moves seeds to safe sites
        ii. heavy storms can remove seeds (not protected under snow).
      d. Light effects mistletoe seed germination and shoot production.
      e. Greater host vigor can result in great mistletoe shoot growth and reproduction..
      f. High C02 may inhibit germination.
      g. Pollination is by entomophily and anemophily, therefore
        i. cold and wet may reduce insect visitation
        ii. but is reproduction pollen limited?
    3. Host-pathogen interactions determine disease progress.
      a. Rapid height growth and physiological vigor of host affects mistletoe increase (complex +/- feedback).
      b. Severe mistletoe infestation increases host vulnerability to drought, bark beetles, and other diseases but killing host eliminates the infection.
      c. Host density and non-host interception affect spread.
      d. Historical increase in seral species may have promoted mistletoe abundance and distribution.
      e. Host-mistletoe water relations are key and well studied.
      f. Mistletoe-infected trees may have reduced root systems.
    4. Ecology and community interactions influence disease and impacts.
      a. There are strong, bi-directional fire-mistletoe interactions.
      b. The linkage with bark beetles varies by host-beetle combination.
      c. Stem canker fungi, foliage herbivores, mycorrhiza, and various tritrophic interactions also involved.
      d. The relation of mistletoe infection and severity with drought is often cited.
  D. Both distribution and forest-stand simulation models have been developed for many mistletoe species.
    1. The Mark-Hawksworth model for southwestern dwarf mistletoe is based on climate.
    2. The Robinson-Geils epidemiological model is linked to FVS and TASS.
    3. Various distribution/severity models are based on plant association.

VI. STEM RUST OF PINE (*CRONARTIUM*) ARE AERIALLY-DISPERSED FUNGAL PATHOGENS.

  A. The fossil record for fungi is meager but phylogeography promises to provide a long-term, Evolutionary perspective.
  B. The biogeography of stem rusts is relatively well studied but could benefit from additional synthesis.
    1. The lack of apparent climate signal for white pine blister rust in BC may indicate it's all-favorable there.
    2. The spread of white pine blister rust into regions formerly considered unfavorable indicates either
      a. greater adaptability than recognized b. more microsites favorable that thought?
    3. Yellowstone region was described as too dry and too cold for severe white pine blister rust.
    4. Observed reduction in white pine blister rust near smelter indicate flume gases may affect rust directly or through effect on needle retention.
    5. Provenance and screening tests reveal host population differences.
    6. Several years of extreme cold purged ponderosa pine population of comandra blister rust at Mink Creek, Idaho.
  C. There is a large body of science on the fundamental biology of stem rusts.
    1. Information is moderately extensive in genetics.
      a. Selection and resistance are related.
      b. There is a concern that selection for rust resistance resulted in decrease in cold hardiness.
    2. Life history focuses on epidemiology and ecophysiology.
      a. Stem rust life cycle includes alternating between annual host and perennial host. Although stem rust

are not especially long-lived, potentially, populations an expand rapidly with large number of spores dispersed at scales from meters to mega-meters.

 b. Strong environmental effects on rate of development (spore development, dispersal, germination) are due to effects of
  i. temperature
  ii. humidity
  iii. air flow (local and synoptic)
  iv. as well studied in western gall rust, comandra blister rust, white pine blister rust.
 c. Wave year phenomena demonstrates importance of and sensitivity due to weather, especially that:
  i. regional climate and annual/monthly statistics may not be sufficient
  ii. hot and dry weather may decrease infection by effect on rust and on defoliation of alternate host
 d. Loss of uredinial stage for stalactiform blister rust observed in dry regions where spread on repeating host is not climatically favorable.

3. Host-pathogen interactions determine disease progress.
 a. Infection is through open stomates so whatever affects stomates affects infection.
 b. Foliage susceptibility differs over time and age.

4. Ecology and community interactions influence disease and impacts.
 a. Western gall rust and terminal weevil are associated.
 b. Spermatization is insect mediated.
 c. Various insects and fungi are associated with cankers and host necrosis, but their importance is variable and disputed.
 d. Rust trees may provide habitat for non-outbreak bark beetles.
 e. The spatial connection of alternate host populations is important.
 f. Nearby sites may have very different microclimates.
 g. Comandra blister rust outbreak was postulated as result of increase in comandra not change in climate, but this assertion has also been refuted.
 h. Lodgepole pine severely cankered by comandra blister rust have lower sugar levels than non-cankered trees which may be related to susceptibility to mountain pine beetle.

D. Hazard models and epidemiological simulation models are developed for several stem rusts.
1. Stalactiform blister rust distribution is predicted from elevation/habitat type.
2. Western gall rust incidence is related to topographic position and elevation.
3. Comandra blister rust is related to weather and site factors.
4. White pine blister rust predicted for many regions, scales, approaches. For example, the McDonald model is used to assess differences in epidemiology under contrasting climates, northern Idaho and southern California.
5. The TASS model used to assess volume loss of rusts of lodgepole pine.

VII. KNOWLEDGE GAPS AFFECT THE ABILITY OF SCIENCE-BASED MANAGEMENT TO ASSESS AND MITIGATE THREATS.

A. The nature of threat assessment and mitigation of forest diseases and their changes under future climates require adaptive ecosystem management, element of which are:
1. Integration, communication, and uncertain decision-making in multiple, biased environments
2. Long-term, ecosystem-wide strategy rather than a tactical approach focused on battling individual, emerging disease
3. Anticipating interactions between emerging diseases and other global change processes
4. Distributed information systems that deliver information on risks, identification, and response strategies.

B. Multiple perspectives are needed from global to site-specific scales, including:
1. Global assessment methodologies
2. Integrating invasive species/emergent diseases into sustainable forestry frameworks such as the Montreal Process and forest certification programs
3. Developing suitable approaches for ecosystem and landscape management.

C. Surveillance and monitoring are key elements for effective control that would benefit from improved technologies, especially:
   1. Baseline conditions (taxonomic-genetic identification and georeference)
   2. Advancing technologies for molecular identification, expert systems, and remote sensing
   3. Improved cost estimates to inform choices about international trade and pest suppression efforts.
D. Several examples illustrate specific needs and concerns.
   1. Whether assisted migration is required or prudent depends on:
      a. Estimation and monitoring of species distributions
      b. Biogeographical modeling
      c. Community interactions
      d. Long-distance dispersal
      e. Genetic diversity.
   2. BC is very concerned over potential maladaptation in forest tree planting using seed zone and standards that no longer match the current environment.

Economic and Ecological Impacts of Non-Native Forest Pests and Pathogens in North America
Juliann Aukema[1]

Project Overview

The ecological effects of some non-native forest pests and pathogens in North America have been well documented. The economic costs of these effects, however, have not been estimated credibly. Moreover, few studies have examined coupled interactions among pests, their hosts, and climate. Climate science has informed ecology, but rarely has communication been bidirectional. The goal of this project is to synthesize ecological data on forest invaders and conduct complementary economic analyses. We will examine the extent to which current knowledge allows credible prediction of the effects of emerging pests and pathogens. Integration of ecological and economic data will lead to development of least cost and greatest benefit approaches that can be implemented by practitioners. Here we provide an overview of several primary activities being conducted under the project umbrella.

Working Group I

Non-native forest pests and pathogens arrive in the United States from numerous other countries and through many different pathways. This working group is compiling a list of pests and pathogens by presumed pathways of introduction. The pests will be categorized according to their economic impact on the basis of criteria such as geographic area occupied, hosts infested, and demographic rates. Using this database, the group is examining rates of detection and economic damages of pests and pathogens according to three major pathways of introduction—nursery, solid wood packing, and hitchhiking. They will extrapolate historical rates of detection to estimate future rates of introduction into the United States. Further, the group is conducting a detailed economic assessment for one or two high-impact species for each pathway. Finally, the group will explore alternative policy options or management strategies that may reduce future economic impacts of forest pests and pathogens.

[1]J. Aukema is from the National Center for Ecological Analysis and Synthesis, Santa Barbars CA. aukem@nceas.ucsb.edu.

Working Group II

This group will address the potential response of non-native forest pests and pathogens to climate change and estimate associated ecological and economic impacts. The group will explore physical and biological relationships among pests, their hosts, and the lower atmosphere that may affect the distribution and virulence of pests as climate changes. On a regional scale, feedbacks may exacerbate or alleviate the effects of climate change on the pest. Participants may examine how rates of arrival or establishment might change, identify regions that may experience changes in resilience to invasion, and identify regions that may become sources of immigrants. Participants may use example pest species or ecosystems to model potential changes in pest and host ranges, physiological responses to climate variables, interactions with other disturbances such as fire, and feedbacks between tree damage (e.g., defoliation) and regional climate. Complementary economic analyses can follow from scenarios of pest introductions, distributions, and damages.

Distributed Graduate Seminar

Distributed graduate seminars are designed to allow multiple universities to conduct research on the same topic simultaneously, typically with data from their geographic region. We are conducting a distributed graduate seminar to determine the extent to which ecological and economic impacts of non-native forest pests and pathogens can be quantified. The seminar involves groups of students at seven universities: Northern Arizona, Colorado State, Minnesota, Montana, State University of New York, North Carolina State, and Oregon State. Each university-based seminar has two leaders, an ecologist and an economist. Each university has selected one to several forest pests or pathogens of local to regional concern. They are gathering key data and evaluating ecological and economic impacts. We will bring together representatives from each university for a comparison and synthesis of results, modeling approaches, and data gaps.

Contact. For more information, please contact Erica Fleishman, National Center for Ecological Analysis and Synthesis, fleishman@nceas.ucsb.edu, (805) 892-2530. Key Research Personnel Juliann Aukema, and Tom O'Halloran, o'halloran@ nceas.ucsb.edu, The Nature Conservancy supports this project.

Fuel and Stand Characteristics in P. Pine Infested With Mountain Pine Beetle, Ips Beetle, and Southwestern Dwarf Mistletoe in Colorado's Northern Front Range

Jennifer G. Klutsch[1], Russell D. Beam[1], William R. Jacobi[1] and Jose F. Negrón[2]

In the ponderosa pine forests of the northern Front Range of Colorado, downed woody debris amounts, fuel arrangement, and stand characteristics were assessed in areas infested with southwestern dwarf mistletoe (*Arceuthobium vaginatum subsp. cryptopodum*), mountain pine beetle (*Dendroctonus ponderosae*) and Ips spp. One hundred fifty plots, each 0.04 ha, were measured and an ANCOVA and Tukey's Multiple Comparison Procedure were used to assess differences in plots with high infestation level compared to plots with trees not infested. Dwarf mistletoe contributed more to the fuel arrangement than to fuel amounts, with the amount of 1, 10, and 1000 hr time lag fuel classes not being different in plots with dmr > 4 as compared to plots with dmr < 4. Height to bottom of crowns was lower and percent live crown was greater in plots with dmr > 4 as compared to plots with dmr < 4. Bark beetles selectively infested large trees in plots without the presence of dwarf mistletoe (p=0.09), while the distribution of trees killed by bark beetles was equal across diameter size classes in plots with dwarf mistletoe and bark beetles (p=0.84). Bark beetles contributed to differences in the amount of fuel and fuel arrangement.
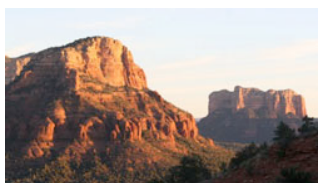
Condition of Soils and Vegetation Along Non-Paved Roads Treated With Magnesium Chloride (MgCl2) Based Dust Suppression Products

Betsy A. Goodrich[1], William R. Jacobi[2] and Ronda D. Koski[3]

This study investigated roadside vegetation and soil conditions related to the use of magnesium chloride (MgCl2) dust suppression products on non-paved roads. Sixty roadside and 79 drainage vegetation health plots were established on 15 and 19 roads, respectively, with a range of MgCl2 application rates. Evaluations were completed of foliar damage, tree health, biotic and abiotic damage agents, soil and foliar chemistry and other common stand characteristics of lodgepole pine, trembling aspen, Engelmann spruce and subalpine fir, and lower elevation plots dominated by shrubs and grasses. High concentrations of foliar and soil magnesium and chloride were measured along straight road segments in the first 3 to 6.1 meters adjacent to treated roads, along with increased foliar damage not apparent along control roads. High concentrations of both ions and subsequent foliar damage were measured in drainages between 24 and 98 meters from the road. Foliar chloride concentrations correlated consistently and positively with percent foliar damage in all species ($r = 0.50 - 0.80$, $p < 0.0001$), whereas the incidence of biotic damages did not correlate with foliar damage. Positive relationships between foliar chloride and application rates were strong and thus MgCl2 application amount can be used to predict foliar concentrations and subsequent damage to roadside trees.

[1]Betsy Goodrich is a Graduate student in Ecology and the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO. betsy.goodrich@colostate.edu.

[2]William Jacobi is a professor of plant pathology in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO. william.jacobi@colostate.edu.
[3]Ronda Koski is a Research Associate in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO.
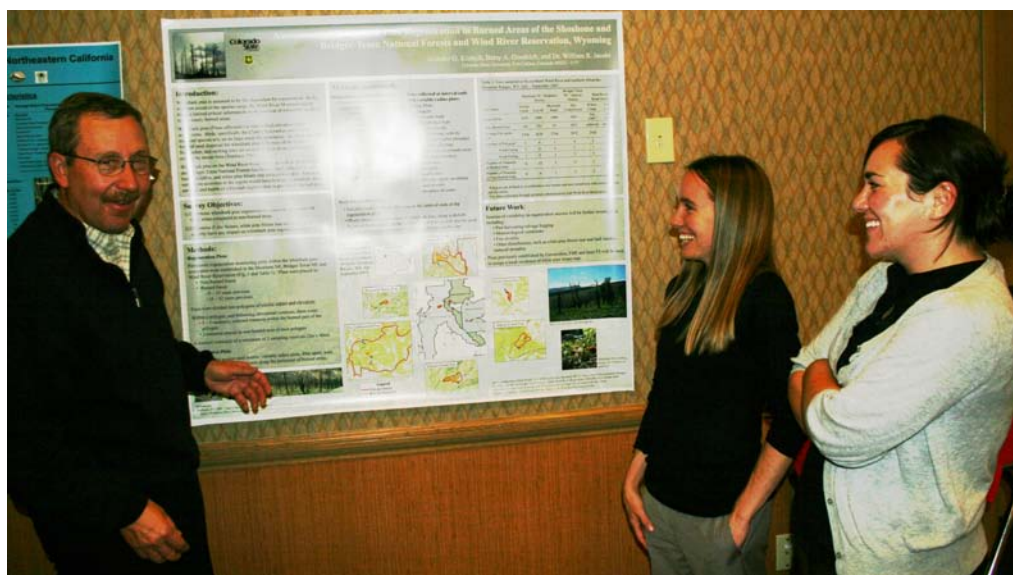
An Armillaria Survey in Mexico: a Basis for Determining Evolutionary Relationships, Assessing Potentially Invasive Pathogens, Evaluating Future Impacts of Climate Change, and Developing International Collaborations in Forest Pathology

Phil Cannon[1], Ned B. Klopfenstein[2], Mee-Sook Kim[2], John W. Hanna[2], Rosario Medel[3] and Dionicio Alvarado Rosales[4]

Abstract

In September 2007, a collaborative effort was made to survey Armillaria species in three general areas of south-central Mexico. Collected Armillaria isolates will be subjected to DNA analyses to examine genetic relationships with other Armillaria species. These studies will provide baseline information for examining evolution of Armillaria spp., assessing potential for hybridization or invasive species risk of Armillaria spp., and evaluating potential impacts of climate change. During this trip, observations were made that may provide insights into other forest diseases, such as Annosus root disease, white pine blister rust, dwarf mistletoe, oak wilt, and scorch. Because of common research interests in forest pathology, it seems essential that collaborations are strengthened between the USA and Mexico.

Overview Of The Trip

During 2007 September 9–26, a trip was taken to Mexico in search of *Armillaria* spp. in forest areas south and east of Mexico City. The locations are shown in figure 1 and listed in table 2 along with their respective elevations. Collection sites were located between 1,300 and 3,600 m (4,300 to 11,800 ft) above sea level. As a general rule, the closer the

[1]Cannon, Phil is a Regional Plant Pathologist at the USDA Forest Service Forest Health Protection, Region 5, Vallejo, CA. pcannon@fs.fed.us.
[2]Klopfenstein, Ned B., Kim, Mee-Sook, and Hanna, John W. are Research Plant Pathologist, contract Plant Pathologist, and Biological Technician, respectively, at the USDA Forest Service – Rocky Mountain Research Station, Forestry Sciences Laboratory, Moscow, ID.
[3]Medel, Rosario, is a Postdoctoral Researcher at Unidad de Micologia, Instituo de Ecología A.C., Apartado Postal 63, 91070, Xalapa, Veracruz, México.
[4]Alvarado Rosales, Dionicio is a Professor of Forest Pathology at Colegio de Postgraduados, Programa de Fitopatologia, Km. 36.5 Carr. México-Texcoco 56230. Montecillo, Edo. de México.

location was to the Gulf of Mexico, the higher the annual rainfall. With respect to the effect of elevation, higher elevations are typically associated with less rainfall (the moisture drops out at lower elevations) and rain is limited at elevations above ca. 3,200 m (10,500 ft).

Figure 1—Locations visited for the Armillaria collection trip in Mexico.

As expected, forest areas on the Pacific side also receive less rainfall due to a pronounced rain shadow effect. Because the 12 different locations varied in elevation, precipitation, and soils, they also varied in the forest species that occurred (table 2). Although it is difficult to generalize because of site differences, *Abies religiosa*, *Pinus hartwegii*, *Pseudotsuga menziesii*, *Pinus ayacahuite*, *Quercus* spp., *Alnus* spp., and *Arbutus* sp. were among the tree species found at higher elevations (2,500 to 3,500 m). *Liquidambar* spp., *Quercus* spp., *Platanus* spp., Araucaria spp., *Carpinus* sp., Miconia sp., and *Pinus* spp. were found at elevations below 2,500 m. Many of the coniferous forests were managed using a system of partial cutting that removes a small proportion (about 40%) of the

timber volume on a piece of ground about once every 12 years. We saw no evidence of clear-cutting.

Soils in these areas of Mexico are commonly derived from various pyroclastic materials. In some locations, soils were composed of volcanic ash that had accumulated to great depths. On these deep ash sites, classic Andept soils were frequently found, especially at the cooler, higher elevations where organic matter deposition outpaced organic matter mineralization. More commonly, however, soils found on this trip were composed of a shallow deposit of ash (10 cm to 1.5 m) on top of ignimbrite. Usually, this ignimbrite had been shattered into small rock fragments, so at least some portion of a tree's root system could penetrate to significant depth. Ignimbrite (rock) was common near the soil surface on steep hogback ridges, and these sites were sometimes associated with the occurrence of Douglas-fir. At similar elevations, *Pinus ayacahuite* and several species of oaks could be found on soils where deeper deposits of ash occurred, which was likely associated with more available soil moisture.

Although this trip focused primarily on collecting *Armillaria* spp., other forest diseases were observed. Some preliminary information and samples were collected that may provide useful information for establishing additional collaboration between Mexican and USA forest pathologists. It should be noted that *Armillaria* spp. had been reported previously on some trees in Mexico (see ahead); however, DNA-based identification of Mexican *Armillaria* spp. has not been previously reported.

Finally, the timing of this trip was noteworthy. The trip began the week of September 9 after Hurricane Felix had passed through the area and near the end of the rainy season. These circumstances were fortuitous for our collection work. The mushrooms and diverse fungal fructifications were quite abundant during our surveys, although the local mycologists said that the peak season for collecting *Armillaria mellea* mushrooms began 6 weeks earlier (i.e., early August) and *A. ostoyae* (or *A. polymyces*) mushrooms are more prevalent in December. Additionally, whole trees had been wind-thrown in many locations due to the hurricane. This was particularly evident on the northern slopes of El Cofre de Perote where wind gusts had reportedly reached 160 km/hr. These freshly wind-thrown trees, although a negative impact on the forest, allowed our group to easily examine the base of the root wad and survey for fungi and rotten roots.

The Quest For Armillaria Species

Background

A research group from the Rocky Mountain Research Station in Moscow, Idaho (Klopfenstein, Kim and Hanna), has been studying molecular diagnostics, genetic characterization, and evolutionary relationships among Armillaria species, especially *A. ostoyae* (Hanna and others 2007a, b; Kim and others 2000, 2001, 2006). These studies involve the DNA analyses of diverse *Armillaria* samples from several countries in the Northern Hemisphere. These studies, coupled with the fact that Mexico is well known as the center of diversity for many coniferous genera (which are the principal hosts of *A. ostoyae*) provided the impetus for the expedition to the coniferous forests of Mexico. The study of Mexican Armillaria isolates might provide insights into the southernmost extent of *A. ostoyae*, origin of Northern Hemisphere Armillaria species, and evolutionary relationships among *Armillaria* spp. This baseline information is needed to evaluate the potential invasive species risk associated with Armillaria root disease pathogens. In addition, the epidemiology and ecology of the *Armillaria* pathogens in Mexico may provide insights into the future behavior of these pathogens in the USA as affected by climate change.

Methods

Some forest pathology research and related papers have reported the existence of Armillaria species in Mexico (e.g., Alvarado-Rosales and Blanchette 1994; Alvarado-Rosales and others 2007; Murrill 1911; Pérez-Silva and others 2006; Shaw 1989; Tkacz and others 1998; Valdés and others 2004). Additional information on *Armillaria spp.* in Mexico is found in reports of edible mushrooms, commercial mushrooms, and ethnobotany (e.g., Montoya and others 2003; Montoya-Esquivel and others 2001; Ruán-Soto and others 2006). Specific information is also found in surveys of forest fungi and mycological forays, such as Ph.D. theses by Dionicio Alvarado-Rosales (in collaboration with Robert Blanchette) and Florencia Ramírez Guillén (in collaboration with Gastón

Guzman). Currently, there are no reports of DNA-based diagnostics to identify Armillaria species from Mexico.

Four of the sample locations shown in table 2 were selected based on these earlier reports. Four other sample locations were selected because native Douglas-fir stands were known to exist in these areas, which represent the southern limits of this species. Douglas-fir stands were selected because of their relationship to forests of the western USA and to examine whether *Armillaria* spp. and Douglas-fir had co-evolved. The remaining four locations were chosen because they were convenient and represented promising habitat for *Armillaria* spp.

At each collection site, Armillaria surveys were conducted by inspecting representative tree species present, regardless of health status. In addition, trees displaying potential symptoms of root rot were also inspected. Using a small, sharp hoe, collectors inspected trees by excavating and pulling away all of the duff and soil from around the base of the tree and along two of the major roots to a depth of about 20 to 30 cm. Brushes were used to clear away dirt that was in close contact with the roots. In cases where wood was infected, a hatchet or knife was used to chop out small sections of the wood to reveal zone lines or wood rot in these sections.

Collectors sought out mushroom caps or rhizomorphs, the most readily observable signs of *Armillaria* spp. (figure 2). Flexible rhizomorphs (ca. 10–60 cm) were collected from a tree, and placed into labeled, 15-ml collection tubes. Mycelial fans or decayed wood with zone lines were considered as an alternative source for Armillaria isolation, though it is more difficult to isolate this fungus from decayed wood. Armillaria mushrooms, mycelial fans, and/or pieces of rotted wood with zone lines were placed in labeled paper bags and stored in an ice chest until the fungus could be isolated. Additional data recorded included a few photos of the tree, GPS readings (for latitude, longitude, and elevation), slope, aspect, host species, host symptoms, and associated plants. At the end of each collection effort in each of the three areas (Texcoco, Xalapa, and Oaxaca), one day was devoted to making isolations and establishing cultures.

Figure 2—Signs of *Armillaria* spp.: fruiting bodies (A); rhizomorphs (B); mycelial fan under bark (C); and zone lines in infected wood (D).

The following procedure was followed when making the isolations from the rhizomorphs. The rhizomorphs were rinsed, in their collection tubes, with tap water to remove soil and other debris. They were then soaked in a 20% Clorox® solution for 10 minutes, and rinsed with filter-sterilized, distilled water. They were then soaked in a 3% hydrogen peroxide solution for 10 minutes and rinsed again with filter-sterilized, distilled water. Finally, the rhizomorphs were cut into 1-cm-long sections and placed into media slants within culture tubes. Two types of media were used: BDS (Benomyl-Dicloran-Streptomycin; Worrall and Harrington 1993) and "Very Cold Armillaria Medium" (VCAM; containing 1.5% agar, 0.75% malt extract, 0.5% peptone, and 0.75% dextrose).

Wood samples were treated by dipping the sample in alcohol, briefly flaming it, and excising a small fragment from it with a sterile scalpel. Fragments of infected wood were placed onto media slants within culture tubes. Similarly, small sections of fungal material from the mushrooms were retrieved from the pileus or stipe, which had already been alcohol flamed and split open to allow the removal of small interior sections for culturing in the slants. Each isolate was cultured in six to 10 culture tubes containing BDS and VCAM.

Preliminary Results

Armillaria spp. were found in some sites at each of the three areas (Texcoco, Xalapa, and Oaxaca) visited. Where found, the fungus was usually in very small infection foci (one to three trees). On one occasion we

found a larger Armillaria infection center of approximately 1 hectare in extent. In general, we were more successful in finding Armillaria in the more humid areas, for example on alder, oak, or sycamore in valley areas as opposed to on the dry pine or Douglas-fir areas that occurred in high, dry slopes and ridges. Overall, we obtained 30 different sample collections - 23 from rhizomorphs, five from wood, and two from fruiting bodies. It is especially noteworthy that *Armillaria* spp. were not found in association with Douglas-fir and were quite rare on true fir.

The Next Steps

At the end of this trip, we sent all samples to the USDA-APHIS-PPQ where they were checked to ensure safe packaging before they were delivered to the laboratory in Moscow. The shipment of Armillaria cultures from Mexico was in compliance with a USDA-APHIS-PPQ permit issued to the Moscow laboratory. In Moscow, each of the isolates that grows in culture will be sub-cultured onto Petri plates containing the same culture media. After a few weeks in culture, the isolates will be ready for DNA analyses.

In general, DNA analysis involves using a small scrape of the mycelium from the culture plate, which serves as the DNA template for polymerase chain reaction (PCR). The PCR process allows one region of the ribosomal DNA (rDNA, such as nuclear large subunit or intergenic spacer) to be amplified to produce millions of copies. The amplified region is then subjected to DNA sequencing, which allows comparisons among the isolates. The rDNA sequences from Mexican Armillaria isolates will be compared with the rDNA sequences of Armillaria from many other regions across the Northern Hemisphere. These comparisons will allow determinations of the genetic relationships among many different genets and species of Armillaria.

Heterobasidion Annosum

Heterobasidion annosum (previously *Fomes annosus*) has been previously reported in Mexico (e.g., Asiegbu and others 2005; Garbelotto and Chapela 2000; Guevara and Dirzo 1998; Johannesson and Stenlid 2003; Maloney and Rizzo 2002; Martínez-Barrera and Sánchez-Ramírez 1980; Ruiz-Rodriguez and Pinzon-Picaseno 1994; Sinclair 1964). Unfortunately, this

pathogen has not been well characterized using DNA analysis. However, studies are currently underway to examine the phylogenetic relationships of *Heterobasidion annosum* P-type intersterility group (P ISG) from Mexico with other members of this group from North America and Eurasia (Linzer and others 2007). We found *Heterobasidion annosum* on a *Pinus patula* stump (ca. 40 cm in diameter) on the top of a ridge at the Ciclo Verde Christmas Tree Plantation near Xalapa (figure 3). We located several additional *P. patula* stumps with *H. annosum* conks. The conks were typically quite small with about 1–2 cm2 worth of freshly sporulating surface per conk (figure 3).

Figure 3—*Pinus patula* stump in the Ciclo Verde Christmas Tree Plantation, Xalapa, Mexico (A); fruiting body of *Heterobasidion annosum* found on a stump (B).

Many of the native *P. patula* in this area had recently been felled in an intensive selective cut that left about 40 well-spaced trees per hectare. The wood from the felled trees was processed for lumber, and Christmas trees (*Cupressus lusitanica*, *P. ayacahuite*, and *Pseudotsuga menziesii*) were planted in the understory of the residual trees. A few of the residual trees had blown over as a result of Hurricane Felix, which had reportedly delivered winds of 160 km/hr through this stand. This provided an easy opportunity to study the base of the roots in these upturned trees. In one situation, we found that several of the principal "tap" roots had been severely decayed and observed *H. annosum* fructifications on the stump and root system. The *Annosus* root disease had pre-disposed this tree to wind-throw. Further indications showed that this root disease was becoming a new problem in this area. No well-established, root-rot foci were found, and none of the stumps examined showed much more than incipient decay.

Although this disease is not well known in Mexico, it seems likely that the management practices at Ciclo Verde could have contributed to the recent build-up of

the pathogen. More importantly, the build up of *Annosus* root disease could have important long-term consequences on the native *P. patula*. This pathogen presents a problem for the *P. ayacahuite* in the Christmas tree plantation, as it can spread by root contact or spore-derived infections of freshly cut stumps. We talked with the foreman, Don Lupe, about the problem. He listened carefully, and he was eager to learn more about possible control measures.

Figure 4—Unknown fungal pathogen on *Abies religiosa* roots.

Unknown Root Disease Of True Fir

We found an unknown root-rot pathogen infecting *Abies religiosa* growing at high-elevations in Tlaxcala and Hidalgo. This root disease was apparent in wind-thrown and dead/declining trees. No fruiting bodies were observed; however, white mycelia were apparent under the root bark of infected trees (figure 4). Further investigation of this disease is warranted, and perhaps rDNA sequencing can help identify the unknown pathogen.

Rust

Forest pathologists in the USA (e.g., Brian Geils, USDA Forest Service) are establishing collaborations with forest pathologists in Mexico to assess the risk of white pine blister rust (*Cronartium ribicola*) to any of the five-needled, white pines that are native in Mexico. To date, white pine blister rust has not been reported in Mexico. However, blister rust does occur on *Pinus strobiformis* in New Mexico (Hawksworth 1990; Van Arsdel and others 1998; Conklin 2004), and *P. strobiformis* also occurs in the northern half of Mexico.

Several other species of five-needled, white pines occur at diverse locations in Mexico.

Figure 5—Infected *Ribes* leaf with rust uredinial pustules (A) and non-infected *Castilleja* plants (B).

To assist in the effort to assess the risks of white pine blister rust, we briefly inspected *Ribes* and *Castilleja* plants. *Ribes* plants were found in essentially every area that we visited, except Oaxaca, and *Castilleja* spp. were common in most areas (figure 5). We frequently found uredinial pustules on *Ribes* leaves at several locations. The GPS location of these occurrences was recorded and photos were taken of the rust infections and *Ribes* plants. The identification of this rust remains unknown, but it appeared widespread within the areas we visited. Preliminary observations indicated that the rust could potentially belong to the *Coleosporium* genus; however, DNA-based diagnostics are needed to conclusively identify the rust pathogen of *Ribes* spp.

Figure 6—Native *Pinus ayacahuite*, which is potentially susceptible to white pine blister rust. A native tree near Oaxaca, Mexico (A), and a Christmas tree plantation of *P. ayacahuite* near Xalapa, Mexico (B).

The identification of rusts in Mexico may help assess risks for white pine blister rust. Although we never observed any symptoms of rust on any of the white pines, time did not permit more that a cursory inspection. The planting of susceptible *P. ayacahuite* is quite common in Christmas tree plantations at diverse

locations in Mexico (figure 6). These plantations may significantly influence the risks associated with white pine blister rust. Furthermore, rust could potentially "over-winter" as uredinia on *Ribes* in Mexico, thereby maintaining a pathogen population in the absence of aecial host infection.

Oak Wilt

Surveys are needed to determine whether oak wilt occurs in Mexico (Appel 1995). Although we observed thousands of Mexican oak trees of diverse species during this visit, we did not see any with symptoms of oak wilt. Indeed the vast majority of oaks that we looked at seemed quite healthy (Figure 7). However, it cannot be ruled out that oak wilt could occur in Mexico, and it is unknown if oak wilt-infected trees will display typical oak wilt symptoms in Mexico.

Figure 7—One example of the many oak (*Quercus*) species found in Mexico.

Xylella Fastidiosa

Xylella fastidiosa is a bacterial pathogen that causes scorch and dieback in a number of forest trees, fruit trees, vines, and ornamental plants in the USA (Hopkins 1989). It is well known for its effects on sycamore (*Platanus* sp.) and sweetgum (*Liquidambar* sp.). On sycamore throughout most of the southeastern

USA, it causes a devastating disease on trees that are 4 years old and older. Breeding programs to develop resistance to this pathogen are underway (sycamore) and being considered (sweetgum). Mexico is known as a source of potentially resistant germplasm for both of these species and their nearby relatives.

We observed several hundred sycamore and sweetgum trees (figure 8). None showed the apparent symptoms commonly associated with *Xylella fastidiosa*. It is unknown whether the absence of leaf scorch is due to host resistance to the disease/insect vector or absence of the pathogen or its most common vectors (glassy-winged sharpshooters) in this environment. However, we did not see any evidence to indicate that these Mexican sycamore and sweetgum sources were susceptible to this pathogen.

Figure 8—Apparently healthy sycamore, *Platanus* sp. (A), and sweetgum, *Liquidambar* sp. (B).

Dwarf Mistletoe

Dwarf mistletoe was extremely abundant in most of the conifer stands that we visited and perhaps most abundant in the forests that were being managed for timber production via a system of partial cutting (figure 9). The Forestry Stewardship Council (FSC) certified some of these forests as model forests. The very heavy brooming that is commonly found when dwarf mistletoes are established on some conifers was sometimes observed, indicating that these mistletoes are exerting a burden on their coniferous hosts. The practice of partial cutting, which allows infected trees to rain vast numbers of dwarf mistletoe seeds down on the regeneration that follows the harvest, may be contributing to heavier levels of infection.

For many decades, dwarf mistletoe has been recognized as a serious problem in many coniferous forests of the USA, and control measures have been designed to address this problem (Hawksworth and Wiens 1996; Geils and others 2002). It is likely that many management practices used in the USA would be useful in Mexico. Translating this subject matter would ensure its availability in Mexico. It is worth noting that Frank Hawksworth, the world's leading authority on dwarf mistletoes, made several trips to Mexico to document the species of this pathogen in the coniferous forests. Currently, Bob Mathiasen (Northern Arizona University, taxonomy and distribution) and Brian Geils (USDA Forest Service-RMRS, epidemiology) have the expertise to work with dwarf mistletoes in Mexico. Forest Health Protection pathologists from Regions 2 and 3 are working with cooperators in northern Mexico on a dwarf mistletoe–silviculture project (see Howell and others, these proceedings).

Figure 9—Dwarf mistletoes near Popocatépetl.

Bark Beetles

We observed little evidence of bark beetles; however, we did see some bark beetle-attacked trees 10 km northeast of San Miguel del Valle. As expected, they were located on the drier parts of the landscape. According to Demitrio Policarpo Santiago (Oaxaca), their company removes bark beetle-impacted trees during timber harvest (figure 10). This management practice appeared to be efficacious, although it requires extra effort during the harvest period to access these dying trees that are typically scattered across the landscape. Previously we mentioned that partial cuts are likely a contributing factor to the dwarf mistletoe problem. To the contrary, partial cutting and rapid removal of bark beetle-infested trees should relieve

between-tree competition that can exacerbate further infestations of bark beetles.

Previous and ongoing collaborations among Mexican and USA forest entomologists may have helped to develop sound management practices to reduce bark-beetle problems. Regardless of how these practices came about, it was apparent that the coniferous forests we visited have been well managed for stand density. This management likely contributed significantly to the overall health of the residual trees in the forest.

Figure 10—An example of bark beetle damage to *Pinus* sp. in Oaxaca.

Climate Change

Because our trip occurred toward the end of the rainy season, the areas we visited were at their absolute greenest. During our travels, we rarely observed forested areas that were experiencing higher than normal levels of natural mortality. This was somewhat surprising since mortality rates in coniferous forests of some other areas have become exceedingly high within the last few years (e.g., about 50% mortality of mature coniferous trees has been observed in National Forests in southern California during the past 5 years).

The local foresters in Mexico indicated that the past 5 years, including the current year, have been far drier than previous years. One forester commented, "The

rains are disappearing in September now, whereas they used to go until the end of October." Still, the drought situation in sites that we visited has apparently not resulted in the severe impacts that have been recently observed in the southwestern USA.

The Douglas-fir stands in Oaxaca represent the southernmost extent of this species (Delbreczy and Rácz 1995). These "island" populations are at the limit of the species range, and thus may be most at risk to climate change. Some mortality was found in these stands, but root rot was not found (figure 11). The mortality was associated with several years of dry weather, which may be associated with changing climate. The most common symptom observed in these Douglas-fir stands, however, was a paucity of older foliage and smaller duskier tufts of needles at the branch ends. This, too, is very symptomatic of extreme and sustained drought, but so far, the trees have demonstrated remarkable resilience.

Figure 11—Douglas-fir stands in Oaxaca, Mexico. Some mortality was found in these stands, perhaps due to climate change (prolonged drought) (A). Pine mortality is also potentially associated with climate change (B).

Future Collaboration

Many opportunities exist for collaboration among Mexican and USA forest pathologists. Although many Mexican forest pathologists and related professionals can speak English, Mexico–USA collaborations would benefit substantially by the establishment of bilingual (English–Spanish speaking) forest pathologist positions in the USA that would maximize information exchange with Mexico and Latin America.

The DNA-based studies of Armillaria may result in collaborative publications and raise new phylogenetic questions that warrant further study. Forest pathologists from both countries need to understand the distribution

and ecological behavior of diverse Armillaria species and subspecies. This baseline information will help forest managers understand the risks of hybridization and/or invasiveness and shed light on potential interactions with climate.

Figure 12—Interactions with our Mexican contacts at various collection sites and local institutions in Mexico.

The incipient *Heterobasidion annosum* situation would benefit greatly from information sharing and collaboration. Collaboration among USA and Mexican forest pathologists and foresters should promote information exchange about measures that can be successfully deployed to limit the spread of this disease while it is still in the incipient stage. The USA has extensive experience in managing this disease in the southeastern and western forests. However, forest pathologists in the USA need to understand the ecological behavior of *H. annosum* in Mexico. Matteo Garbelotto (University of California, Berkeley) is currently studying the phylogenetic relationships of *H. annosum* from Mexico and comparing them with those found in the USA and other Northern Hemisphere regions. This work is important to understanding the identity and ecological behavior of these pathogens. Similarly, the USA and Mexico could benefit from collaboration and information sharing focused on reducing the impact of dwarf mistletoe diseases (e.g., eight USA states have fairly aggressive dwarf mistletoe programs).

Rust was evident on *Ribes* in many locations, but we found no evidence of it on coniferous hosts. Experienced rust pathologists should further investigate and monitor this disease since the presence of rust on *Ribes* could help determine whether suitable environments for white pine blister rust occur in Mexico. Additional opportunities exist for forest pathology collaboration between Mexico and the USA, but these opportunities lie outside the initial focus of our Armillaria collection trip (figure 12).

Acknowledgements

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Table 1—Mexican and USA collaborative contacts for *Armillaria* collection trip.

Name	Expertise	Affiliation
Principal Contacts		
Dionicio Alvarado Rosales	Forest Pathology	Colegio de Postgraduados, Texcoco
Carlos Martinez	Plant Pathology	Colegio de Postgraduados, Texcoco
Rosario Medel	Mycology	Instituto de Ecologia, Xalapa
Patricia Negreros-Castillo	Forestry	Universidad Veracruzana, Xalapa
Florencia Ramírez Guillén	Mycology	Instituto de Ecología, Xalapa
Gastón Guzman	Mycology	Instituto de Ecología, Xalapa
Armando López	Mycology	Instituto de Genética Forestal
Elfego Chávez Gonzáles	Forestry	Consultor Privado, Oaxaca
Marcario Pérez López	Forestry	Consultor Privado, Oaxaca
Victor Hernández Guzmán	Forestry	Gerente de la Empresa Forestal La Cumbre
Félix Luis Ramírez	Forestry	Mayordomo El Carrizal
Alejandro Hernández	Forestry	Vice President San Miguel Del Valle
Demitrio Policarpo Santiago	Forestry	Treasurer, San Miguel Del Valle
Additional Contacts		
Efren Cázares	Mycology	Oregon State University (via Monterrey)
Jesús García Jiménez	Mycology	Instituto Tecnológico de Cd. Victoria,Tamaulipas
Gonzalo Guevara	Mycology	Instituto tecnológico de Cd. Victoria, Tamauliaps
Miguel Angel Muniz Castro	Mycology	Instituto de Ecología, Xalapa
Matt Smith	Mycology	Harvard University (from Dave Rizzo lab)
Greg Bonito	Mycology	Duke University (Rytas Vigalys' lab)
Jim Trappe	Mycology	Consultant (via USDA FS, Corvallis, OR)

Table 2—Field locations visited during this trip.

Locations in Mexico	Examples of host genera sampled	Elevation (m)	Accompanied by
Near Texcoco, Mexico State			
Popocatépetl	*Abies, Ribes*	2,900 – 3,300	Dionicio Alvarado Rosales
Parque Nacional El Chico	*Abies, Pinus, Pseudotsuga*	2,800	Dionicio Alvarado Rosales
Villarreal	*Abies, Pinus, Ribes*	3,300	Carlos Martinez
Bosque University of Chapingo	*Abies, Alnus, Pinus, Senecio*	3,400	Dionicio Alvarado Rosales
Near Xalapa, Veracruz			
Mesophyll Caceres	*Carpinus, Liquidambar, Miconia, Quercus*	1,500 – 1,600	Rosario Medel
Cofre de Perote	*Abies, Pinus, Ribes*	3,500 – 3,600	Rosario Medel
Reserva Ecológica, San Juan del Monte, south of Las Vigas	*Alder, Pinus*	2,300	
Instituto Genética Forestal, El Haya Parque Ecológíco, Xalapa	*Platanus, Quercus*	1,400	Armando López
Asseradero 100, Xalapa	*Araucaria*	1,400	Armando López
El Ciclo Verde Christmas tree farm, Las Vigas	*Pinus*	2,500	Rosario Medel
Areas near the "Hayas parking zone", Instituto de Ecologia, A.C.	*Eriobotrya, Platanus, Quercus*	1,300	Rosario Medel
Near Oaxaca, Oaxaxa			
Peña Prieta, in Parque La Cumbre - Ixtepeji	*Arbutus, Pinus, Pseudotsuga, Quercus*	2,800	Marcario Pérez López
El Carrizal, 10 km north-east of San Miguel Del Valle	*Alnus, Pinus, Pseudotsuga, Quercus*	2,600 – 2,800	Demitrio Policarpo Santiago
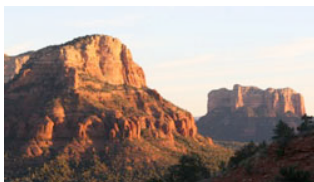
Reforestation and Genetic Strategies for Southern California Forests

Patricia E. Maloney[1], Detlev R. Vogler[1] and David M. Rizzo[2]

Recent drought conditions in southern California have caused widespread mortality of forest trees across montane landscapes in the region. Fire suppression and stand densification have predisposed trees to root diseases and other pathogens, reducing tree vigor and making them more susceptible to bark beetles and other insects during extended drought periods. A complex of stressors is significantly affecting forest stand conditions in the region, and may warrant developing alternative reforestation and genetic strategies. The Sierra San Pedro Martir, SSPM, Northern Baja, Mexico, harbors the southern extent of mixed-conifer species, and lies at the southern margin of the North American Mediterranean climate zone. Species from the SSPM may be more "drought tolerant" since climatic conditions are more xeric than in southern California. This project (pending funds from S&PF Western Competitive Grants 2008) will begin with seed collections from SSPM and southern California populations of Jeffrey pine, sugar pine, and white fir for common garden studies, to evaluate genetic and environmental influences on survival, growth, water-use efficiency, phenology, response to atmospheric pollutants, and other traits.

If SSPM or more xeric-adapted southern California genotypes of these species prove to be more "drought tolerant" than some populations in southern California, these individuals will likely be less susceptible to insect pests, especially to those that preferentially attack drought-stressed trees; they may also exhibit increased tolerance to some pathogens. Additionally, drought adapted and water-use efficient phenotypes may be more ozone resistant, given tight regulation of stomatal conductance. Such information will be useful in developing reforestation strategies in the region with genetic material physiologically and phenologically suitable and more resilient under warmer and drier conditions. Given the severity and frequency of wildfire in southern California and the intact fire regime in the SSPM, establishing a seed-bank of southern California and SSPM species will protect this valuable germplasm, offering ecological and environmental benefits for future generations.

[1]Patricia E. Maloney and David M. Rizzo are with the Department of Plant Pathology, University of California, Davis, CA. patricia-maloney@sbcglobal.net and dmrizzo@ucdavis.edu.
[2]Detlev R. Vogler is with the Institute of Forest Genetics, Pacific Southwest Research Station, USDA Forest Service, Placerville, CA. dvogler@fs.fed.us.

Risk of Interstate Movement of Invasive Tree Pests

Dr. William R. Jacobi[1] and Betsy A. Goodrich[2]

The invasive exotic insect emerald ash borer (*Agrilus planipennis*) provides a model invasive organism to assess the risk of various movement pathways of exotic pathogens or insects into and within urban and forest ecosystems in the Western United States. The emerald ash borer can be moved on nursery stock, firewood, and other wood products such as pallets and minimally processed logs. Natural tree connections will most likely allow the insect to move west to the plain states (Nebraska, Kansas, etc.) in 30 years and then down river corridors which contain naturalized ash to Colorado. Nursery stock movement is complex in the U.S. and is a high risk pathway for moving pathogens and insects from state to state. The amount of nursery stock moved into and within the west is not known, but estimates suggest there are approximately 4.7 million trees and 30 million shrubs planted each year.

Quantifying the movement of insects and pathogens on commercial firewood has not been assessed, and the volume and directional movement across state lines is not known. A west-wide retail firewood survey of 137 outlets in 27 cities across 10 states was conducted in spring 2007 and revealed that 38% of the wood assessed came from companies whose headquarters were located out of state. Sixty percent of the wood was conifer, 30 % was hardwood, and 10% of the bundles contained both. We do not know the precise origin of the wood, but most of the out-of-state wood did not appear to be from the local state. An ongoing survey of firewood in Colorado will assess the amount of insect and fungal presence in the firewood. A survey conducted in summer 2007 of campers in Colorado campgrounds noted that 9% of visitors had brought wood from their home state instead of purchasing the wood in Colorado. Future work will continue to assess the incidence of out of state firewood movement throughout Colorado and western US. Monitoring insects and fungi on collected firewood samples will provide further information on the incidence of infestation.

Bark Beetle Outbreaks in the West: Influences of Past, Current, and Future Climate Change

Jeffrey A. Hicke[1] and Jesse A. Logan[2]

Abstract

Climate variability and change are important drivers of bark beetle outbreaks through effects on life cycle development rates, overwintering mortality, and host tree capacity for defense. Several widespread bark beetle infestations in recent years have been influenced by these aspects of climate. Quantitative models of climate and stand suitability for insect outbreak allow future predictions given expected climate change. Projected warming in the coming decades is likely to continue and enhanced current trends of northward and eastward across the boreal forest and upward range expansion and extensive severity.

Introduction

Bark beetles are significant disturbance agents in forests throughout western North America. In recent years, bark beetles have affected over 4 million ha in the western United States (USDA Forest Service, 2005). Mountain pine beetle (*Dendroctonus ponderosae*) is the most damaging bark beetles, affecting over 2 million ha in the early 1980s (USDA Forest Service, 2005) and has affected over 9 million ha since 2000 in British Columbia and Alberta, Canada (British Columbia Ministry of Forests and Range, 2006). Maps of bark beetle outbreak locations in 2003 reveal that almost every forested region in the western United States is being subjected to attack (USDA Forest Service, 2005). Various factors facilitate infestations of bark beetles. Mountain pine beetle is one of the most well-studied bark beetles in western North America. Populations of this insect reach epidemic proportions in response to factors associated with their host trees (principally lodgepole pine (*Pinus contorta*) and ponderosa pine (*Pinus ponderosa*), but also whitebark (*Pinus albicaulis*) and limber pine (*Pinus flexilis*)). Stand structure

[1]Hicke, Jeffrey A. is an Ecological Geographer in the Department of Ecology, University of Idaho, Moscow, ID.
[2]Logan, Jesse A. is a retired USDA Forest Service Entomologist now living in Emigrant, MT.

characteristics such as older and denser stands are more susceptible as tree vigor, and therefore defensive capacity, is reduced for these stands (Shore and others, 1989). Droughts also stress trees and cause them to be more susceptible to attack (Carroll and others, 2004). Beetle populations themselves also respond to climate factors through temperature. Again we use mountain pine beetle as an example. The annual course of temperatures governs the developmental rate of the different life stages of mountain pine beetles (Bentz and others, 1991). When temperatures confer "adaptive seasonality," beetles shift from semi- to univoltinism (one life cycle per year), are synchronized for mass attack that enables overcoming tree defenses, and result in adult beetle emergence during late summer (Logan and Powell, 2001). Similar responses of voltinism to temperature have been observed for spruce beetle (Hansen and Bentz, 2003). In addition to adaptive seasonality, extremely low winter temperatures kill overwintering mountain pine beetle larvae (Safranyik and Linton, 1998; Regniere and Bentz, 2007).

Although it seems unequivocal that climate plays a role in driving bark beetle outbreaks, the nature of climatic influences is complex. For example, a study of adaptive seasonality of mountain pine beetle in the western United States revealed that although most locations that experienced adaptive seasonality had mean annual temperatures of 5ºC, the range of mean annual temperatures for such locations spanned 8ºC (Hicke and others, 2006). Thus, mean annual temperature is only a rough index of adaptive seasonality and illustrates the usefulness of mechanistic models for assessing climate effects on beetle populations. In addition to the complexity of climate, the relative importance of the different climate drivers has yet to be determined. Such understanding will be important for predicting outbreaks in the context of future climate change.

Recent Outbreaks And Climate Change

A number of severe outbreaks of bark beetles have occurred in recent decades, and some form of climate variability or change has been linked to each. Populations of mountain pine beetle have increased since 2000; these infestations affected almost 1 million ha in 2003 (USDA Forest Service, 2005). Locations of major outbreaks in recent years include central Idaho and north-central Colorado. The Sawtooth Valley in central Idaho is typically too cold to support outbreaks, yet an extensive epidemic has occurred there since the late 1990s (Logan and Powell, in press). Both regions have experienced drought and increasing temperatures. A modeled switch from semivoltinism to univoltinism in the mid-1990s in the Sawtooth Valley followed by exponentially increasing outbreak area is strong evidence of the role of warming there (Logan and Powell, in press). Carroll and others (Carroll and others, 2004) analyzed the extensive outbreak in British Columbia with a model that included the simplified effects of adaptive seasonality together with winter mortality and drought stress. The authors showed the increasing climate suitability in British Columbia in recent years in response to the warming

Mountain pine beetles are undergoing both northward range expansion (Carroll and others, 2004) and extensive outbreaks in high-elevation whitebark pine ecosystems in response to the recent warming (Logan and Powell, 2001). Whitebark pine mortality due to mountain pine beetle is currently high across the northern Rocky Mountains in the US (Gibson, 2006). Although mountain pine beetle outbreaks have occurred in the past (Perkins and Roberts, 2003), today's continued warm conditions seem different from the transient warming of the 1930s and 1940s during the last major outbreak (Logan and Powell, 2001).

Other species of bark beetles have also undergone extensive epidemics linked to climate change. Spruce beetles (*Dendroctonus rufipennis*) attacked spruce over an area of 1.5 million ha in Alaska and the Yukon Territory since the early 1990s (Berg and others, 2006). Berg and others (2006) implicated warm conditions that induced univoltinism, warm winters, and drought stress.

In conjunction with a severe drought in the American Southwest, Ips beetles killed >1.2 million ha of pinyon pine (*Pinus edulis*) in the early 2000s (Breshears and others, 2005). Although precipitation anomalies were greater in the 1950s drought in this region, warmer conditions led to greater drought stress on trees. The role of the Ips beetle was acknowledge through hypothesized warming effects on population development rates (Breshears and others, 2005), but little has been quantified.

Future Behavior Of Bark Beetles

Future patterns of outbreaks of bark beetles depend on host stand structure as well as the changing climate. Susceptible conditions of both factors are needed. A recent analysis estimated stand susceptibility of lodgepole pine to mountain pine beetle attack across the western United States (Hicke and Jenkins, in press). Forest inventories by the USDA Forest Service provided measurements of tree species, age, and stem density (Alerich and others, 2004). A model of stand structure susceptibility (Shore and others, 2006) was driven by these inventories to estimate the extent of lodgepole pine stands in susceptible conditions. Over 46% of lodgepole pine (2.8 million ha) was found to be in a condition that could lead to basal area losses of 30% or greater in the event of mountain pine beetle attack (Hicke and Jenkins, in press). Similar conditions exist in Canada (Taylor and Carroll, 2004). Based on forest inventories, stand ages across all tree species in the western United States appear to be in highly susceptible age classes (Hicke and others, in press), suggesting susceptibility to a variety of bark beetle species.

Model projections of climate change in the coming century predict warming of up to 3ºC in some locations (Bradley and others, 2004). In fact, the greatest summertime warming along the American Cordillera, which includes the Andes and Rocky Mountains, is predicted to be at latitudes within the United States. This expected change will have important implications for biological and ecological processes such as bark beetle outbreaks.

Responses of bark beetle populations to projected climate change continue patterns apparent in recent decades. Continued northward expansion of mountain

pine beetle may lead to invasion into jack pine (*Pinus banksiana*) in the boreal forest (Logan and Powell, 2001). Jack pine is a suitable host for mountain pine beetle (Cerezke, 1995), and its distribution ranges across boreal Canada from just eastward of the Rocky Mountains eastward to the coast.

More severe or extensive outbreaks of mountain pine beetle may occur in the future. An example might be emerging in northern Colorado, where an ongoing outbreak that began in 2000 shows no sign of abatement. Indeed, inspection of high-resolution satellite imagery during 2007 reveals many watersheds with nearly 100% mortality.

A final example of changing outbreak regimes can be found in high-elevation five-needle pine ecosystems. Temperatures in whitebark pine ecosystems are projected to continue to increase in the coming decades. We used a mechanistic model of adaptive seasonality (Logan and Powell, 2001) to investigate how future warming will influence mountain pine beetle populations (Hicke and others, 2006). We drove the model with coarse spatial resolution (0.5°) climate projections across the western United States. At the highest elevations, the area of adaptive seasonality at these elevations is predicted to increase substantially in response to rising temperatures (Hicke and others, 2006). Thus, the current warming is expected to continue in the coming century, and the predicted temperature suitability for mountain pine beetle outbreaks may allow for more extensive, frequent, and severe outbreaks in whitebark pine ecosystems.

Summary And Conclusions

Multiple aspects of climate influence bark beetle outbreaks, including the annual course of temperatures, minimum temperatures, and drought stress on trees. In recent years, several extensive outbreaks ranging across western North America and multiple insect species have been associated with aspects of climate change. Currently, bark beetles are expanding northward and upward in response to climate change, and outbreaks may be more severe or extensive than in the past. The current structure of forests in western North America implies potential susceptibility to insect infestations over wide areas. Future projections continue these patterns in response to expected warming in the next century.

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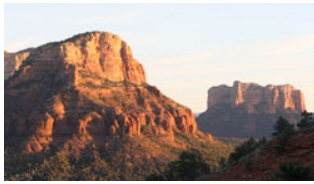
Pathology Without Pathogens: Opportunities for Forest Pathologists in a Changing Climate
Paul E. Hennon[1]

Abstract

New roles for forest pathologists in the field of climate change are suggested. Climate has the potential of altering every forest tree disease in some manner, but some diseases have only subtle effects in forest ecosystems. Climate change can also lead to retraction or expansion of species' ranges or entire regime shifts in forests. The forest declines exemplify widespread tree mortality where climate often plays a major role in initiating such wholesale forest change. This paper uses yellow-cedar decline as a case study. Pathologists have valuable expertise to offer in investigations on forest declines, and these opportunities are expected to increase as forest trees become maladapted in response to rapidly changing climate. Pathologists can also contribute in multidisciplinary studies on carbon sequestration by offering an understanding of heart rot (stem decay), decomposition rates, and sequestered carbon in the modified lignin form of brown rot. Pathologists can engage in the development of new principles of forest health and forest management to deal with predicted effects of climate change as well as uncertainty. A new approach is needed to guide forest managers to create managed forests that are resilient, with enough species and functional diversity to absorb some of the expected and also largely unpredictable effects of climate change. New roles for forest pathologists in the field of climate change are suggested.

Introduction

Climate warming has received considerable recent attention in public, policy, and scientific arenas. This topic offers considerable opportunities in several areas of forest pathology. This paper suggests some roles for forest pathologists in the fields of climate change and carbon sequestration.

Pathologists recognize the roles of the environment and pathogens operating together to result in disease of a host plant (i.e., the plant disease triangle). Weather or broader climate patterns can serve as important environmental factors where temperature and precipitation probably affect every plant disease in some manner. Many of these disease situations may be considered minor, with pathogens causing only subtle

changes to ecosystems. This paper does not treat such biotic diseases even though some may become more damaging to forests under changing climaticconditions. By contrast, altered climate can trigger adjustments in ranges of individual tree species or initiate entire regime shifts. Through pollen and macrofossil analyses, paleobotanists have documented many historic transformations in forest cover or composition (Heusser 1952). These shifts, which are sometimes relatively abrupt, are consistently assumed to be driven by changes in climate. How would they appear on the landscape to an observer? I suggest that they appear as forest declines and widespread bark beetle outbreaks in situations where the forest is altered irrevocably and does not return to its original composition.
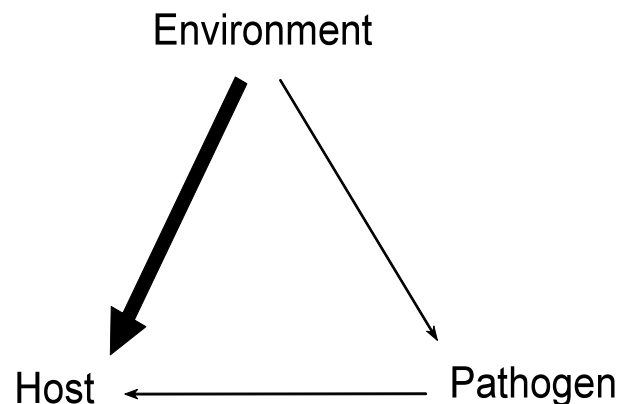
Figure 1—The plant disease triangle, where the environment, or more specifically climate, is a dominant factor in influencing the health of host trees compared to the more subtle effects of pathogens. Forest trees that regenerated and grew to canopy status in a previous climate regime can become maladapted as the climate changes. In severe cases, they suffer from widespread mortality interpreted as forest decline.

Forest Declines, The Face Of Climate Change

Forest declines are often described as widespread spatially, long-term temporally, and involve complex factors (Manion and Lachance 1992). Complexity often leads to an unresolved etiology; some reports even suggest that forest declines cannot be elucidated. The cause of some declines is still a mystery (e.g., ohia decline in Hawaii (Hodges et al. 1986)), but in others

(e.g., pole blight (Leaphart and Stage 1971) and yellow-cedar decline (Hennon et al. 2007)) there is now a basic understanding of the primary and secondary causal factors.

Many, but not all, forest declines exemplify the collapse of tree species initiated by climate change. Various opinions and concepts of forest declines are given in Manion and Lachance's (1992) book on forest declines. Of particular help is the sorting of various abiotic and biotic factors as predisposing, inciting, and contributing. Predisposing factors are longer-term and establish conditions for trees to become vulnerable to the other two factors. Inciting factors are near-term and cause acute stress to trees, which are then attacked by contributing factors, usually secondary insects and pathogens. Climate can function as both predisposing and inciting factors, as indicated by pole blight of western white pine (Leaphart and Stage 1971), a new aspen decline (Worrall et al. 2007), and yellow-cedar decline (Hennon et al. 2007). Each of these examples also highlights the importance of landscape and site factors, usually as predisposing factors, in dictating where the declines occur. For example, a drought between 1916 and 1940 was the inciting factor in pole blight, but only white pines in a particular age class (young, dense pole stage) growing on soils with poor moisture holding capacity were killed (Leaphart and Stage 1971).

Climate Involvement In Yellow-Cedar Decline

Research on yellow-cedar decline has taken several lines of inquiry to resolve the cause of tree death. A deductive approach was used to list and evaluate the most likely causes of mortality, with an emphasis on pathogens. Inductive approaches have been employed to study various site and landscape variables associated with the presence or absence of yellow-cedar decline (D'Amore and Hennon 2005), and then use these as clues to construct working hypotheses for further evaluation (Hennon et al. 2007). The inductive approach has helped clarify the varying importance for each of the environmental and spatial factors related to this forest decline. Clues from spatial (associations with snow) and temporal patterns (onset of decline at the end of the Little Ice Age) implicated climate as a factor. The factors and the interplay of climate in yellow-cedar decline are organized below using Manion and Lachance (1992) terminology.

Predisposing factors, longer-term and related to climate--Local geomorphology (especially slope and contributing hydrologic area) and mineral soil properties combined with a shift in climate to cooler and wetter conditions about 5,000 years B.P. to alter decomposition and produce wetter soils. Peat accumulated, sometimes reaching more than 2m deep, which further inhibited soil drainage. Poorly drained soils caused reduced forest productivity and the inherent open canopy conditions, as well as root systems growing closer to the soil surface to avoid anoxia.

Cooler and wetter climate about 5,000 years B.P.
→Peat accumulation
   →Wet soils
      →Shallow roots
         →Canopy openings initially driven
           by hydrology, then by tree death

Inciting factors, short-term seasonal weather patterns (Warm late winters and early springs)—Climate warming initiated the end of the Little Ice Age and accelerated in the latter half of the 1900s. Recent research with University of Alaska Fairbanks confirms the 20th century trend of warmer winters, reduced snow, but persistence of frequent freezing events in late winter and early spring (Beier et al., in press).

Warm late winters
→Dehardening of yellow-cedar tissues
→Reduced snow
→Removal of protective insulation covering roots
   Sudden freezing weather
    →Proximate injury in dehardened, exposed roots

Contributing factors—Biotic factors are necessary as the coup de grâce (complete death in a declining tree) for some forest declines, but are only minor or even unnecessary in yellow-cedar decline. Yellow-cedar trees die from the primary stress (i.e., freezing) whether or not either of these agents is present. At best, colonization by these agents speeds the death of trees that would otherwise succumb in a few years.

   Armillaria spp.—colonize roots, root collar, and lower bole of declining trees

   Phloeosinus cuppressi—colonize phloem in bole of declining trees

Mitigating factors—Knowledge of pre-disposing site factors and the interaction with inciting climate factors can be used to make decisions about where to plant and favor yellow-cedar in the future. Yellow-cedar is generally healthy and not declining in areas that have

snow pack that persists into March or April and also on well drained soils where roots grow deeply enough to escape damaging ($<-5°C$) temperatures. This information is the basis for guidance to resource managers about where to favor yellow-cedar in the future.

Well drained soils—yellow-cedar roots escape freezing injury and shade maintains cool soils to slow dehardening.

Snow—provides insulation that buffers against damaging soil temperatures.

Thus, climate is involved as predisposing and inciting factors in yellow-cedar decline. This understanding would not have been possible without first uncovering the actual factors that are influenced by climate (i.e., wet soils, shallow rooting, canopy exposure) and focused work on tree injury (Schaberg et al. 2005, 2008). In other words, interpretations about any possible role of climate must come after there is a basic understanding of the various factors involved in a forest decline.

When Pathogens Do Matter

Pathogens are not typically primary factors in forest declines, but they can be. The "mal del cipres" forest decline in Patagonia region of Argentina of Austrocedrus is an example of a decline problem that was very difficult to resolve until a team of pathologists uncovered a hard-to-isolate *Phytophthora* species (Hansen and Greslebin 2007). This fungus-like organism appears to be the overriding cause of the cypress mortality. Thus, mal del cipres has now been shown to be a more simple biotic disease. The cause of this problem would not have been elucidated if pathologists had not been involved in the etiology of this forest decline.

Carbon Sequestration & Wood Deterioration

Knowledge of wood decomposition is a major gap (and error) in models of carbon cycling in forests. For example, most carbon models do not account for differences in the fate of carbon between white rot and brown rot decomposition. The coastal old-growth forests of Alaska and British Columbia are assumed to be in equilibrium with primary production. This assumes that wood deterioration releases carbon to the atmosphere as CO_2 and to streams as dissolved carbon. This assumption would not create a significant error if

most wood carbon decomposed through the white rot process. Most conifer wood in these forests decomposes through the brown rot process, led predominantly by *Fomitopsis pinicola*, which leaves most lignin in the soil in a modified form (Edmonds 1999, Gilbertson and Ryvarden 1986). Since fire is absent, the lignin (about 20 to 25 percent of wood carbon is lignin) in advanced brown cubical rot eventually becomes incorporated into the soil as a stable form of carbon. Old-growth forests may thus operate as a carbon sink with approximately one fourth of their wood carbon accumulating in soils.

Internal decay of live trees is often not included in carbon models, although pathologists know that great differences exist among tree species and by tree or stand age. Some of the best information available comes from the classic defect or cull studies performed by pathologists. For example, Kimmey (1956) estimated that approximately 31 percent of the gross volume in the forests in southeast Alaska was in the form of heart rot, but stands less than 100 years old are nearly defect free. He reported vastly higher decay rates in live western hemlock, mainly in the form of white rot, than in live Sitka spruce, which was mainly brown rot. Models of carbon cycling will continue to have large errors until this type of information is incorporated.

Pathologists are also familiar with natural heartwood compounds that limit wood decay in live trees. The long term persistence of wood carbon from trees that have this natural chemical defense (e.g., redwoods, cedars, junipers) may be several orders of magnitude longer than other tree species, regardless of whether trees are left to die in forests or used as wood products. The simple decision to favor a cedar over a hemlock in a managed forest could yield a doubling or tripling of sequestered carbon due to the unique heartwood compounds in the decay-resistant tree species.

New Forest Management Goals In A Changing Climate: Resilience

Rapidly changing climate regimes create new challenges for forest management. The old concept of returning forests to previous (e.g., "pre-settlement") conditions now seems inappropriate, yet many ecologists and forest resource managers continue to use restoration as a management goal. Tree species that regenerated and grew to their canopy status can become poorly adapted when climate changes. The most precarious situations are those at or near

environmental and biological thresholds. For example, the mean winter temperature of 0°C where yellow-cedar occurs in Alaska is at the rain-snow threshold. These thresholds may represent a more precarious situation than at the limits of a species' range. Forest declines are a manifestation of this maladaptation, and suggest specific physiological vulnerabilities for the declining tree species. Considerable knowledge on the autecology of tree species will be needed to predict which tree species may be maladapted given certain climate projections. Where that knowledge of species autecology is incomplete, scientists and managers will need to rely on new principles to guide them given these high levels of uncertainty.

A new forest health concept is needed to provide a vision of how to structure forests in an uncertain future threatened by climate change. Creating forests with resilience (Folk et al. 2004) as a primary goal may be the best way to deal with uncertainty, or even the expected loss of one or more species. A resilient forest would have enough species diversity (i.e., redundancy) to absorb species loss from the sometimes unpredictable effects of climate change. The forest declines inform us that some tree species will be more vulnerable than others to new climate conditions. We should not expect to witness intact forest community assemblages moving north or to higher elevation. Changes will likely come one species at a time (Webb 1987), with the most vulnerable affected first. Thus, redundancy of tree species is desirable insurance against the losses from climate change.

Climate scientists warn that climate shifts will occur at a pace faster than many species can adapt or spread. This prediction will force policy makers and managers into a philosophical decision: will they passively observe climate effects such as forest declines and then act to manage the aftermath? Or, will they actively move species, i.e., "facilitated migration," anticipating where tree species will be best adapted to new climates? Regardless of their actions, forest pathologists will be called upon to evaluate newly emerging forest declines, and probably also new disease situations where tree species are moved into new environments.

A Conservation Strategy For A Climate-Sensitive Species

Our yellow-cedar team is working with some of the concepts mentioned above to develop a conservation strategy for yellow-cedar in Alaska in the context of a warmer climate (Hennon et al. 2007). Our strategy partitions the landscape into areas that are favorable and unfavorable for yellow-cedar. Yellow-cedar was once well adapted in the entire region, but due to warmer winters, less snow, and continuing spring freezing, yellow-cedar is now maladapted on wet soils at low elevation. We suggest to harvest the dead cedars from some of these areas to offset logging of yellow-cedar where it is still well-adapted. Also, we advise resource managers to favor other tree species in the declining forests, and essentially give up on yellow-cedar there. The more challenging part of this strategy is to identify portions of the landscape that are favorable for yellow-cedar now, and will continue to be favorable well into the future as the climate continues to warm. To do this, we are blending distribution maps of yellow-cedar decline, performing yellow-cedar habitat modeling, and constructing new snow models based on climate projections. This approach is similar to "shifting climatic envelopes" (Hamann and Wang 2006), except in this case of yellow-cedar, we are able to include specific climate factors that cause tree injury. This specific information from our research on yellow-cedar should produce better predictive models than the standard method of relying on climate variables associated with current tree species' distributions. Our project will identify areas where we advocate active yellow-cedar management in the form of planting and thinning. Some of this long-term suitable habitat is already occupied by yellow-cedar; but in others, we will recommend moving yellow-cedar to areas of high snow or on well drained soils by transporting the species over short or perhaps even long distances consistent with the idea of "facilitated migration".

Conclusions

Forest declines and wood deterioration offer opportunities for forest pathologists to serve important roles in climate and carbon sequestration issues. Some existing forest declines can be used as examples of how climate change will affect forests. New forest decline situations will emerge as forest trees become stressed by changing climatic conditions; each of these will need to be investigated by pathologists and other scientists to sort out etiology and then interpret the importance of climate as a factor. Lessons from forest declines can be applied to new concepts that will guide policy makers and resource managers as they prepare for dramatic changes in forest ecosystems in a warming climate.

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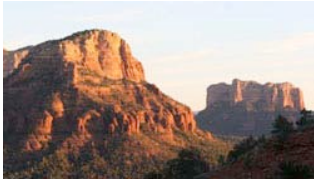
Aspen Decline on the Coconino National Forest

Mary Lou Fairweather[1], Brian W. Geils[2] and Mike Manthei[3]

Abstract

An accelerated decline of aspen occurred across the Coconino National Forest, in northern Arizona, following a frost event in June 1999, and a long-term drought that included an extremely dry and warm period from 2001 through 2002, and bouts of defoliation by the western tent caterpillar in 2004, 2005, and 2007. From 2003 to 2007, we monitored aspen mortality and regeneration, and measured associated stand and site variables on randomly-selected sites of the Coconino National Forest where aerial survey had detected dieback or decline. Year of death was observed or estimated since 2000. Xerophytic forests sustained greater mortality than mesophytic forests. Aspen on low-elevation xeric sites (<7500 ft) sustained 95% mortality since 2000. Mid-elevation sites (7500–8500 ft) lost 61% of aspen stems during the same time period; mortality is expected to continue in these sites because some remaining trees have 70 to 90% crown dieback. Less aspen mortality (16%) was observed on more mesic high-elevation sites (>8500 ft). Low-elevation sites are located on northerly aspects while mid- and high-elevation sites are located on various aspects. Overall, diameter distributions showed mortality was not skewed to any particular size class, however, trees with diameters >9 inches generally took longer to die than smaller size classes. Several insects and pathogens were associated with aspen mortality but appeared to be acting as secondary agents on stressed trees. Although aspen ramet production occurred to some degree on all sites with the death of mature trees, aspen sprouts were nearly nonexistent by the summer of 2007 due to browsing by elk and deer. None of the sites studied are grazed currently by domestic cattle. Widespread mortality of mature aspen trees, chronic browsing by ungulates, and advanced conifer reproduction is expected to result in rapid vegetation change of many ecologically unique and important sites.

Introduction

Severe dieback and mortality of aspen occurred over the past several years on the Coconino National Forest (NF) due to impacts from drought, frost, and insect defoliation (USDA Forest Service 2006). In 1999 frost

damage occurred on approximately 6,000 acres following a severe June snowstorm (Fairweather 1999, USDA Forest Service 2000), resulting in early defoliation and death of twigs and stems. Damage was greatest in aspen clones whose leaves were succulent and expanding. Although many afflicted clones produced new leaves, damage was severe enough to be detected during aerial detection surveys later that summer (USDA Forest Service 2000). Greater crown dieback and mortality of aspen occurred in 2002 and 2003, during one of the driest periods on record. Aerial surveyors mapped over 5,000 acres of aspen dieback and mortality in 2002 and 2003 (USDA Forest Service 2003, USDA Forest Service 2004). Defoliation by the western tent caterpillar (Malacosoma californicum) was observed in 2004, 2005, and 2007, including areas previously affected by drought and/or frost.

This paper presents the results of a monitoring project in which permanent plots were established to track dieback and mortality of aspen on the Coconino NF. The objectives were to describe the timing and distribution of mortality, assess regeneration, and identify stand and site factors related to dieback and mortality of aspen.

Methods

Areas with aspen crown dieback and mortality were randomly selected from the 2002 Arizona Zone-Forest Health Protection Office aerial detection survey. A series of 1/20th acre permanent plots were established on a grid of one plot for every five acres of an aspen delineated site. Plot data included slope, aspect, and elevation. Trees >5 inches diameter at breast height (dbh) were recorded. Information included species, status (live or dead), dbh, height, crown rating, and presence and severity of damage agents.
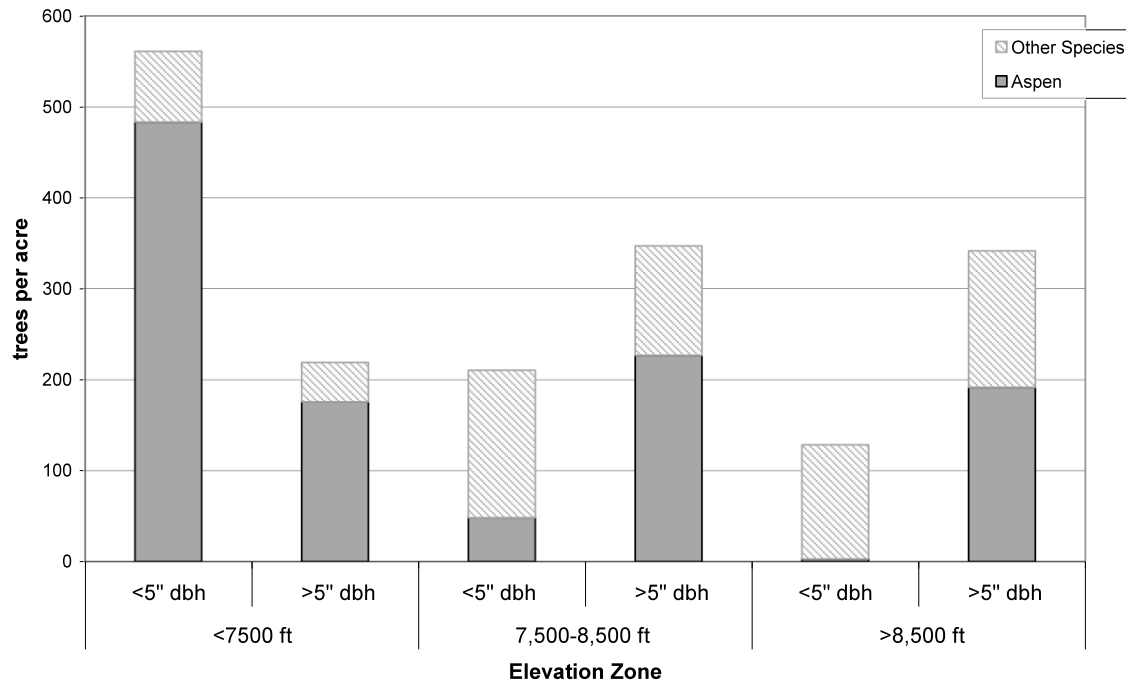
Figure 1—Stand composition in trees per acre, 2000.

During plot establishment, estimated year of death was determined by presence of leaves, buds, flowers, fine branches, and bark condition. Categories for year of death included current year, 1 year, 2 years, and more than 2 years.

In order to quantify branch dieback and decreases in leaf area of a live tree, percent live crown remaining was estimated for all live aspen. For example, a rating of 10% was given to a tree that had 90% recent branch mortality. All live aspen trees >5 inches dbh were tagged. Damage agents included borers, defoliators, canker fungi, decay fungi and ungulate damage. Tree heights were measured on the first two live aspen in each plot. Regeneration on 1/100th acre subplots was recorded by species, size, status, and damaging agent. Plots were reexamined through 2007.

Results

Site conditions were reconstructed to the year 2000 based on year-of-death estimates for 83 plots, and categorized into three elevational zones: <7500 ft, 7500–8500 ft, and >8500 ft (Figures 1 and 2). Aspen

was the dominant forest species in affected stands across all elevational zones; compared to other tree species, aspen trees (>5 inches dbh) were the most numerous and comprised the greatest volume. For all tree species combined, basal area increased with increasing elevation, ranging from approximately 110 to 240 ft2/ac. However, aspen basal area was nearly constant at 100 ft2/ac, regardless of elevation. Due to advanced decay, stem age could not be determined for most sampled trees; but those that were readable showed stand age to be about 100 years. Associate tree species at low-elevation sites include ponderosa pine and Gambel oak, at mid-elevation sites were ponderosa pine, Gambel oak, southwestern white pine, and Douglas fir, and at high-elevation sites were ponderosa pine, Douglas fir, white pine, white fir, subalpine fir, and spruce. Aspen was dominant in the understory only in low-elevation sites. There were 58 aspen snags per acre in low-elevation sites in 2000, while mid- and high-elevation sites had 15 and 14 snags per acre, respectively. Low-elevation, aspen sites were mostly restricted to northerly aspects; at higher elevations aspen sites occurred on all aspects.
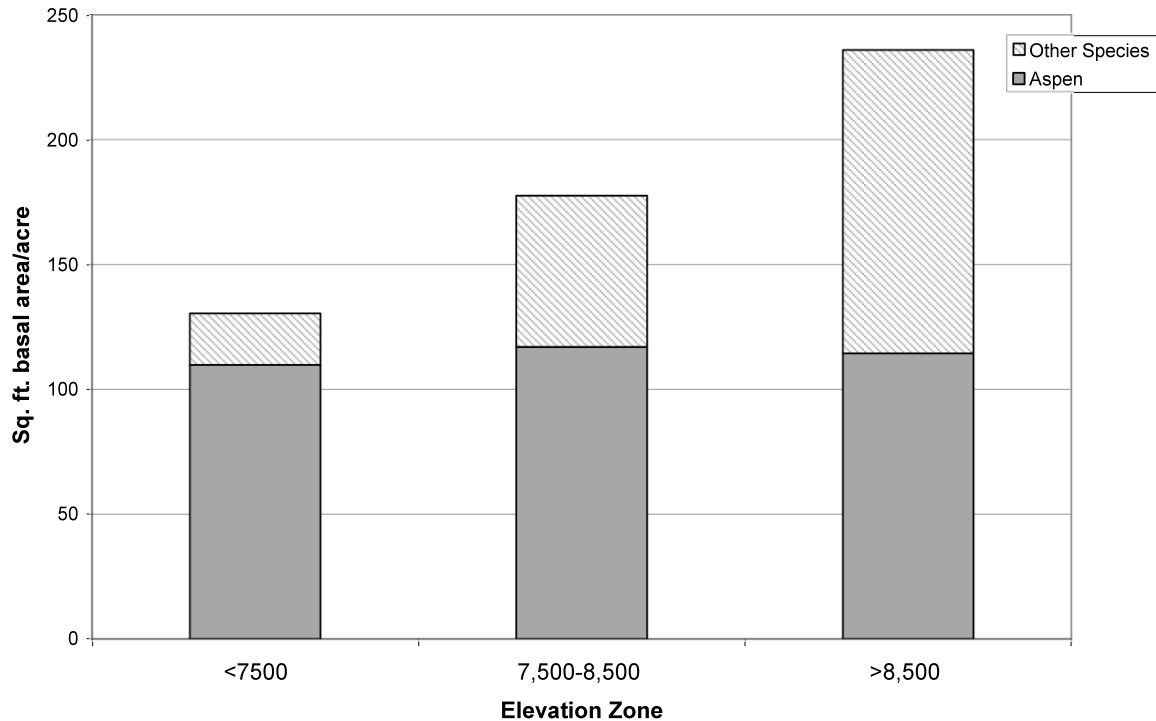
Figure 2—Stand composition in square ft. of basal area per acre, for live trees >5" dbh, 2000.

Stand composition changed considerably in affected stands since 2000 (Figure 3 and 4), particularly at low-elevation sites where aspen was completely removed from several stands and nearly eliminated from most others. Affected sites <7500 ft elevation experienced on average >95% aspen mortality by the summer of 2007; sites 7500– 8500 ft had 61% mortality; and 16% mortality was observed >8500 ft (Figure 5). The annual mortality rate was near 20% from 2001 through 2004 in the low-elevation sites (Figure 6).

In general, mortality within each elevation zone was distributed among tree-size classes with little striking trends (Figure 7). At lower elevations, a higher percentage of saplings (<5 inches dbh) died than larger trees, while a lower percentage of mid-sized trees (5–9 inches dbh) died than smaller or larger trees. In mid-elevation sites, a different pattern was observed; percent mortality decreased by size class but with only a difference of 20% between the smallest and largest size classes. At the high-elevation sites, the difference in percent mortality between mid-sized trees and the larger trees was only about 10%; on these sites, there were only 3 saplings/ac to begin with and none died.

Overall, there was a linear relationship between crown rating and mortality, and no threshold crown rating determined likelihood of survival. Larger trees (>9 inch dbh) took longer to die than smaller trees. Trees with severe branch dieback and/or reduced foliage often produced enough green foliage to be rated as "live" rather than "dead", even though death was probably imminent. Where low crown ratings were associated with western tent caterpillar activity, in the absence of prior decline, trees survived and the crown rating improved after insect populations declined. However, defoliation likely hastened death of many, previously stressed trees.

Several insects and pathogens were associated with aspen mortality but appeared to be acting as secondary agents on stressed trees (see Hinds 1985, Jones et al 1985). These agents include *Cytospora* canker (*Valsa sordida*), bronze poplar borer (*Agrilus liragus*), aspen bark beetles (*Trypophloeus populi* and *Procryphalus mucronatus*), poplar borer (*Sperda calcarata*) and a clearwing moth (*Paranthrene robinae*). *Cytospora* canker was present to some degree on all sites, but the occurrence of other agents varied by location.
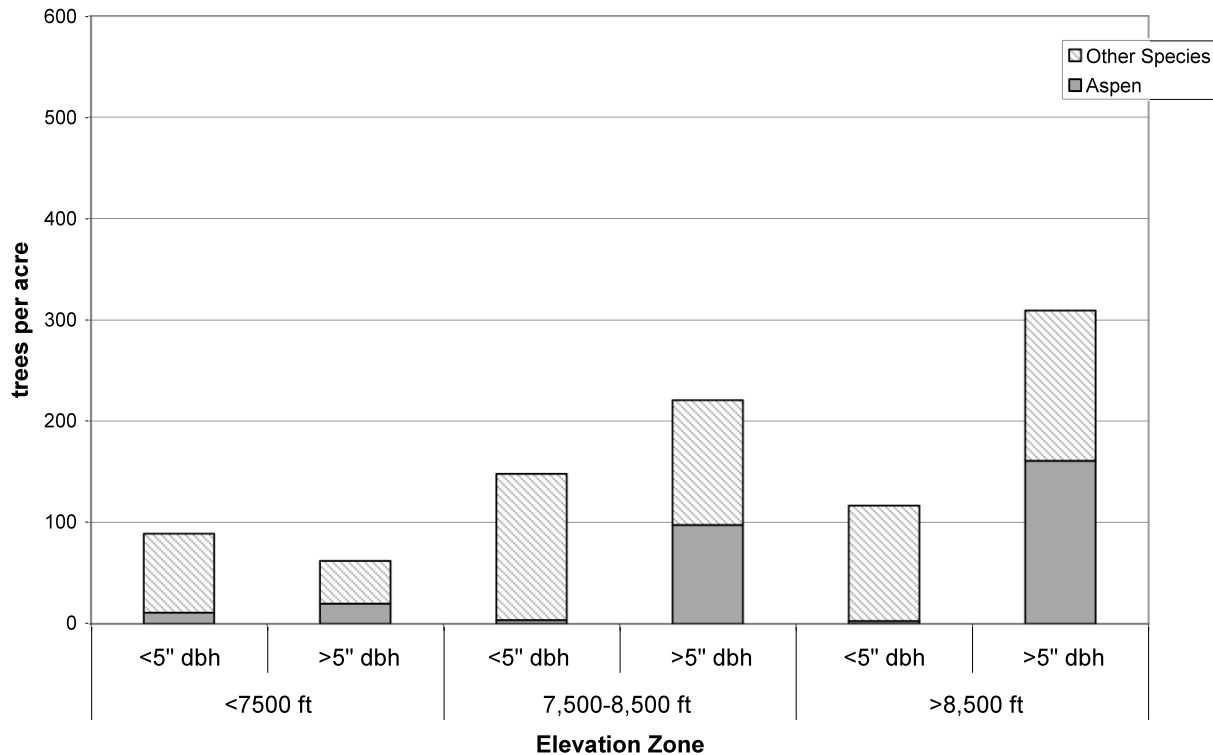
Figure 3—Stand composition in trees per acre in sites affected by aspen decline, 2007.

Western tent caterpillars severely defoliated aspen throughout northern Arizona in 2004, 2005, and 2007 and were the most consistent biotic agent contributing to aspen decline.

Reproduction by suckering was highest within the first few years of plot establishment and monitoring (Figure 8), reaching an average of 1,000 stems/ac on low- and high-elevation sites. Site averages varied from as low as 10 stems/ac to over 4,000 stems/ac. Nearly all ramets were browsed on an annual basis across the Forest and none exceeded a height of 2 ft.

Discussion

Aspen mortality observed over the past 7 years on the Coconino NF resembled a decline syndrome (Manion1991) with various but identifiable predisposing, inciting, and contributing factors including site, climate, weather, insects, and pathogens. We detected substantial mortality as early as 2000 (58 snags in low- elevation sites), likely resulting from a frost event in 1999 (Fairweather 1999) and the stress of long-term drought beginning

in 1996. Mortality rates stayed high through the more severe drought years of 2002–2003 and the western tent caterpillar activity of 2004 to 2005. By 2007, affected sites <7500 ft elevation sustained 95% mortality; 61% mortality was observed at 7500–8500 ft; and 16% mortality in sites >8500 ft. This is much greater than damage reported by others. Gitlin et al (2006) found approximately 15% mortality associated with the 2002–2003 drought in a random sample of affected and non-affected aspen stands >7500 ft on the San Francisco Peaks, Coconino NF. Our greater mortality levels are likely due to several factors: observing cumulative mortality since 2000; including sites <7500 ft; monitoring sites detected to be in decline based on aerial detection surveys rather than including non-impacted sites; and capturing the slower mortality rates of larger trees by surveying sites through the summer of 2007. A recent study in southern Colorado reported 32% mortality from 2004 to 2006 (Worrall et al 2007), which was linked to a particularly dry, hot spring and early summer of 2005. Similar to the Coconino NF, aspen mortality was found to be inversely related to elevation.
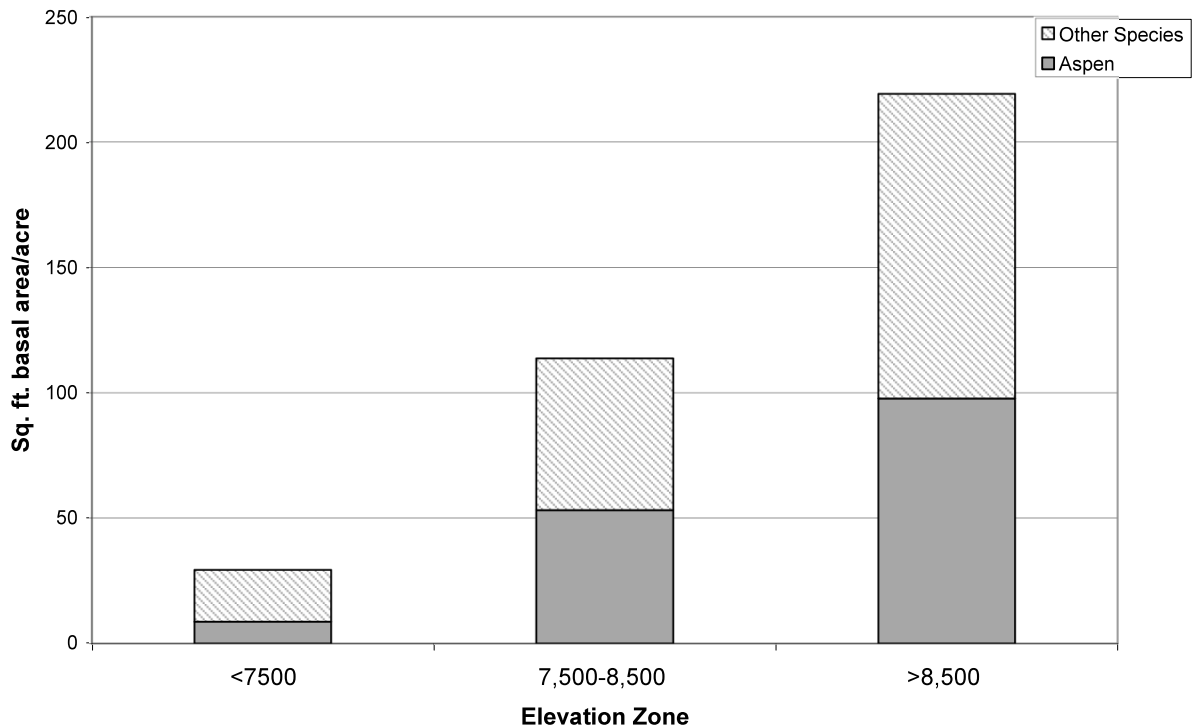
Figure 4—Stand composition in square ft. of basal area per acre, trees >5" dbh, in sites affected by aspen decline, 2007.

Aspen was not the only species affected by the severe drought of 2002–2003, as hundreds of thousands of acres of lower elevation piñon pine and ponderosa pine perished across northern Arizona; however, a different pattern of mortality occurred between conifer and aspen.).

Stressed conifers either survived the drought or died as the result of bark beetle attack. The full impacts of the drought (and frost event) on aspen took much longer, 2–3 years, to materialize. A similar phenomenon was observed in Alberta and Saskatchewan, Canada, following the severe drought of 2001–2003 (Hogg et al 2006).

The decline of aspen reported here is distinct from the successional replacement of aspen by conifers. A decrease in the area dominated by aspen due to succession was addressed in Amendment 11 of the Coconino NF Forest Plan (USDA Forest Service 1996), but this recent and widespread mortality increases concern for the future of aspen on the Forest. The structural change in aspen forests is believed to be the result of: 1) altered fire regimes and livestock grazing since European settlement which promoted succession to conifer forests (USDA Forest Service 1994, Dahms and Geils 1997, Cocke et al. 2005); and 2) heavy browsing of aspen suckers by large ungulates, especially Rocky Mountain elk (Shepperd and Fairweather 1994, Rolf 2001). Merriam (1890) describes the flora and fauna of the San Francisco Peaks, including distributions and correlations with climate, elevation and aspect. He characterizes aspen as common in forests above 8200 ft but descending "considerably" on mesic aspects and forming large groves of tall stems where fire had removed the conifers. Among the identified mammals, he lists the black-tailed deer as abundant, the mountain sheep as present; but gives no mention to any elk. Cocke et al (2005) report a 456% increase in basal area since 1876 in aspen forests on the San Francisco Peaks. The density of aspen in these forests decreased while conifer species increased. They report little evidence of aspen recruitment since the 1940s. Although some land managers think future large-scale fires will ncrease aspen regeneration across the landscape, it appears browsing impacts will limit any success.

Aspen typically sprout profusely following disturbance (upwards of 30,000 stems/ac), but the number of suckers produced by a clone maybe related to the levels of carbohydrate reserves and hormonal growth promoters in the roots (DeByle et al 1985). The substantial sprouting observed in the early stages of decline in some aspen sites in this study may have allowed for the production of a young stand of trees. Unfortunately, browsing by elk and deer (none of these sites were grazed by cattle), eliminated the potential.

Young aspen trees typically grow an average of 3 to 6 ft the first 2 years and a total of 9 to 15 ft in 5 years (Shepperd 1993 and Miller 1996), but in the 4 years since plot installation not a single sprout has grown over 2 ft in height. A similar scenario has occurred following disturbance by harvest or fire in aspen type across the Forest, where regeneration of aspen by suckering has not been successful unless well protected by fencing (Shepperd and Fairweather 1994, Rolf 2001, Bailey and Whitham 2002 and 2003). Aspen regeneration outside of protective fences is nearly non-existent—typically restricted to rocky areas or in steep terrain where ungulate activity is minimal.

Browsing of aspen regeneration from large ungulates, particularly elk has been a major concern since the 1960s when regeneration treatments failed because aspen sprouts were consumed in spite of attempts at protection with fencing suitable for cattle (Rolf 2001). Since the mid-1980s, forest managers have built 6½ ft tall fences around aspen regeneration to prevent elk browsing; but these fences are expensive to install and maintain. Although originally intended as a temporary measure until the trees obtained sufficient height to escape browse, it was soon realized that fences need to remain for a longer period (Shepperd and Fairweather 1994, Bailey and Whitham 2002). Rocky Mountain elk are the primary browser, and were introduced into the region after Merriam's elk was extirpated in the early 1900s. Although Merriam's elk were mainly

limited to the White Mountains of eastern Arizona, and the Mogollon and Sacramento Mountains of New Mexico, Rocky Mountain elk are now present in great abundance throughout Arizona (Truett 1996, Heffelfinger et al 2002). Although Merriam's was considered a subspecies of Rocky Mountain elk, recent research suggests it may be a different species altogether (Heffelfinger et al 2002).
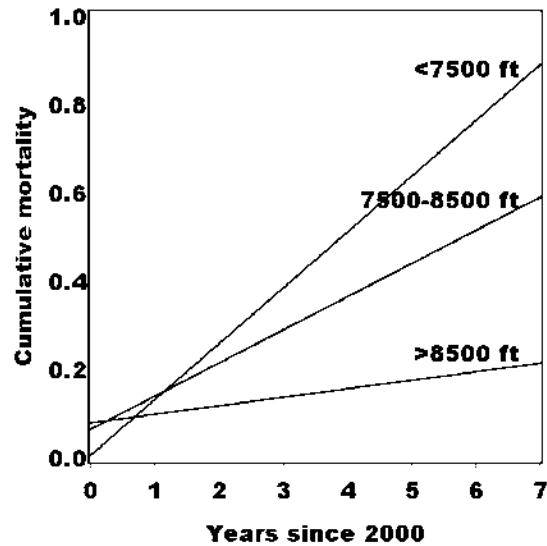

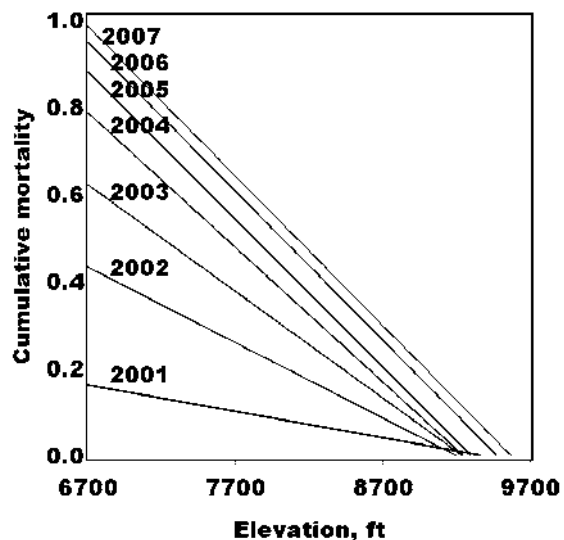**Figure 5**—Cumulative mortality of aspen stems >5" dbh from 2000 to 2007 by elevation zone.

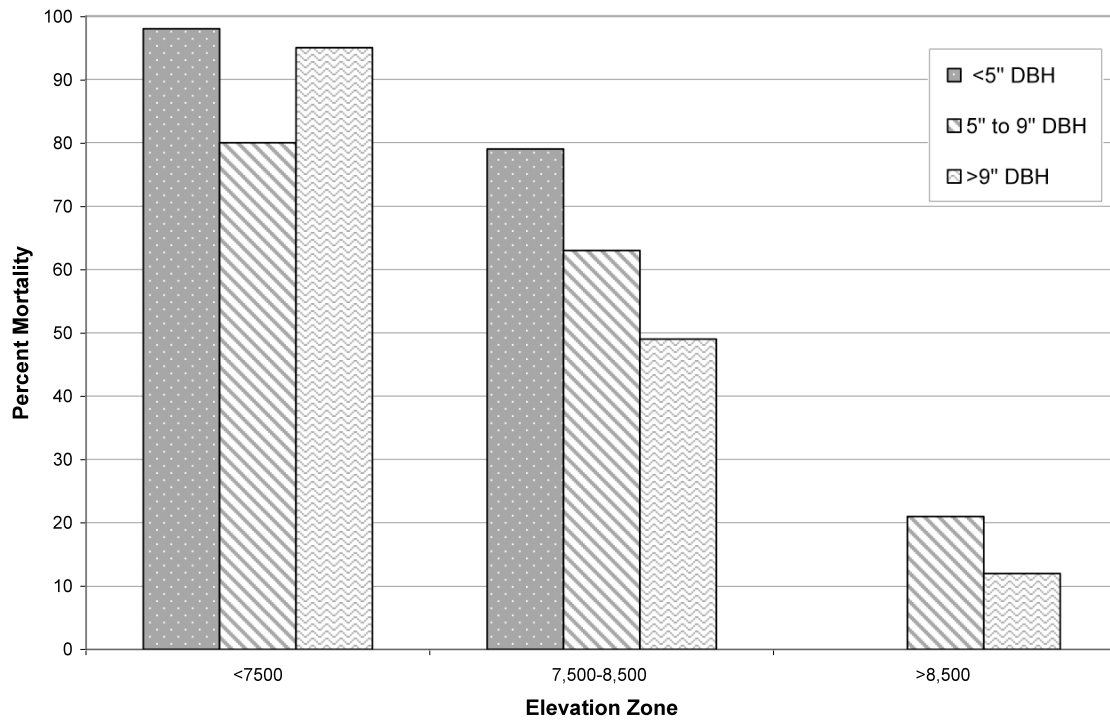**Figure 6**—Cumulative mortality of aspen stems >5" dbh from 2001 to 2007 by elevation.

Figure 7. Cumulative percent mortality of aspen stems by size class and elevation zone, 2007.
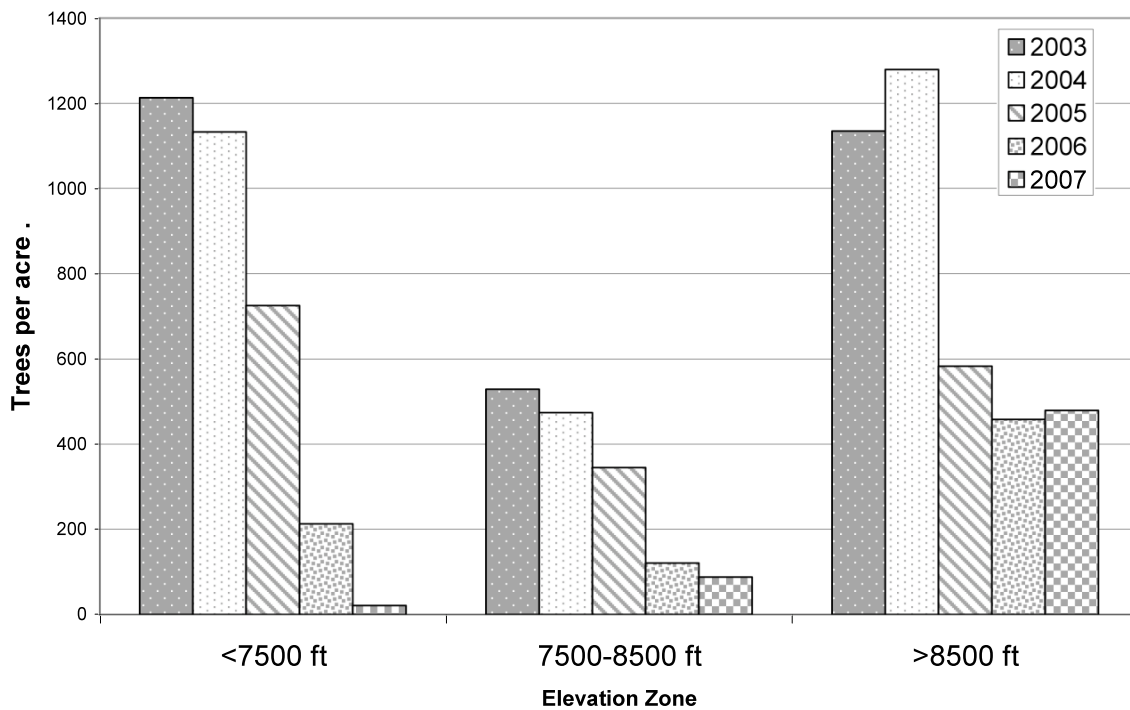

Figure 8—Aspen regeneration (stems less than 4.5 ft. high) by year and elevation zone.

Elk damage aspen in three ways: they browse new shoots, rub flexible saplings with antlers and gnaw or bark trees for phloem. The browsing of shoots is so prolific that it is rare to see these ramets survive more than a couple of years, and that only happens when the mature tree is still living. Barking and rubbing has been positively correlated to damage by secondary pathogenic fungi causing stem cankers and decay (DeByle 1985, Hinds 1985) that play a role in the death of trees. Stem decay fungi contribute to stem instability as the trees grow (Hinds 1985).

Permanent exclusion fences are required to allow for successful reproduction following silvicultural treatments or fire (Rolf 2001, Bailey and Whitham 2003, Fairweather et al. 2006). Coupled with the inability of aspen regeneration to survive browsing, the mortality events are resulting in virtually complete loss of aspen in many stands.

The mortality of aspen on the Coconino NF over a period of several years is similar to that observed recently in other parts of North America (Frey et al 2005, Worrall et al 2007), which were also linked to severe drought and defoliation. In Colorado, Worrall et al (2007) named the phenomena as Sudden Aspen Decline (SAD) to distinguish it from the gradual succession of aspen to conifer forests. A decline disease is defined as an interaction of three or more sets of abiotic and biotic factors to produce a gradual general deterioration often ending in tree death (Manion 1991). Although there are many possible factors, none can be shown to produce decline individually. The factors involved in declines are grouped into predisposing, inciting and contributing categories. Predisposing factors alter the trees' ability to withstand or respond to injury-inducing agents. In this case, affected stands were composed of mature aspen that were succeeding to conifer with little chance of recruitment. Inciting factors are short-term biotic or abiotic events that often result in branch dieback. Examples of incitants here include late spring frost, drought, and defoliators. Contributing factors are environmental factors or biotic agents that are able to act more aggressively on stressed trees. On the Coconino NF, canker fungi, wood boring insects, and bark beetles were some of the contributing factors associated with the death of

mature trees. Elk are contributing to the decline of aspen on the Forest, as they are not allowing newly sprouted ramets to grow and mature. Although death of large numbers of mature trees is a good thing that is required for proper development of the next generation of aspen, we observed no chance of survival of new sprouts.

Conclusion

Rapid and abundant mortality of aspen occurred on more xeric, aspen-dominated sites of the Coconino NF in response to several abiotic and biotic factors. Low and mid-elevation sites sustained 95% and 61% mortality, respectively. Affects of aspect were related to elevation; low-elevation sites are located on northerly aspects and aspect was variable for sites above 7500 ft. Larger trees took longer to die than smaller size classes. Although regeneration occurred following the death of overstory trees on some sites, successful regeneration of aspen is doubtful due to widespread browsing of young trees by elk and deer. Extensive mortality of mature aspen trees, chronic browsing by ungulates, and advanced conifer reproduction is expected to result in rapid vegetation change of many ecologically unique and important sites.

Acknowledgements

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Sudden Aspen Decline in Southwest Colorado: Site and Stand Factors and a Hypothesis on Etiology

Jim Worrall[1], Leanne Egeland[1], Tom Eager[1], Roy Mask[1], Erik Johnson[2], Phil Kemp[3] and Wayne Shepperd[4]

Abstract

An initial assessment of rapid dieback and mortality of aspen in southwest Colorado suggests that it represents a decline disease incited by acute, warm drought. Predisposing factors include low elevation, south and southwest aspects, droughty soils, open stands, and physiological maturity. Contributing factors include Cytospora canker, two bark beetles, poplar borer, and bronze poplar borer. Because this is a true decline disease distinct from "aspen decline" as often discussed in the literature, we refer to it as sudden aspen decline. There has been little regeneration response to overstory loss.

Introduction

Since about 2004, rapid dieback and mortality of trembling aspen stems have been increasingly observed in Colorado. By 2006 it became clear that the damage was substantial and that it was not the type of mortality typically seen in older aspen stands. The results of our initial assessment of the problem are presented here. Based on this assessment, we use the name sudden aspen decline (SAD) for this phenomonen.

Since the WIFDWC meeting, much of the data in this presentation has been published (Worrall and others 2008). Therefore, the information here will be abbreviated, with emphasis on aspects not presented in the publication.

Methods

This study was conducted in southwest Colorado, where aspen spans an elevation range of about 2100 to 3300 m. Detailed observations were made of sites with recent aspen dieback and mortality in the area. To analyze landscape patterns of damage, we used geographic information from the 2006 aerial survey on aspen damage, together with the aspen cover type from databases of Rocky Mountain Region, USDA Forest Service. Analyses were restricted to Forest Service land, where continuous cover-type information was available. The Grand Mesa, Uncompahgre and Gunnison National Forests, and the Mancos-Dolores District of the San Juan National Forest were analyzed. Healthy aspen was considered to be the aspen cover type after removing damaged aspen (from the aerial survey). Digital Elevation Model (DEM) data, including elevation, aspect and slope, were compared between healthy and damaged aspen.

Stand data, using standard methods, were taken from 31 stands on two sites (Haycamp Point and Turkey Knolls) on the Mancos-Dolores Ranger District east of Dolores. These stands were pure or nearly pure aspen. Data presented here include only aspen. Per stand examination protocol, stems $\geq$ 12.7 cm DBH are considered part of the overstory; smaller stems are considered regeneration.

Results

Before 2005, aspen damage noted in aerial survey totaled less than 10,000 ha each year in Colorado. By 2006 that figure had reached 58,374 ha, of which 56,091 was recorded as "aspen decline." Even taking into account the increased attention paid to aspen, clearly there was a rapid and substantial increase in damage area.

Ground Observations

Ground observations revealed the following:

Recent and rapid—The great majority of the damage appeared to be very recent and had occurred rapidly. Some stands were nearly completely dead, with intact bark and fine twigs still on the stem, almost as if they were dormant. Others had similarly recent mortality along with stems that had dieback and/or thin foliage.

Landscape scale—Where damage was common, extensive landscapes, including many stands, were similarly affected. Damaged areas of several kilometers in extent have been seen.

Sparse regeneration—Many stands had understory stems (< 12.7 cm DBH), but generally these were sparse and most were large enough that they appeared to have arisen before the current mortality event.

Biotic agents—Agents that typically cause the most mortality of mature aspen in Colorado, most notably sooty-bark canker (Hinds 1964; Juzwik and others 1978), were observed infrequently. Instead, a group of secondary, stress-related agents was found associated with dieback and mortality. Typically a few of them were found together in each deteriorating stand, but in various combinations. They were *Cytospora* canker, poplar borer (*Saperda calcarata*), bronze poplar borer (*Agrilus liragus*), and two species of bark beetles (*Trypophloeus populi* and *Procryphalus mucronatus*).

Effect of prior management—In damaged areas, patches that were cut in the past consistently stood out as solid green, healthy areas, often surrounded by dead and dying residual, unmanaged overstory.

Geographic Analysis

Elevation—The frequency distribution of aspen vs. elevation forms regular bell curves on all four forests that were studied (figure 1). The damaged aspen (expressed as a proportion of the cover type) was generally highest at low elevations and decreased to nearly zero at high elevations. However, the Grand Mesa was anomalous in this regard, with a peak of mortality near the upper end of the elevational range.

The Grand Mesa is capped with a thick layer of basalt derived from lava flows (figure 2). Around the cap is a "landslide bench" derived from broken fragments of basalt (Yeend 1969). The landslide bench is a coarse jumble of basalt fragments with poor soil development. The anomalous high-elevation aspen mortality on the Grand Mesa occurs precisely on this landslide bench, most likely as a result of its droughty soil characteristics.

Aspect—Aspen cover type was most frequent on north aspects at low elevation and south aspects at high elevation. Damaged aspen tended to be most frequent on south and southwest aspects at all elevations.

Slope—On all forests and all elevation bands, damaged aspen consistently occurred on flatter slopes than did healthy aspen.

Stand Data

In the 31 measured stands, mortality ranged from 0 to 100% of standing trees. Findings were as follows:

Mortality associated with open stands—Stands of high density (up to 1200 stems ha-1) had low levels of mortality. Open stands (as low as 200 stems ha-1) had more variable levels of mortality. As a result, mortality was significantly and negatively correlated with density (R = -0.38, P = 0.033.
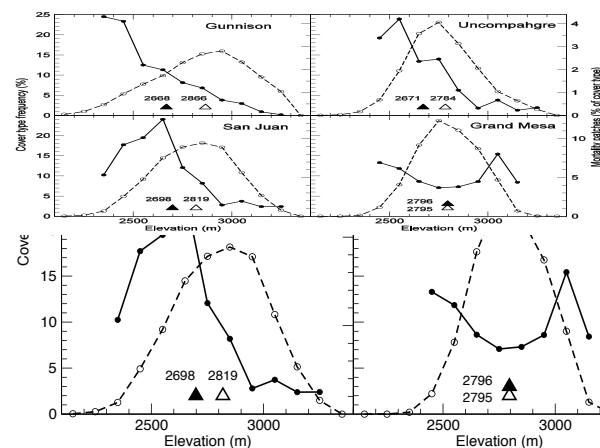
Figure 1—Elevation distributions of aspen cover type (dashed lines) and damage (solid lines; as a percentage of cover type) on four national forests. Triangles indicate mean elevations of healthy (hollow) and damaged (solid) aspen).
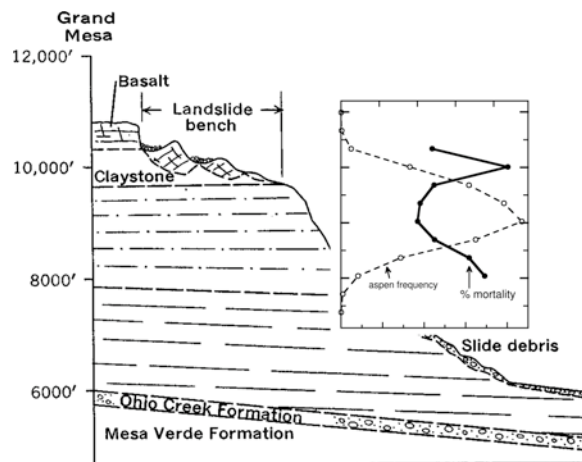
Figure 2—Geological profile of the Grand Mesa, showing the landslide bench, made of coarse fragments broken from the basalt cap of the Mesa (Yeend 1969). The anomalous high-elevation peak of mortality in the superimposed graph of aspen damage frequency (derived from figure 1 and scaled to the diagram) occurs on the landslide bench.

Larger stems affected—Overall, current mortality (recently dead and dying) was skewed to large trees (>30 cm DBH). Size of affected trees, relative to remaining healthy stems, was strongly correlated with incidence of current mortality in the stand (R = 0.64, P < 0.001).

Sparse regeneration—Regeneration averaged about 2500 stems ha-1, and did not increase with mortality. That level of regeneration is the average expected under intact, undisturbed stands in southwest Colorado; after a clearcut, the average density of suckers is 76,600 ha-1 (Crouch 1983).

Discussion

Cause—Colorado and much of the interior West recently experienced an acute drought that has been termed a "global-change-type drought" because it was exacerbated by extremely high temperatures (Breshears and others 2005). Aspen is intolerant of drought (Niinemets & Valladares 2006), and drought and high temperatures have been tied to deterioration of aspen often in the past (Frey and others 2004; Gitlin and others 2006; Shields & Bockheim 1981). In our study, the preponderance of damage at low elevations and on south and southwest aspects is consistent with moisture stress as an important cause. In addition, the association of stress-related biotic agents with the mortality strongly indicates the existence of a prior stressor. We suggest that this syndrome is consistent

with the features of a true decline disease (Guyon 2006; Manion 1991; Manion & LaChance 1992) and propose the following etiology in that context:

Predisposing factors—Site factors that predisposed aspen to inciting factors include low elevations, south and southwest aspects, and droughty soil conditions. Stand factors that increased susceptibility include physiological maturity (large stem size) and low density.

Inciting factors—The acute drought with high temperatures during the growing season was the key event that triggered SAD.

Contributing factors—The biotic agents associated with SAD are secondary, stress-related agents. They seem to be interchangeable and can each play a similar role in different areas that are affected. This is one of the key features of a true decline.
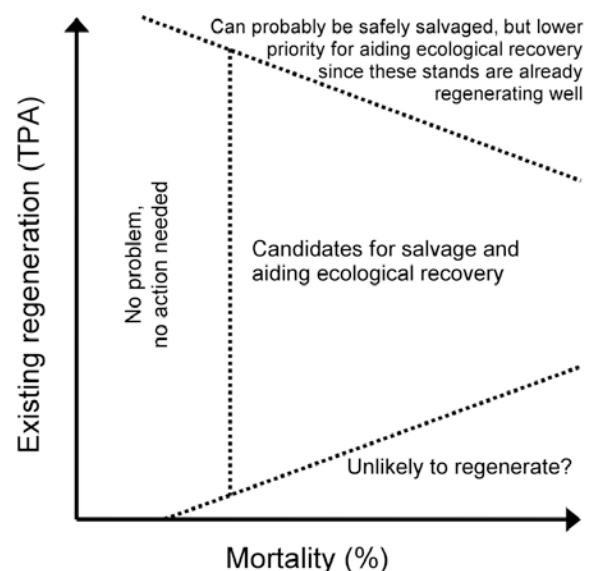
Figure 3—Conceptual diagram for selecting stands for treatment based on incidence of mortality and density of existing regeneration. Quantification of the axes is best done by silviculturists with survey data in hand.

Sad vs. Aspen decline—"Aspen decline," as generally described, refers to a long-term loss of aspen cover type due to succession under an altered fire regime, often exacerbated by ungulate herbivory of suckers (Kulakowski and others 2004; Ripple & Larsen 2000; Rogers 2002). Sudden aspen decline is distinct from that process for a number of reasons: (a) although both may result in succession to other vegetation types,

aspen decline is driven by succession whereas SAD is driven by damage to aspen; (b) SAD occurs on a landscape scale rather than on a stand scale; (c) SAD is rapid, resulting in large areas of mortality in just a few years; and (d) the mortality agents associated with SAD are different from those that typically kill mature aspen in Colorado.

Regeneration—Evidence from two sites suggests that there is often little or no regeneration response in aspen stands that are being opened up by SAD. This is confirmed by more recent results over a wider area (unpublished). Apparently, in many stands the cumulative stress has exceeded the regenerative capacity of root systems. The result may be conversion to other vegetation types, particularly if ungulates remove the sparse regeneration that is present.

Management—Although we have ample experience with management and regeneration of healthy aspen stands, we have little experience with stands deteriorating from SAD. We may be able to provide an abrubt disturbance through cutting or burning that will stimulate optimal suckering by the remaining root system, maximizing the likelihood of recovery. However, there is probably a point of deterioration, beyond which suckering will be inadequate, regardless of disturbance. A conceptual diagram (figure 3) has been offered to managers to use in prioritizing stands for treatment. We will learn from the results of such treatment and adapt management accordingly.

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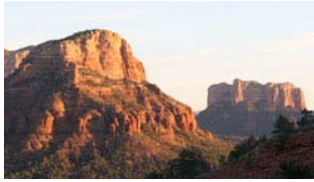
Survivor Aspen: Can We Predict Who Will Be Voted Off The Island?
F.A. Baker[1] and J. D. Shaw[2]

Abstract

During the past few years, aspen have been dying at rates that appear to exceed normal rates. We believe that this mortality should not be unexpected, given the severe drought of the past 10 years. We examine the literature and FIA data and identify several factors that indicate such mortality should be expected.

Aspen, *Populus tremuloides*, is a tremendously successful species. It produces abundant seeds (sexual reproduction) and suckers from its roots (asexual reproduction). In the western US, establishment by seed is thought to be rare, thus suckering is usually considered the most important means of reproduction. Maximum sucker production, however, requires disturbance. Fire and harvesting are the most common disturbance types, but wildfire control and a limited market for aspen products in the west has limited the amount of disturbance in aspen stands in the last 100 years.

This 100-year time frame is important, because aspen is a relatively short- lived species. Baker (1925) stated that "even on the best sites rotations of more than 80 years will be infrequent", and, "On poorer sites decay is the limiting factor, and trees should seldom be grown for longer than 70 years." Krebill (1972) noted for Utah that "senility" occurs with aspen at about 120 years of age. and Meinecke (1929) states that "wild aspen forest as a whole does not reach much beyond 130 years." Schier (1975) suggests that aspen matures in 80-100 years and declines rapidly with decreasing age. DeByle (1989) reports that trees older than 100 years are common, but stands begin to break up between 120 and 140 years. Jones (1967), however, states that aspen lives much longer and grows more

[1]Fred Baker is Associate Professor, Department of Wildland Resources and Ecology Center, Utah State University, Logan, UT. fred.baker@usu.edu.
[2]John Shaw is with Forest Inventory and Analysis, USDA Forest Servie, Rocky Mountain Research Station, Ogden, UT. jdshaw@fs.fed.us.

slowly in the southern Rocky Mountains than in the east. He notes working in two stands older than 200 years. This apparent contradiction maybe due in part to the author's concept of, as succinctly stated by Meineke (1929) "the wild aspen forest as a whole". Mueggler (1989) concludes that aspen stands mature between 60 and 80 years, and deteriorate "rather rapidly" after about 120 years. With the exception of Jones' observation, these estimates of the demise of aspen stands coincide with the time since effective fire control.

Mueggler (1989) found that 94% of randomly selected aspen stands were mature or overmature. We used the USDA Forest Service Forest Inventory and Analysis (FIA) data to examine the age of aspen stands in Arizona, Colorado and Utah. Most of the aspen in these areas is older than Baker's (1925) suggested maximum rotation age of 80 years (Figure 1), and many are older than 110 years, well beyond the rotation ages recommended by Baker (1925).

Mortality is a constant factor in aspen stands, especially in their early years. It is stand mortality in later years that is of interest to this discussion. Trees begin dying at a relatively early age, and mortality continues (Figure 2). The mortality rate in the Site Index 57 stand was about 7% for a ten-year period. Mueggler (1994) reported that as stands aged, the number of trees decreased by at least 90% over 60 years (Figure 3). Mortality was greatest among the smaller diameter stems. Average mortality on the best site in Mueggler's (1989) study was 14% for a ten-year period. We are analyzing FIA data to characterize long term and recent aspen mortality rates. Shields and Bockheim (1981) characterized factors affecting longevity of aspen stands in the midwestern United States (Table 1. Their longevity index was calculated as the difference between predicted basal area and observed basal area; that is, stands with greater basal area would be expected to live longer. In general, they found environmental variables much more useful than soil variables. Of the environmental variables examined, mean annual temperature was the best predictor of aspen longevity. The variables correlated

with stand longevity suggest that aspen prefer cool, moist environments. If we consider weather in the Intermountain West during the past ten years, it has been much warmer and drier than normal, factors which should increase aspen mortality rates.
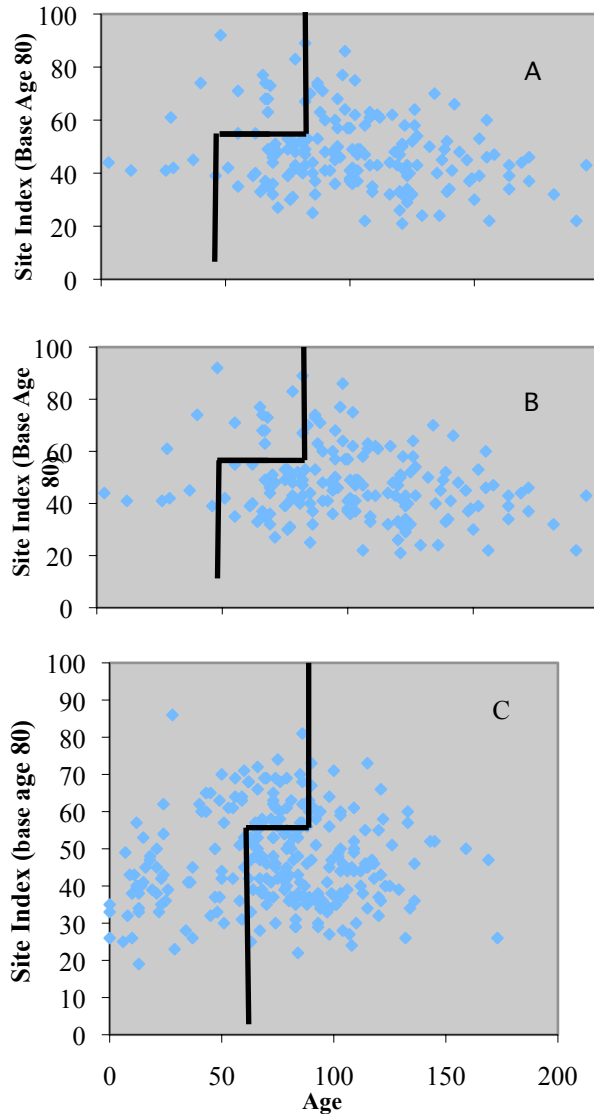
Figures 1—Age of aspen by site index in Arizona (A), Colorado (B), and Utah (C). Data taken from USDA Forest Service Forest Inventory and Analysis. Data are from aspen plot conditions. Some stands from Arizona and Colorado were omitted for consistency among graphs. Lines represent the rotation ages recommended by Baker (1925) for different site categories. Points below and to the right of the lines are overmature for their site quality.

Daniel (1980) attributes better aspen growth in the southwestern Rocky Mountains to a longer growing season and more reliable summer rain. Thus, there is at least some indication that these variables may be important in the western US.

At some point in time, perhaps driven by increased temperatures, decreased precipitation, or other factors, trees surrounding gaps caused by aspen mortality are no longer able to fill the gap. Stands in this stage are considered to be "deteriorating" (Fralish 1972; Shields and Bockheim 1981; Mueggler 1989),"breaking up" (Baker 1925; Fralish 1972), "senile" (Krebill 1972), rapidly seral (Harniss and Harper 1982), defective (Meineke 1929), declining (Pothier et al. 2004) or "decadent" (Weigle and Frothingham 1911; Baker 1925; Mueggler 1989). Unfortunately, all of these terms are subjective and difficult to quantify; i.e., we know it when we see it. Pothier et al. (2004) indicate that age alone is not a sufficient predictor of stand condition. They proposed using the ratio of dead to live basal area to identify the point at which a stand has a periodic annual increment equal to 0, and using this time as the onset of stand decline. This measure is good only for short term assessments. Perhaps it would be more useful to discuss stands as being overmature, which really is the cause of their decline. If we add their age and site index to the discussion, we can often gain an understanding of what is happening in a stand.

Variable	Correlation coefficient
ln mean annual temp C	-0.85
Latitude	0.78
ln mean August temp C	-0.77
ln mean September temp C	-0.77
ln mean July temp C	-0.65
Frost-free period (days)	-0.65
Ln mean June temp C	-0.62
Annual precipitation – potential evapotranspiration	0.36

Table 1—Variables correlated with aspen longevity index. From Shields and Bockheim, 1981.

The age at which aspen breakup occurs and the rate of mortality once the process begins have been attributed to many causes. Moisture stress may be the most important determinant of aspen longevity (Shields and Bockheim 1981). Given the recent drought in the Intermountain West, it seems logical to expect increasing mortality, especially in a population of stands older than optimal. Several authors note that
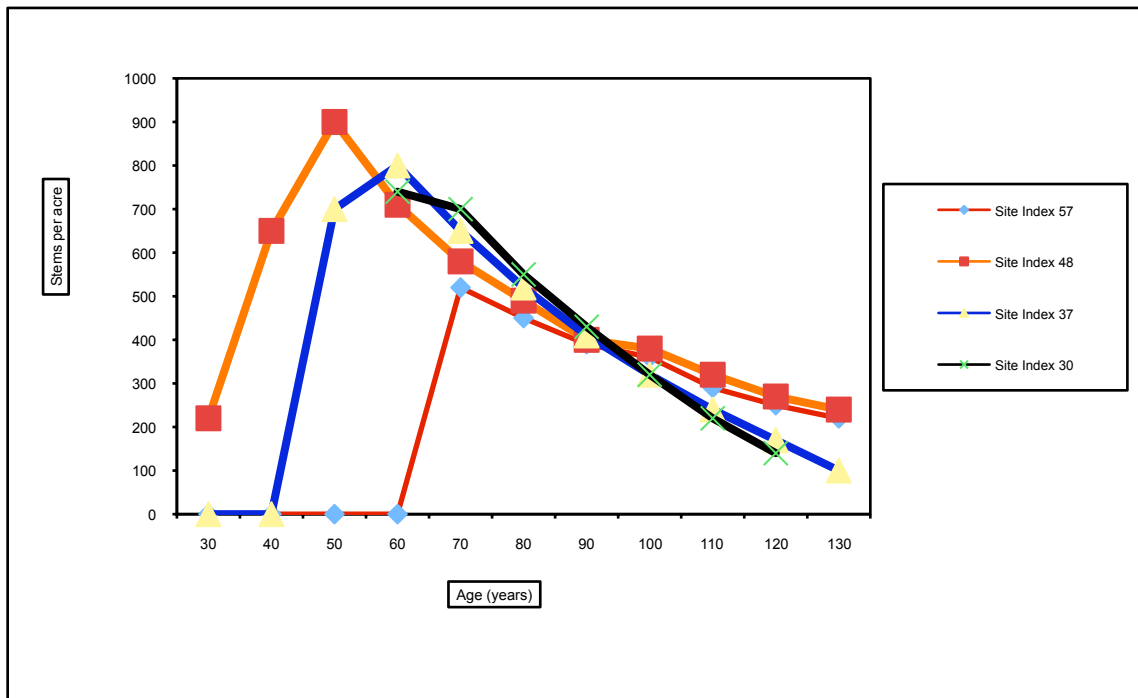
Figure 2—Aspen stand density over time on various sites. Data are fro trees >4" dbh from table 17 in Baker (1925). Site class converted to site index after Jones (1967).
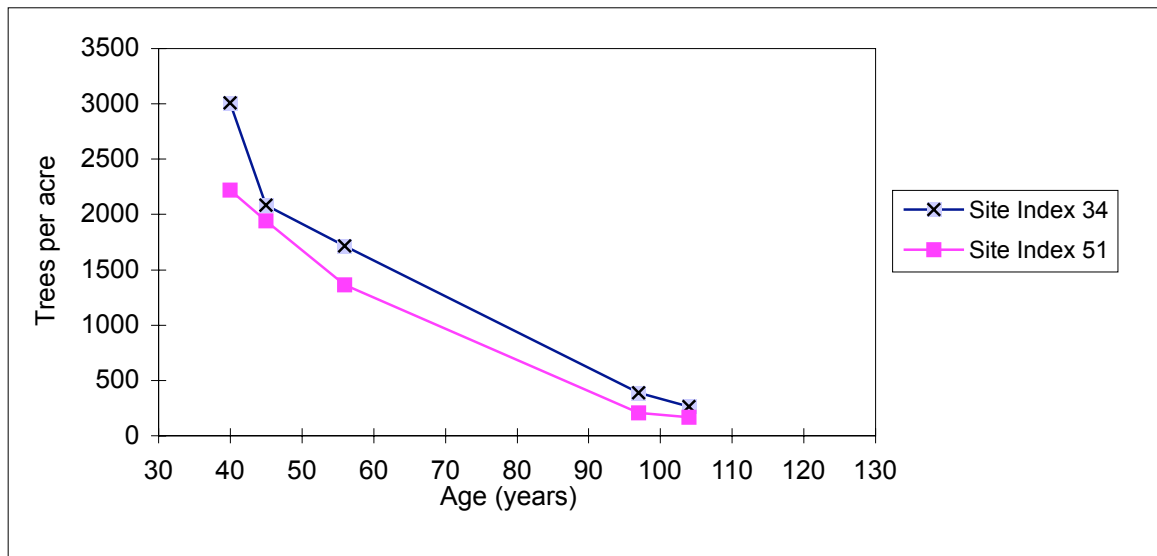
Figure 3—Decline in aspen density with age. From Mueggler, 1989.

once mortality starts, in an aspen stand, it proceeds quickly. Graham et al. 1963 noted that deterioration following the opening of aspen crown canopies can sometimes render. . "within a 5- or 10-year period a valuable stand . . to a worthless condition." Shields and Bockheim (1981) illustrate "a well -stocked stand" that was " reduced to a few diseased trees in as short a time as 6 years." Presumably, increased exposure to sunlight and associated sunscald results from opening

the canopy, and increases the susceptibility of remaining trees to harmful insects and diseases (Krebill 1972.) Decay caused by *Phellinus tremulae* (= *Fomes igniarius*) is a common feature of old aspen stands, and can also contribute to rapid stand deterioration (Weigle and Frothingham1911.) Aspens with trunks weakened by decay often snap when exposed to wind. In addition to these primary causes of mortality, several additional factors may have increased aspen mortality.

Defoliation by *Marsonnina* is quite common some years in the Intermountain West. Spring frosts have occurred in some areas as well; at least two spring frosts have damaged aspen in northern Utah in the past ten years. The effects of these events can be manifest for many years. For example, after forest tent caterpillar defoliation, Perala (1978) stated, "8-18 years following defoliation, [basal area mortality] was still elevated, peaking at 13 years." He goes on to state "This 13-year delay in peak mortality also seems reasonable, because defoliation exerts a stress on the tree that is not immediately fatal, but that subtly tips the balance of survival, perhaps much like the stress induced by hot, dry, Julys." Thus, we should expect to see aspen mortality continue for some years after precipitation returns to normal.

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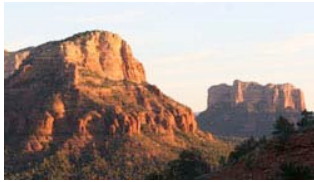
Under-Burning and Dwarf Mistletoe: Scorch 'N' Toe
David A. Conklin[1] and Brian W. Geil

Abstract

Relatively little quantitative information has been available on the effects of low-intensity fire (under-burning) on dwarf mistletoe. Here we summarize results from six operational prescribed under-burns in second-growth ponderosa pine in New Mexico (Conklin and Geils, in press). For 1585 trees on 14 plots, crown scorch, bole char, and DMR were significant predictors of post-burn survival in both logistic (3-yr response) and proportional hazard models (over 6 to 10 yrs). Reduction in DMR on surviving trees (scorch pruning) increased linearly with increasing scorch. Reduction in average DMR (sanitation) was observed on 12 plots, and increased with increasing average scorch and mean DMR before fire. A sanitation model estimates that an area with an initial DMR of 3.0 receiving 50% average scorch will have a reduction in mean DMR of 0.7. Longer-term monitoring indicates that burns generating 50% average scorch should provide about 10 years of control. Results indicate that under-burning can be a useful tool for management of dwarf mistletoe, given sufficient fire intensity.

Introduction

Increased understanding of natural fire regimes has led to interest in the use of low-intensity fire (under-burning) for management of dwarf mistletoes, especially in ponderosa pine forests of the interior West. However, relatively little quantitative information has been available on this topic. Early work by Koonce and Roth (1980) and Harrington and Hawksworth (1990) report a reducing effect of under-burning on dwarf mistletoes (*Arceuthobium*), but both are short-term studies involving relatively small samples. In this study, we continue work begun by Conklin and Armstrong (2001) for under-burning of ponderosa pine (*Pinus ponderosa var. scopulorum*) infected by southwestern dwarf mistletoe (*A. vaginatum sp. cryptopodum*). We relate tree survival following six prescribed under-burns to the risk

factors of initial dwarf mistletoe severity, crown scorch, and bole char; we quantifyreduction in mistletoe severity (scorch pruning) for surviving trees; and we assess potential reduction in average mistletoe severity (sanitation) across a range of burn intensities indicated by average crown scorch. In this paper, we summarize methods and results presented with greater detail in Conklin and Geils (in press) and expandfurther on implications for prescribed fire as a tool for managing dwarf mistletoe.

Field Methods

Study sites were mostly second-growth ponderosa pine (50 to 80 yrs old, with scattered older trees), typical of much of the accessible type in the Southwest. Prior to each fire, we installed rectangular plots in areas where a majority of trees were visibly-infected with dwarf mistletoe. Trees were rated for infection using the standard 6-class DMR system (Hawksworth 1977). Our sample included a total of 1585 ponderosa pine $\geq$ 4 inch dbh on 14 plots. The fires were operational Forest Service prescribed burns: four were about 200 ac and the others a few thousand acres in size. They were not "shaped" to kill or prune individual trees; the primary objective of each burn was fuels reduction. Most plots burned relatively uniformly and at intensities sufficient to generate some crown (needle) scorch on most trees. Post-burn DMRs were first taken 3 yrs after fire (by that time, scorched needles had fallen and branch mortality had stabilized). All DMRs, as well as crown scorch (percent of crown length affected, rated prior to flush of new growth) and bole char (relative severity class) ratings, were made by the first author (an attempt to maintain consistency in this long-term study). These burns were conducted between 1995 and 1999, and the sites have now been monitored for 8 to 12 years for subsequent mortality (dated to year of death) and changes in DMR.

Results And Discussion

Crown scorch increased with tree DMR on sites not recently thinned, but not on recently thinned sites. (On

recently thinned sites, scorch was strongly influenced by thinning slash and most heavily-infected trees had been cut.) Harrington and Hawksworth (1990) had similarly reported a pattern of increasing scorch with increasing tree DMR. The most likely explanation for this pattern involves simple difference in crown geometry; differences in fuel loading may also contribute.

Tree survival

High survival (3 yrs post-burn) was observed for trees with <90% crown (needle) scorch; survival at 90% scorch averaged about 65% but was quite variable across sites; survival at 100% scorch ranged from about 10% to 30%. (Note that several trees in our 100% scorch class "torched," so survival among those affected by convective heating alone was somewhat higher than indicated. Survival of 100%-scorched ponderosa pine has been reported in other studies, but has usually not been emphasized--and remains a surprising phenomenon to many). Only DMR 5 and 6 trees had decreased survival compared to uninfected trees. Bole char, although a more subjective measure than crown scorch or DMR, was clearly a strong and very useful predictor of mortality.
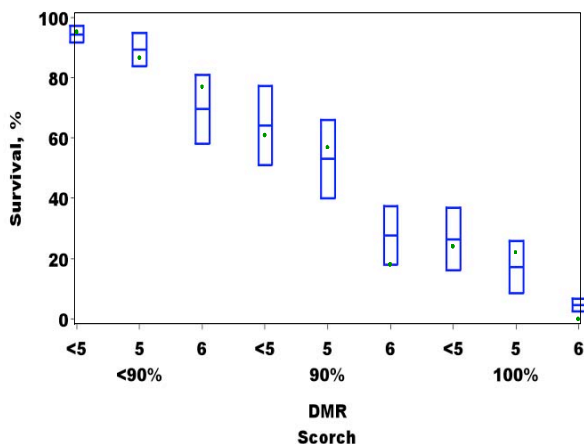
Figure 1—Tree survival 3 yrs post-burn by percent crown scorch and DMR (for average char rating); bars indicate expected tree survival (± 95% CL); dot marks observed percent of trees surviving.

Survival 3 yrs post-burn was modeled as a function of scorch, bole char, and DMR (figure 1). The model adequately describes the data (area under the ROC, c > 0.91). Note that tree size (dbh) was correlated with other risk factors (especially crown scorch), and (for simplicity and because of its rather negligible added

value) not entered into the logistic model; however, each of these fires tended to provide a thinning-from-below. A proportional hazards model quantifies relative risks of mortality due to scorch, char, and mistletoe over a 6 to 10-yr post burn period (Conklin and Geils in press). A DMR 5 tree had about the same risk as one with 90% scorch, and a DMR 6 tree had about three times the risk of a DMR 5. A drought in 2002 and 2003 increased risk (hazard ratio = 6.2). Crown scorch was not a risk factor after 3 years, while bole char continued to be significant 4 to 6 years post-burn (but not after 6 years). Heavy mistletoe was a significant risk factor in both the initial 3-yr period and later. We observed signs of bark beetle activity on about two-thirds of dead trees, both in the initial 3-yr period and later. Although bark beetles clearly increased total mortality, their role as either causal or opportunistic mortality agent could not be determined for many trees.

Except for the influence of heavy mistletoe infection, our observed survival (3 yrs post-burn) was similar to that in many other reports on post-fire ponderosa pine. Like Harrington and Hawksworth (1990), we found that dwarf mistletoe infection reduces survival of scorched trees. Not only do heavily-infected trees tend to receive more crown scorch, but, at a given level of scorch, they are more likely to die than other trees (presumably because of additional stress from mistletoe). However, this latter effect (described by our logistic model) is considerably less than suggested by Harrington and Hawksworth. More specifically, we found that this effect was significant only among DMR 5 and 6 trees (they assumed an increasing effect throughout DMR classes). In addition, our moderately-scorched DMR 5 and 6 trees had considerably higher survival rates than they reported. Their results (which they rightly described as "preliminary") were based on a single plot with 191 trees and determined only 1 yr post-burn.

Scorch pruning

Scorch pruning was assessed 3 yrs post burn, and based on surviving trees with an observed reduction in DMR (figure 2). The proportion of trees scorch-pruned increased with increasing scorch (R2 = 0.99). At 30% scorch, about one-fifth were scorch pruned; this increased to about one-half at 60% scorch. Above

40% scorch, an increasing proportion of trees decreased more than one DMR class ($R2 = 0.83$). Analyses of residuals demonstrate that these relations were largely independent of site, dbh, and initial DMR. Our estimate of scorch pruning is somewhat conservative because some "normal" intensification of mistletoe occurred between pre- and post-burn DMRs. Early reports (Koonce and Roth 1980; Harrington and Hawksworth 1990), due to their short-term nature, appear to have overestimated scorch pruning: we observed that usually about one-half the scorched portion of a ponderosa pine crown survives—along with any mistletoe present—due to bud survival.
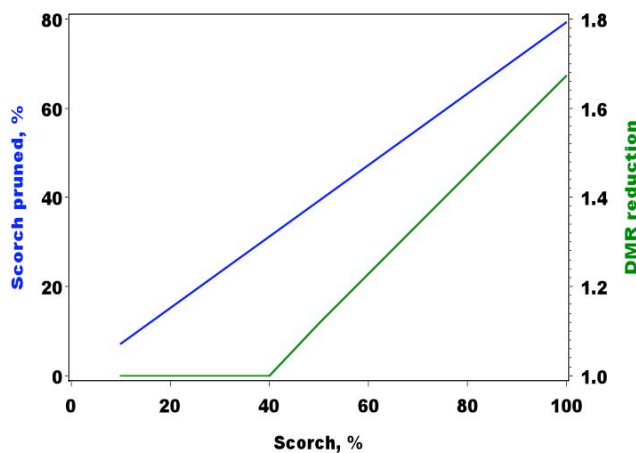
Figure 2—Proportion of initially infected, surviving trees with a reduction in DMR 3 yrs after fire (upper, blue line) and expected reduction in DMR of trees for which DMR decreased (lower, green line).

Conklin and Armstrong (2001) discuss an attempt to estimate reductions in mistletoe populations in scorched crowns—an initial estimate was later determined to be about 40% high! Note that virtually all reduction in tree DMR after fire resulted from "lifting" of the live crown (figure 3); heat (or smoke) alone appeared to have little effect on mistletoe shoots. In a broader sense, scorch pruning can be considered any fire-induced mortality of infected branches, regardless of effect on DMR. Some scorched trees whose crown geometry is not changed enough to reduce DMR will have delayed intensification; conversely, rebound (faster than normal intensification) will occur on some trees that show measurable reduction. On recently infected trees, scorch can eliminate latent infections (i.e., new infections without visible shoots), effectively preventing or delaying mistletoe development. Scorch

pruning will tend to improve host vigor, as well as reduce reproduction and spread of the parasite; however severely-scorched trees will experience growth loss, offsetting some of these gains. Long-term benefits/effects of scorch pruning will undoubtedly vary from tree to tree. Following an under-burn of sufficient intensity, it seems reasonable to expect that enough trees would benefit from scorch pruning to make a difference in the long-term development of the stand.

Figure 3—A portion of one study plot 6 yrs after fire. These trees each experienced 90 to 100% crown (needle) scorch. Three were lightly-infected before fire; two of them appear to have been completely sanitized.

Sanitation

Reduction in mean DMR (sanitation) was observed on 12 of 14 plots three years after fire. The two exceptions had < 10% average crown scorch. Reductions were a result of biased mortality (skewed toward heavily-infected trees) and scorch pruning; scorch pruning was the greater contributor on most plots. While reduction increased with increasing average crown scorch, its magnitude was also dependent on mistletoe severity (average DMR) before fire; at similar levels of scorch, plots with higher pre-burn DMR experienced greater reduction.

A sanitation model (figure 4), based on the combined tree-level logistic model for mortality and linear models for scorch pruning, determined expected plot-

level reductions that correlated well (r = 0.95) with observed reductions. Expected reductions were positive above DMR 1.0 and 25% average scorch and increased to 1.2 for an initial DMR of 4.5 and 85% average scorch.

The model estimates reduction of 0.7 with 50% average scorch and initial DMR of 3.0. Our sanitation model fit well with independent data from a prescribed fire in northern Arizona (observed reduction here 4 yrs post-burn was 0.5, equal to the model's expected reduction). Similar reductions in average DMR may be obtained in other ponderosa pine stands in the Southwest and Rocky Mountain regions after prescribed under-burning. The model should perform best for areas with relatively uniform fire intensity.
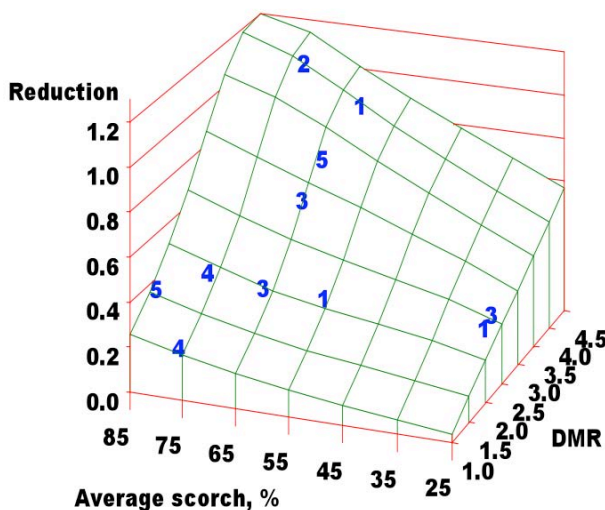
Figure 4—Expected reduction in average DMR (sanitation) for groups of trees 3 years after fire. Surface is smoothed estimate from combination of expected mortality (a logistic model) and expected scorch pruning. Labels indicate observed reduction, scorch, and DMR for 11 plots with adequate and even under-burning.

An alternative view of sanitation was developed for 3 sites with 10 years of observation (figure 5). Two of these fires (Road 145 and San Juan Mesa), each generating about 50% average scorch, provided about 10 years of control, i.e, 10 yrs of stand growth before average DMR returned to its pre-burn level. On the Blanco site, both the reduction in average DMR and the "years of control" may well approach the maximum sanitation possible without actively shaping

an under-burn to reduce mistletoe. Note that post-burn intensification of dwarf mistletoe was similar on each of these 3 sites, and similar to that observed on similar unburned plots in northern New Mexico.
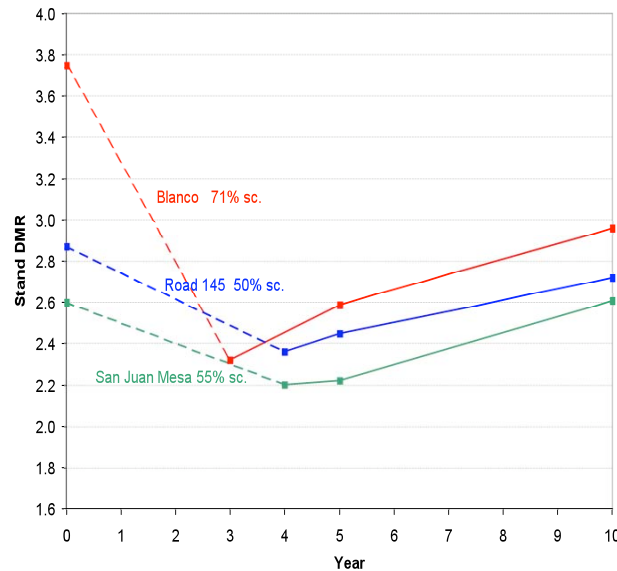
Figure 5—Observed change in average DMR for three under-burned sites monitored over a 10 yr period. Dashed lines represent observed reduction 3 yrs after fire (3 or 4 yrs after plot installation).

Additional Implications

The sanitation model provides expected reductions in average DMR within infested portions of stands. Because of spread from ballistic seed (with limited range), dwarf mistletoes usually have patchy distributions within stands and across larger landscapes. Study plots were intentionally located within mistletoe "infection centers" (rather than using a random or systematic design) to allow efficient sampling of infected trees. Although the model applies most directly at the patch scale, it should clearly have application at both the stand and landscape level.

The potential for sanitation in any given area necessarily depends on the proportion of the area infested—and the coverage and intensity of the fire. Most areas with "light" infestation include areas without mistletoe; clearly the potential for reduction (in absolute numerical terms) in these areas is less than in those with heavier, more extensive infestation. Also note that most infested stands, like our 14 plots, contain a broad range of dwarf mistletoe severity (DMR classes) on individual trees: the difference

between a "lightly-infested" and a "heavily-infested" stand is usually more a difference in size of the infection centers than in the proportion of lightly- and heavily-infected trees. (These spatial relations can be quantified with the alpha-statistic described by Robinson and Geils, 2006.) The potential for sanitation in recently thinned areas may be less than in others, since thinning itself often reduces average DMR. Nonetheless, under-burning in both lightly-infested and recently thinned areas can potentially set back mistletoe for several years.

We studied relatively young, relatively open stands because it is easier to quantify mistletoe infection and changes in DMR in these conditions than in older and/or denser stands. However, our data suggest that these results should apply in somewhat older and/or denser forest conditions. Each of these 6 burns was conducted during dormant seasons; higher mortality might be expected in summer burns. Because these fires were not intentionally shaped to discriminate against mistletoe, results should also generally apply to wildfires or Wildland Fire Use areas (or at least portions of these) that burn at relatively low intensity. Although its controlling effect is more modest than previously suggested, our results indicate that under-burning can be a viable tool for managing dwarf mistletoe, given sufficient fire intensity. Burns generating little or no crown scorch would have little or no effect on dwarf mistletoe. Furthermore, we suggest that burns of such low intensity will often have relatively little effect on fuels, fire hazard, or other forest conditions.

Under-burning appears to be a good ecological approach for managing dwarf mistletoes on many ponderosa pine sites in the Southwest and Interior West. Periodic/repeat burning should provide additional mistletoe reduction, depending upon stand age and how much the crowns were lifted by the initial burn. Mistletoe control is usually not the primary objective of prescribed burning, but it is one of many potential benefits and should be an important consideration in the development of burning plans. There is general agreement that a century of fire suppression/exclusion has been favorable to dwarf mistletoes.

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Permanent Plots for Measuring Spread and Impact of Douglas-Fir Dwarf Mistletoe in the Southern Oregon Cascades: Results of the Ten Year Remeasurement

Katy M. Mallams[1]

Abstract

Ten permanent plots to measure spread and impact of Douglas-fir dwarf mistletoe were installed in 1992 in the Southern Oregon Cascade Mountains on the Rogue River-Siskiyou and Umpqua National Forests. They were remeasured in 2002. Analysis of the data showed that after ten years heavily infected Douglas-firs had less growth and higher mortality than uninfected and lightly infected Douglas-firs. The impact was greatest in small diameter and understory Douglas-firs. The number of infected Douglas-firs increased substantially in ten years, but the majority of newly infected trees were within 25 feet of a previously infected tree. The data were used to validate growth and yield model predictions of Douglas-fir growth and mortality. At low levels of infection diameter growth in the plots was similar to model predictions. At high levels of infection there was less diameter growth in the plots than the model predicted. Predictions of mortality in the model were less than actually occurred in the plots in heavily infected and small trees.

Introduction

Douglas-fir dwarf mistletoe (*Arceuthobium douglasii Engelm*.) is a parasitic, flowering plant found almost exclusively on Douglas-firs (*Pseudotsuga menziesii* (Mirb) Franco). It is widespread on Douglas-firs in the Cascade and Siskiyou Mountain ranges of Southwest Oregon. Studies have shown that heavy infection of Douglas-fir dwarf mistletoe reduces growth of host trees and contributes to an increase in mortality (Mathiasen and others 1990, Filip and others 1991). Dwarf mistletoe infection also results in formation of brooms. These brooms, especially large ones, are widely used for nesting and hiding cover by a variety of wildlife species including the northern spotted owl (Hawksworth and Wiens 1996). Because Douglas-fir dwarf mistletoe is widespread and has significant impacts on its host, stand development and ecosystem functions, its impact is included in models used to predict stand development. The Forest Vegetation Simulator Model (FVS) is the most widely used growth and yield model in the Pacific Northwest Region. It incorporates the impacts of dwarf mistletoes using the Dwarf Mistletoe Impact Modeling System (DMIM), which runs in conjunction with FVS. Much of the information used to develop relationships between spread and intensification of dwarf mistletoes and tree growth and mortality in the model was derived from pre-existing data and "best guesses" (David 2005).

In 1990 a methodology for establishing permanent plots to measure the effects of dwarf mistletoe for use in vegetation simulation models was developed for the Forest Service, U.S. Department of Agriculture Forest Pest Management (FPM) by Mathiasen (1990). The field manual describing this method (Work Plan version 2.0, 1990) was used to install permanent plots in Douglas-fir dwarf mistletoe-infested mixed conifer stands in the southern Oregon Cascades. The data collected in these plots were used to validate FVS model projections of growth and mortality of Douglas-firs in dwarf mistletoe infested stands in Southwest Oregon.

Methods

In 1992 I installed ten plots in the southern Oregon Cascade Mountains on the Rogue River-Siskiyou and Umpqua National Forests. I followed the procedures developed by Mathiasen (1990) to install plots and collect data. The plots were located in mid-elevation mixed-conifer stands, both even and uneven-aged, in the white fir and western hemlock plant series. Five of ten plots had no evidence of logging. Five were salvage logged, thinned or selectively logged at some time before the plots were installed. The plots were square and varied in size from one third to one acre. Plot size was determined by the number of trees per acre. The corners and center of each plot were monumented with fiberglass pipes and tagged, painted reference trees.

[1]Katy M. Mallams is a Plant Pathologist with the Forest Service U.S. Department of Agriculture, Southwest Oregon Forest Insect and Disease Service Center, Central Point, OR. kmallams@fs.fed.us.

At each plot, slope, aspect, site index and plant association (Atzet and others 1996) were determined. All live trees greater than two inches diameter at 4.5 feet (dbh) were tagged. Data were collected on species, dbh, crown class and damage agents. The level of dwarf mistletoe was measured using the Broom Volume Rating system, a modification of the Hawksworth Six-Class Dwarf Mistletoe Rating System developed by Tinnin (1998) for rating Douglas-fir dwarf mistletoe-infected trees. Broom Volume Rating (BVR) is similar to the Dwarf Mistletoe Rating (DMR) in the Hawksworth system except that BVR is based on the total volume of each crown third occupied by mistletoe brooms, while DMR is based on the proportion of infected branches in each crown third. Tinnin compared the two methods and found that in a sample of 137 infected Douglas-firs the mean rating differed by only 0.2 classes (Tinnin 1998). Differences in ratings may result from underestimating the level of infection in the upper crown of large Douglas-firs using DMR, and overestimating the level of infection using BVR when one large broom occupies more than half a crown third.

Total height, height to the base of the live crown, breast height age and ten-year radial growth were measured in a subsample of live trees. Species, dbh and damage agents were recorded for all dead trees. In five plots the bearing and distance from the plot center to each tree were measured to create stem maps. Nine of the ten plots were remeasured in 2002. The Crooked Fire, part of the Tiller Complex on the Umpqua National Forest, burned through Plot 16 in September 2002. Due to safety restrictions remeasurement of this plot was delayed until June 2003.

Data from the plots were analyzed using Microsoft Office 2000 Excel Data Analysis Descriptive Statistics and SPSS 10.1 (SPSS 2000). Means and standard deviations were calculated for each measured variable. One-way analysis of variance (ANOVA) was used to compare statistical significance of differences in diameter growth, height growth and level of mortality among BVR levels. A P-value of 0.05 was used to determine significance. Pairwise multiple comparisons were made using least significant difference for equal variances and Tamhane's T2 tests for unequal variances.

Results And Discussion

Growth—After ten years, diameter growth of Douglas-firs in BVR classes 1 and 2 was similar to that of uninfected Douglas-firs. Diameter growth of Douglas-firs in BVR classes 3 and 4 was 31 percent less than uninfected Douglas-firs. Douglas-firs in BVR class 5 had 61 percent less diameter growth than uninfected Douglas-firs. Douglas-firs in BVR class 6 had 92 percent less diameter growth than uninfected Douglas-firs. The difference in diameter growth between Douglas-firs in BVR classes 5 and 6 and uninfected Douglas-firs was statistically significant (P = 0.000).

Infected Douglas-firs in every diameter class grew less in diameter than uninfected Douglas-firs. The difference in average diameter growth between infected and uninfected Douglas-firs was greatest in trees less than 12.0 inches dbh and greater than 35.9 inches dbh. However, the only statistically significant difference in diameter growth between infected and uninfected Douglas-firs in the same diameter class was in the 21.0 to 35.9 inch dbh class (P = 0.028).

After ten years height growth of Douglas-firs in BVR classes 1, 2 and 3 was similar to that of uninfected Douglas-firs. Douglas-firs in BVR classes 4 and 5 had 50 percent less height growth than uninfected Douglas-firs. Douglas-firs in BVR class 6 had no height growth. The difference in height growth between Douglas-firs in BVR class 6 and uninfected Douglas-firs was statistically significant (P = 0.003).

Mortality—Sixteen percent of the Douglas-firs died between 1992 and 2002. The level of mortality of Douglas-firs in BVR classes 1, 2, 3 and 4 was similar to mortality of uninfected Douglas-firs. Douglas-firs in BVR class 5 had 48 percent higher mortality than uninfected Douglas-firs. Douglas-firs in BVR class 6 had 80 percent higher mortality than uninfected Douglas-firs. The difference in the level of mortality between Douglas-firs in BVR class 6 and uninfected Douglas-firs was statistically significant (P = 0.000).

Small diameter, suppressed and intermediate infected Douglas-firs had much higher levels of mortality than large diameter, codominant and dominant infected Douglas-firs. The level of mortality, especially among small and suppressed Douglas-firs, may have been influenced by the preponderance of drier than average

years during the study period. Eight of ten years were drier than average. 1994 and 2001 were particularly dry years, with the weather station at nearby Prospect, Oregon recording only 78 percent of average precipitation.

Spread—The number of infected Douglas-firs increased 19 percent between 1992 and 2002. The greatest increase in new infections was in the 12 to 15.9 inch dbh, the greater than 35.9 inch dbh classes, and in the suppressed and intermediate crown classes. New infections in small diameter and understory Douglas-firs are to be expected given the ease with which dwarf mistletoe spreads from overstory to understory trees. The high percentage of new infections in large diameter Douglas-firs may have been an artifact of sample size. There were only five uninfected Douglas-firs greater than 35.9 inches in dbh in 1992. By 2002, two of them were infected.

In three of five stem mapped plots all Douglas-firs that were newly infected in 2002 were within 25 feet of a previously infected Douglas-fir. In the other two plots one newly infected Douglas-fir in each plot was approximately 40 feet from the nearest previously infected Douglas-firs. All other newly infected Douglas-firs in these two plots were within 25 feet of a previously infected Douglas-fir.

Intensification—Forty-five percent of Douglas-firs that were alive and infected in 1992 and 2002 did not change BVR class during the ten-year period. Twenty-eight percent increased by one BVR class. Ten percent increased by two BVR classes. Eight percent increased by 3 or 4 BVR classes. In most cases this large increase in BVR was due to death or breakage of tree tops, which decreased the proportion of live crown relative to brooms. BVR class decreased one to two classes in nine percent of the Douglas-firs. These trees may have lost infected limbs or grown faster in height than the dwarf mistletoe advanced upward in their crowns. The average change in BVR ranged from a decrease of 0.02 classes in the 21.0 to 35.9 inch dbh class, to an increase of 0.40 classes in the greater than 35.9 inch dbh class. Hadfield and others (2000) predicted an average increase of one DMR class every ten years for Douglas-fir dwarf mistletoe. These data suggest that Douglas-fir dwarf mistletoe may intensify at a slower rate in Douglas-firs in mature stands in southwestern Oregon.

Validating FVS—The Dwarf Mistletoe Impact Modeling System (DMIM): User Guide and Reference Manual Nonspatial Model 2005 Update (David 2005) describes equations used to modify FVS to account for the impact of dwarf mistletoe. The diameter growth modification equation for Douglas-fir dwarf mistletoe was based on ten-year diameter growth potential derived from pre-existing studies in Oregon, eastern Washington, Montana and the Southwest. Table 1 compares diameter growth potential of infected and uninfected Douglas-firs by BVR class in DMIM versus the southern Cascades plots. Ten-year diameter growth potential was impacted more severely in BVR classes 3 through 6 in the plots than by DMIM.

Data from the same studies were used to derive the equations for ten-year mortality in DMIM. Analysis of data from the southern Cascades plots indicated that mortality of Douglas-fir less than 9.0 inches dbh was much higher than DMIM in all BVR classes (Table 2). Douglas-fir greater than or equal to 9.0 inches dbh in the southern Cascades plots had much higher mortality than DMIM in BVR classes 5 and 6.

In spite of these differences, DMIM accounted for the effects of dwarf mistletoe on predicted stocking levels and volume of live Douglas-firs fairly well in eight of the ten plots after one ten year cycle. FVS overestimated stocking and growth in one plot with an unusually high proportion of infected Douglas-fir and high mortality, and underestimated it in a plot on an unusually productive site, suggesting that the model may not perform as well in extreme cases.

In addition, the high level of mortality in the southern Cascades plots compared to DMIM may have been influenced by the drier than average years during the study period. It is also possible that use of two different rating systems could account for some of the differences between DMIM and the plot data. To determine if this is a significant issue, a subsample of Douglas-fir in the plots will be re-rated using DMR and the ratings compared to the BVR.

Some problems appeared to be with aspects of FVS unrelated to dwarf mistletoe. The plant associations available for defining habitat types in the Western Cascades and Interior California-southern Cascades variants did not fit actual stand conditions very well.

DMR/BVR	0	1	2	3	4	5	6
DMIM (percent by DMR)	100	98	97	85	80	52	44
southern Cascades plots (percent by BVR)	100	92	100	69	69	39	8

Table 1—Ten-year diameter growth potential by DMR/BVR.

DMR/BVR	0	1	2	3	4	5	6
Dbh < 9.0 inches							
DMIM (percent mortality by DMR)	0.0	0.6	1.6	4.6	9.6	16.5	25.4
southern Cascades plots (percent mortality by BVR)	38.0	29.0	27.0	25.0	31.0	36.0	62.0
Dbh ≥ 9.0 inches							
DMIM (percent mortality by DMR)	0.0	0.5	1.4	3.8	8.0	13.7	21.1
southern Cascades plots (percent mortality by BVR)	6.0	0.0	8.0	5.0	5.0	11.0	45.0

Table 2—Ten-year mortality rate (percent mortality) by DMR/BVR and diameter class.

This affected accuracy of growth adjustment factors used in the model. Mortality in the plots affected growth and volume projections because FVS increased diameter and height of every tree until the end of the ten year cycle, regardless of whether and when it died.

Simulation models like FVS are not intended to provide plot-specific data at the end of every cycle that is comparable to data collected on site. Normally FVS is run for multiple cycles using data from several plots. In this case not only was FVS run for only one ten year cycle, but only one plot was used to represent each stand.

Collecting data in these plots for several more decades would provide the best information for adjusting DMIM equations for the Western Cascades and Interior California-southern Cascades variants of FVS. However, the information generated to date could be used now to make adjustments to the diameter growth and mortality equations. It would be beneficial to incorporate local plant associations (Atzet and others 1996). Additional analysis of this data that could be pursued would be to compare dwarf mistletoe spread and intensification rates in FVS with what was measured in the plots.

Summary

Ten permanent plots to measure the spread and impact of Douglas-fir dwarf mistletoe were installed in the Southern Oregon Cascade Mountains on the Rogue River-Siskiyou and Umpqua National Forests in 1992.

They were remeasured in 2002. Comparison of data from the two measurements showed that after ten years Douglas-firs that were heavily infected had less growth and higher mortality than uninfected or lightly infected Douglas-firs.

The number of infected Douglas-firs increased substantially in ten years. The majority of newly infected Douglas-firs were within 25 feet of previously infected Douglas-firs.

The effects of Douglas-fir dwarf mistletoe infection were greatest in small diameter and understory Douglas-firs. During the ten year period between measurements dwarf mistletoe spread into more small diameter and understory Douglas-firs than into larger Douglas-firs. Infected small diameter and understory Douglas-firs had less diameter growth and higher mortality than large Douglas-firs. Above BVR 4 diameter and height growth decreased significantly and mortality increased significantly. This suggests that widespread and severe Douglas-fir dwarf mistletoe infection is likely to adversely affect efforts to grow young Douglas-firs in southwest Oregon into large old trees and expect them to survive for many decades unless there is some form of management intervention.

Running FVS using data from the plots indicated that DMIM accounted for the effects of Douglas-fir dwarf mistletoe on predicted stocking levels and volume of live Douglas-fir in the majority of plots, in spite of differences between the plot data and data used to derive the equations for DMIM. Comparing model

projections to the plot data revealed that there were problems with FVS unrelated to dwarf mistletoe.

Ideally these plots should be followed for several more decades to confirm whether the initial trends will continue. However, data collected so far could be used now to modify equations used to predict growth and mortality in DMIM.

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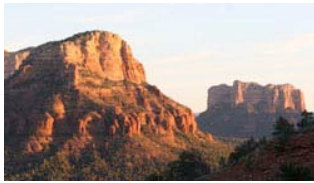
Fusarium Oxysporum Resistance in Koa – Early Results From Seedling Resistance Testing in Hawaii

Nick Dudley[1], Richard Sniezko[2], Robert James[3] and Phil Cannon[4]

Extended Abstract

Koa (*Acacia koa*) is a valuable tree species economically and ecologically in Hawaii. With dramatic declines in sugar cane and pineapple plantations, there is an opportunity and keen interest in utilizing native koa in reforestation and restoration efforts. However moderate to high mortality rates in many of the low-elevation plantings have impeded past efforts. The primary cause for this mortality, particularly in young plantings, is thought to be koa wilt, caused by *Fusarium oxysporum* f. sp. *koae* (FOXY). *F. oxysporum* is a relatively common agricultural and nursery fungus, but the origin of isolates of FOXY virulent to koa in Hawaii is unknown.

As in other tree species (Sniezko 2006) genetic resistance may be the key to developing useful populations and clones of koa to use in restoration or reforestation. Great differences in mortality among seed sources in a young koa field trial planted in 1999 were the impetus for developing a seedling screening test and investigating genetic resistance to FOXY (Sniezko 2003). Virulent isolates of the fungus have recently been identified, and a methodology for assessing young seedlings for FOXY resistance has been developed (Dudley and others 2007). Three small seedling trials (with 15, 23 and 25 seedlots, respectively) were undertaken in 2006 and 2007 to evaluate the levels of FOXY resistence. Many of the seedlots came from native stands or planted forests, but several seedlots were also from survivors within the 1994 and 1999 seed source trials at Hawaii Agriculture Research Center's Maunawili field site. All seedlots were open-pollinated. A composite of five virulent isolates of FOXY were used for inoculations (see Dudley and others 2007 for details inoculum preparation and inoculation methods). In each trial, seedling wilting and mortality in the greenhouse was recorded over a 90 day period after inoculation (in some trials additional mortality was recorded after seedlings were placed on benches outdoors).

In Test 1, seedlot mortality ranged from 5 to 79.2% (mean=49.5%); for Test 2, seedlot mortality ranged from 4.2 to 91.7% (mean= 56.7%), and for Test 3, seedlot mortality ranged from 46.2 to 100% for the 23 seedlots with at least 5 seedlings (mean=81.4%).

The preliminary summary from these first small trials examining koa wilt resistance in young seedlings is encouraging, and further evaluation of these data is underway. Future planned activities include: screening a larger number of FOXY isolates for their potential level of virulence, retesting a subset of seedlots to examine repeatability of results, screening a much larger number of seedlots for resistance to FOXY, and developing koa seed orchards capable of producing FOXY resistant seed.

[1]Nick Dudley is a Forester with the Hawaii Agriculture Research Center, Aiea, HI.
[2]Richard Sniezko is Center Geneticist at Dorena Genetic Resource Center, Cottage Grove, OR. rsniezko@fs.fed.us.
[3]Robert James is a Plant Pathologist with Forest Health Protection, USDA Forest Service, Coeur d'Alene, ID. rjames@fs.fed.us.
[4]Phil Cannon is Regional Forest Pathologist, Forest Health Protection USDA Forest Service, Vallejo, CA. pcannon@fs.fed.us.

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Cronartium ribicola Resistance in Whitebark Pine, Southwestern White Pine, Limber Pine and Rocky Mountain Bristlecone Pine - Preliminary Screening Results From First Tests at Dorena GRC

Richard A. Sniezko[1], Angelia Kegley[1], Robert Danchok[1], Anna W. Schoettle[2], Kelly S. Burns[3] and Dave Conklin[4]

Extended Abstract

All nine species of white pines (five-needle pines) native to the United States are highly susceptible to *Cronartium ribicola*, the fungus causing white pine blister rust. The presence of genetic resistance will be the key to maintaining or restoring white pines in many ecosystems and planning gene conservation activities. Operational genetic resistance programs are well underway for western white pine (*Pinus monticola*), sugar pine (*P. lambertiana*), and eastern white pine (*P. strobus*). However, very little is known about the frequency and types of resistance in the high elevation species of white pines, and in particular little or no data is available on family or seed source variation within species such as southwestern white pine (*P. strobiformis*, SWWP), whitebark pine (*P. albicaulis*, WBP), limber pine (*P. flexilis*, LP) and Rocky Mountain bristlecone pine (*P. aristata*, RMBCP). Separate trials to evaluate resistance within these four high elevation species are now underway using seedling families from individual parent tree seed collections. Parent tree selections aregenerally from canker-free trees in natural stands vary in incidence of rust, for example SWWP selections were from stand where >90% of the trees are infected while many ofthe LP and all of the RMBCP selections were from stands not yet invaded by the rust in preparation for proactive

intervention (Schoettle and Sniezko 2007). The WBP were selected on the basis of cone availability and not necessarily putative blister rust resistance; however, most of the WBP selections were canker-free.

Artificial inoculation of young seedlings of WBP (from tree selections in Oregon and Washington), RMBCP (from Colorado), LP (from Colorado and southern Wyoming) and SWWP (from New Mexico) with *C. ribicola* were undertaken recently (Table 1). Protocols for inoculation and assessment followed established Dorena Genetic Resource Center (Dorena GRC) standards used for western white pine and sugar pine, as well as prototype research work on these species at Rocky Mountain Research Station (RMRS). In the traditional operational rust resistance screening, seedlings are assessed for five years after inoculation to help elucidate different types of resistances. In a separate short-term operational test, very young seedlings are assessed for six months to two years, particularly to note the presence or absence of a hypersensitive response in the needles (HR) and the presence/absence of stem symptoms. The number of families per species the tests discussed here varies from 10 to >100 (Table 1).

For SWWP, WBP and RMBCP, 2- to 3-year-old seedlings were utilized, and seedlings are being evaluated for a range of possible resistance responses including: number of needle spots, presence and number of stem symptoms, latent stem symptoms, associated morphological and growth patterns, mortality, time of mortality, and combinations of these responses. Two-month-old LP seedlings were inoculated in 2006 and are being evaluated for presence of needle spots, stem symptoms, morphological traits as well as mortality. For SWWP, LP and WBP seedlings of some families are also being evaluated for presence of hypersensitive response (HR) in the needles. Previous inoculations of young seedlings at Dorena GRC and Institute of Forest

Genetics (IFG) revealed evidence of families with HR in SWWP (Dorena GRC, unpublished data; Kinloch and Dupper 2002; Vogler, pers. comm.) and presence of HR in a bulked seedlot of LP (Kinloch and Dupper 2002); evidence for HR in RMBCP is currently being evaluated at IFG in cooperation with RMRS (Vogler and others 2006).

Inoculation success was very high in all trials. Final results are still pending for all trials. Early results show that genetic variation in resistance exists for all four species and suggest at least a small number of families of each species have high levels of one type of resistance (low levels of stem symptoms). Sniezko and others (2007) reported some early results from a 2004 inoculation in WBP; in a second trial of additional WBP families (2005 inoculation), results one year after inoculation showed a range in family stem infection from 3% to >85% and continue to suggest a geographic trend in this resistance trait (unpublished data). The phenology of disease symptomology appears slower on RMBCP than other species; stem symptoms were only becoming obvious on seedlings 22 months after inoculation. Sugar pine and western white pine seedlings included as controls in the RMBCP 2005 inoculation appear to be much more susceptible than the RMBCP families, displaying many more needle spots, earlier stem symptoms, and earlier mortality. Assessments of the RMBCP seedlings will continue for several more years. Results from testing of SWWP progenies indicate that two of the SWWP parents tested have HR. However, the other families in the test display other types of resistance including low number of needle spots, moderate levels of canker-free

seedlings, and to this point, low mortality of infected trees. Young LP, inoculated in 2006, showed a range in family stem infections from 9% to 100% one year after inoculation (preliminary unpublished data). At this early stage in the study, several LP families have a large proportion of their seedlings remaining free of stem symptoms; these and all the families will be assessed further over the next year. In testing of high elevation white pines at Dorena GRC, HR resistance has only been noted in SWWP, and thus the resistance in the other species may be due to other resistance mechanisms.

The frequency, types, and levels of genetic resistance shown in these early screening results are encouraging. Further assessments in all trials are underway. In addition, inoculation of hundreds of new WBP families is planned for 2008, as is inoculation of two-year old LP families to evaluate a suite of resistance responses.

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Table 1—Ongoing C. ribicola resistance screening in high elevation white pines at Dorena GRC.

Species[a]	# Families	Test Type[b]	Seed Sources	Sow Year[c]	Inoculation Year[d]
WBP	43	Traditional	Oregon/Washington	2002	2004
WBP	101	Traditional	Oregon/Washington	2004	2005
WBP	24	Traditional	Oregon/Washington	2005	2006
WBP	236	Traditional	range-wide	2007	scheduled for 2008
SWWP	8	Traditional	New Mexico	2002	2003
SWWP	5	Short-term	New Mexico	2003	2003
SWWP	1	Short-term	New Mexico	2007	2007
RMBCP	184	Traditional	Colorado	2002	2005
LP	77	Short-term	Colorado/southern Wyoming	2006	2006
LP	74	Traditional	Colorado/southern Wyoming	2007	scheduled for 2008

[a] Where WBP = whitebark pine, SWWP = southwestern white pine, RMBCP = Rocky Mountain bristlecone pine, and LP = limber pine
[b] Traditional testing consists of inoculation of 2- to 3-year old seedlings and assessment over a period of five years for development of disease symptoms and mortality. Short-term testing involves inoculation of very young seedlings and assessment for the presence of a hypersensitive reaction in the needles and subsequently the presence/absence of stem symptoms and mortality
[c] Year in which the trial was sown for testing
[d] Inoculations are conducted in late August or September.
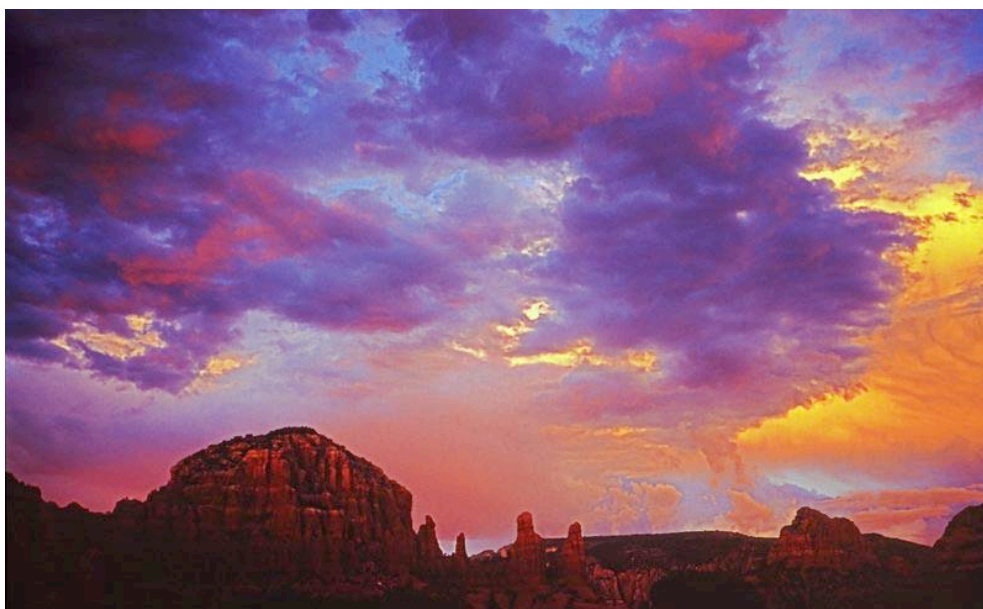
Sunset over the red rocks outside Sedona, Arizona. Wikipedia.org. October 2008

Interactions Between Western Gall Rust and its *Pinus* Hosts, *P. jeffreyi* and *P. contorta*, in Sierra De San Pedro Martir National Park, Northern Baja California, Mexico

Detlev R. Vogler[1] and Brian W. Geils[2]

The Sierra de San Pedro Martir is a mountain range in north-central Baja that comprises the southern-most extension of the Californian coniferous flora, including *Pinus jeffreyi*, *P. contorta*, *P. lambertiana*, *Abies concolor*, and *Calocedrus decurrens*. These forests are similar to those in the State of California, with many of the same pathogens present in both the U.S. and Mexico. In the western U.S., attempts to understand the historic roles that pathogens played in forest development and sustainability have been frustrated by more than a century of fire control and logging. The latter have so altered the ecological context that it is difficult to assess the roles of pathogens as other than pests that cause economic and aesthetic damage. Thus, we can only guess at the pristine state of most western U.S. forests, and are obliged to speculate about the abundance and diversity of pathogens before fire control and logging so dramatically transformed the landscape.

The San Pedro Martir may offer clues to how pathogens functioned in a pre-1850 context. Aside from cattle-grazing, which has been conducted continuously there for two centuries, fire suppression has been minimal and logging almost non-existent (Stephens et al. 2003). Recent research in the Martir (Maloney and Rizzo 2002) has documented the presence of many of the same pathogens that occur in the Sierra Nevada of California.

However, their distribution and prevalence are not well understood. In 2003, the first author examined a set of 49 plots established by the research team of Prof. Scott Stephens (UC Berkeley), and confirmed the presence and abundance of many of the same pathogens reported by Maloney and Rizzo (2002), including white fir leafy mistletoe (Phoradendron pauciflorum), Elytroderma needle disease (caused by Elytroderma deformans) and limb rust (caused by a Cronartium sp.) on Jeffrey pine, and root disease associated with Heterobasidion annosum and an Armillaria sp.. Missing, however, were any records of western gall rust (caused by Peridermium harknessii), which was known to be present in the Martir.

Aware that both Jeffrey and lodgepole pine were commonly infected by western gall rust at Vallecitos Meadows, which is situated at ~8400 ft elevation atop the highest and northernmost of the three plateaus that comprise the Martir, we began a survey there in 2004 to assess the prevalence and host-associations of *Peridermium harknessii*. In 2004 through 2007, assisted by Scott Stephens' students and associates (Andy Amacher, 2004; Richard Rypinski, 2005; and Lana Schide, 2007) and by the second author in 2007, we laid out 15 east–west transects 100 m apart, with circular, 20 m radius plots placed every 100 m along them; transects ranged in length from 1.0 to 1.5 km. Overall, 199 0.03 ha plots were established totaling 5.97 ha, or a 3% sample of the 195 ha meadow area.

Western gall rust was found infecting 7% of all Jeffrey and lodgepole pine at Vallecitos, or 4.4% of the Jeffrey and 10.6% of the lodgepole. Total numbers of each species were similar (4,473 Jeffrey and 3,364 lodgepole), and both species exhibited a similar size distribution, with the majority of stems in the smallest size classes. However, the distributions of infected species by size class were mirror images of each

other: most of the infected Jeffrey pines were in the smaller size classes (< 12" diameter), whereas the majority of the infected lodgepole pines were in the larger size classes (≥13" diameter).

These reciprocal host distributions suggest that western gall rust may regulate the success of Jeffrey pine relative to lodgepole by posing a challenge to survival of Jeffrey seedlings and saplings where lodgepole are already present, since Jeffrey reproduction would be exposed to a rain of spores from infected lodgepole overstory. Further investigations are needed to test this and alternative hypotheses. Does western gall rust maintain a fluctuating equilibrium between the distributions and abundance of the two pine species? Since fire is an active and frequent agent in these forests, the relative susceptibilities of Jeffrey and lodgepole regeneration to fire may also be significant.

At present, it appears that western gall rust plays a role in facilitating the invasion of Jeffrey stands by lodgepole pine, and perhaps vice versa. However, the relative susceptibilities of the two species to the gall rust pathogen may counter-balance these effects, but this needs to be confirmed experimentally. We observed that Jeffrey pine seedlings that were establishing beneath the crowns of infected lodgepole were often not infected. Were they escaping inoculation by "sheltering" beneath the lodgepole crowns, or were they more resistant to the pathogen?

Finally, those few remaining forest lands that have experienced little logging or fire suppression should be preserved and studied if we are ever to understand the historical roles of pathogens in forest succession and sustainability. While treating pathogens solely as pests to be controlled may suffice for production forestry, forest management for ecosystem, recreational, and aesthetic values requires a deeper appreciation of the beneficial roles of pathogens.

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POSTER ABSTRACTS

Forest Fires and the Spread of *Armillaria ostoyae* in a Ponderosa Pine Forest
J. T. Blodgett[1] and J. E. Lundquist[2]

Forest tree diseases are the most pervasive of all disturbances in western coniferous forests, yet they are among the least understood and perhaps most underestimated components of these ecosystems. Diseases, for example, influence forest fires by changing stand structure and generating fuels. Conversely, fires influence the abundance and distribution of fuel-generating diseases, but few studies have examined these relationships.

The Black Hills in South Dakota has been the focus of much attention following recent severe fires. Armillaria root disease, caused by *Armillaria ostoyae*, is arguably the most common disease in this forest. This field study examines the effects of fire on *A. ostoyae* in ponderosa pine (*Pinus ponderosa*), and tests methods of quantifying and detecting this pathogen (1,2).

Five plots were established in the Black Hills National Forest where fires occurred 3 years previously. Each plot consisted of four subplots varying in fire damage intensity (low, medium, high, and unburned). Wood blocks (ponderosa pine and aspen [*Populustremuloides*]) were buried north and south of 15 ponderosa pine trees in each subplot (1,200 total blocks).

After 2 years the wood blocks were examined for colonization by *A. ostoyae*. Results were compared with an associated study 2 years earlier (1) where the

abundance of *Armillaria* rhizomorphs was ranked for each quarter of the same 15 trees per subplot as follows: 0 = no rhizomorphs; 1 = low; 2 = moderate; 3 = high; 4 = very high; and 5 = extremely high abundance.

The proportion of blocks colonized by *A. ostoyae* increased as fire intensity increased (P < 0.001). Ponderosa pine was more heavily colonized than aspen (e.g., 77% vs. 63%, in high intensity plots). Rhizomorph presence was significantly correlated with wood block colonization for both ponderosa pine (P = 0.007, R = 0.586) and aspen (P < 0.001, R = 0.695). Host condition worsened with increasing fire intensity (P < 0.001). Mortality was 0% for unburned and low fire intensity, 59% for medium, and 100% for high.

The wood block trap method offers a simple and relatively inexpensive means of detecting and quantifying Armillaria. Armillaria can survive intense fires and can readily increase its inoculum potential presumably by colonizing roots of fire-killed trees. Wildfires can influence the frequency of Armillaria. Seedling mortality due to Armillaria might be a limiting factor to successful regeneration on burned sites. The health of a site may be impacted over the long term by fire-induced increases in Armillaria.

References

Blodgett, J.T., Lundquist, J.E. 2004. Forest fires Influence the abundance of *Armillaria* root disease in ponderosa pine. Phytopathology 94:S8.

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The Role of Climate and Topography in the Development of *Dothistroma septosporum*
Crystal Braun[1] and Kathy Lewis[1]

Dothistroma septosporum (Dorog.) Morelet is a foliar fungus of pine trees, infecting needles and causing premature defoliation. This results in reduced growth of the tree and, in severe cases, death. Red banding, caused by the mycotoxin dothistromin, along needles where infection has been successful characterizes this fungal pathogen.

Dothistroma needle blight is recognized worldwide as a problematic pathogen, affecting over 60 *Pinus* species in 45 countries. Currently there is a severe outbreak of *Dothistroma* needle blight developing in the Skeena Stikine Forest Region in northwestern British Columbia (BC). Damage in these plantations has ranged from low levels of infection to nearly 100% mortality.

A high concentration of young susceptible hosts and the climate of warm, moist summers and cool, wet falls in this area are thought to be contributing to the outbreak. Heavy fogs generated in plantations close to rivers, lakes, or streams may also facilitate *D. septosporum* development.

The purpose of this project is to identify climatic and site conditions contributing to the development of *D. septosporum* with respect to the severe outbreak happening in northwestern BC. The main objectives are to monitor the variation in disease expression, identify the ranges of temperature and humidity conducive to disease

[1]Crystal Braun and Kathy Lewis are a graduate student and professor respectively, in the Department of Ecosystem Science and Management at the University of Northern British Columbia. Email: braunca@gmail.com, lewis@unbc.ca.

development, and identify the role of site factors such as elevation, slope, and proximity to water in disease severity.

In the Bulkey, Kispiox, and Cranberry Timber Supply Areas (TSAs), four sites were selected according to signs of *Dothistroma* infection, obtaining sufficient geographic coverage, and accessibility. Within each site, three plots were established for weekly monitoring. Weather stations were set up at all twelve plots, and leaf wetness sensors were set up in 6 plots. In each plot six trees were randomly selected. On each tree, four nodes were flagged and ten needles were marked with blue paint. These needles were examined weekly for the development of red bands, fruiting bodies, and spore production. Spore production was assessed using a hemocytometer. When fruiting bodies were detected, needles extracted from the tree for dissection to determine their reproductive states. Data provided by the Ministry of Forests and Range in Smithers, BC, will be used to investigate topographical effects on disease development. This data is a census of the lodgepole pine plantations in the Skeena Stikine Forest Region. *Dothistroma* infection severity was assessed by aerial survey, rated as functional live crown – a function of average live crown and average live nodes on a tree. A spatial analysis of disease severity according to aspect, slope, slope position, elevation, and proximity to water will be conducted. Data analysis is currently underway.

The results will be used to construct a hazard rating system of pine plantations to future *Dothistroma* needle blight epidemics that is adaptable to predictions of climate change. These findings will be shared with the Ministry of Forests (MOF) and Canadian Forest Service (CFS) to assist forecasting climate changes in BC.

Vectoring Capabilities of the Banded Elm Bark Beetle (*Scolytus schevyrewi semenov*) in Relation to the Dutch Elm Disease Fungus (*Ophiostoma novo-ulmi brasier*) in Colorado

Ronda D. Koski[1] and William R. Jacobi[2]

Scolytus schevyrewi, the banded elm bark beetle (BEBB), was recently introduced to the U.S.A. and in some areas is more common than *S. multistriatus*, the smaller European elm bark beetle. It is not known if the BEBB is an effective vector of *Ophiostoma novo-ulmi*, the cause of Dutch elm disease (DED). The DED fungus was cultured from adult BEBB emerged from DED affected elms in an earlier study. Objectives of this research were to characterize feeding wounds and determine if BEBB can transmit the DED fungus and induce disease. BEBB reared from elm logs were infested with the DED pathogen, then placed inside five fabric cages on American elm branches. Infested BEBB were allowed to feed for several weeks. The field experiment was replicated five times during the summer. Similar procedures were carried out on excised elm branches placed in rearing cages in a laboratory. The number of branch crotches, type and number of feeding wounds were recorded. No disease symptoms were induced in the field inoculations. Feeding wounds were plated on amended agar. Of the 1383 BEBB placed in the field cages and 390 BEBB placed in laboratory cages, feeding wounds were produced by 3.4% in the field and 4.6% in the laboratory. The DED pathogen was recovered from 54% of the field trial wounds and 30% of the laboratory trial wounds. Current DED management practices may need modification if the BEBB proves to be an efficient vector of the pathogen.

[1]Ronda D. Koski is a Research Associate in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO.

[2]William R. Jacobi is a professor of plant pathology in the Department of Bioagricultural Sciences and Pest Management at Colorado State University, Ft. Collins, CO. william.jacobi@colostate.edu.

Natural arch rock formation known as Devil's Bridge, Sedona, Arizona. Wikipedia.org. October 2008

Determination of Suitable Climate Space for *Armillaria ostoyae* in the Oregon East Cascades

John W. Hanna[1], Mee-Sook Kim[1], Ned B. Klopfenstein[1], Aaron L. Smith[2] and Helen M. Maffei[2]

A collaborative project to determine the distribution of *Armillaria ostoyae* on the eastern slopes of the Oregon Cascades was started in 2007. The methods and accomplishments of the 2007 field-collection stage of the project are highlighted in this presentation. The next phase of the project will identify Armillaria isolates to species using molecular methods. Sites positive for *Armillaria ostoyae* will be associated with climate data to determine the potential distribution (and disease activity) of *Armillaria ostoyae* within the region. Data from this region can then be added to a global *Armillaria ostoyae* dataset that will help develop climate models to predict the future changes in *Armillaria ostoyae* under various climate change scenarios.

[1]John W. Hanna , Mee-Sook Kim, and Ned B. Klopfenstein are Biological Technician, contract Plant Pathologist, and Research Plant Pathologist, respectively, at the USDA Forest Service – RMRS, Forestry Sciences Laboratory, ID. jhanna@fs.fed.us, mkim@fs.fed.us, nklopfenstein@fs.fed.us.

[2]Aaron L. Smith and Helen M. Maffei are Plant Pathologists at the USDA Forest Service, Forest Health Protection, Region 6, Deschutes National Forest, Bend, OR. alsmith@fs.fed.us, hmaffei@fs.fed.us.
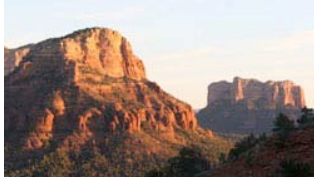
Assessment of Whitebark Pine Regeneration in Burned Areas of the Shoshone and Bridger-Teton National Forests and Wind River Reservation, Wyoming

Jennifer G. Klutsch[1], Betsy A. Goodrich[1] and William R. Jacobi[1]

Whitebark pine (*Pinus albicaulis*) forests in northwestern Wyoming, including the Wind River Reservation, Shoshone National Forest and Bridger-Teton National Forest, have been severely affected by bark beetle, wildfire, and white pine blister rust induced mortality. In previously burned areas, there is limited critical information on the regeneration of whitebark pine seedlings. The purpose of this study was to determine the amount of regeneration in recent and old fires as compared to non-burned areas and also to determine what site factors impact regeneration occurrence. A survey of whitebark pine regeneration within six areas that have experienced stand replacing fires 8 to 32 years previous was conducted in the Wind River Mountain Range in summer 2007. Fires were divided into polygons of similar aspects, elevations, and forest types; with each polygon comprising a minimum area of burned forest of 1.76ha (80m x 220m) and an associated minimum area of non-burned forest of 1.10ha (50m x 220m). A total of 14 polygons were samples, with 74 transects in burned areas and 28 in non-burned areas. Each transect was 2m in width and a minimum of 200m in length, and in which tree regeneration was tallied by height class. To assess the nearest whitebark pine seed source, variable radius plots were established every 80m in the non-burned perimeter of the fire. This study will benefit restoration activities in the region by providing information on the survival and health of whitebark pine regeneration in previously burned areas.

[1]Jennifer G. Klutsch, Betsy A. Goodrich, and William R. Jacobi are graduate students and professor respectively, in the Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Colins CO. jennifer.klutsch@colostate.edu, betsy.goodrich@colostate.edu, and william.jacobi@colostate.edu.

Predicting Risk of Infection by Comandra Blister Rust on Lodgepole Pine in the Sub-Boreal Spruce (SBS) Dry Cool Biogeoclimatic Subzone

Richard Reich[1] and Sally John[2]

The objective of this project is to model the influence of multiple factors in predicting risk of infection on a comandra blister rust resistance trial. Factors include: site, climate, ecology, host resistance, and alternate host abundance and susceptibility. The trial layout involves 50 trees of each of 130 families planted on a 1.5m square grid on each of 3 different sites in the SBSdk. Counts of the number of rust infections of comandra blister rust (CBR), stalactiform blister rust, and western gall rust were made in 2006 and also in 2007. Counts of the number of stems of the alternate hosts for the respective blister rusts were conducted on a 1.5 meter grid in 2007. Additional data collected included a decimetre accuracy differential GPS survey for terrain, and a standard ecoclassification for each site. A preliminary spatial analysis of the percent incidence and the intensity of CBR infection based on the distance from the alternate host *Geocaulon lividum* was conducted. The preliminary spatial analysis showed that risk of infection is very high (50 – 60%) when lodgepole pine seedlings are in close proximity to *Geocaulon*, drops dramatically over the first several meters to ~ 20%, and gradually decreases to close to zero by 25 to 35 meters away. Future work will focus on modelling the influence of major variables. We hope to involve a graduate student in statistics in planning and implementing analyses.

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[1]Richard W. Reich is a Forest Pathologist with the Ministry of Forests and Range, Prince George, BC, Canada. richard.reich@gov.ba.ca.

[2]Sally E.T. John is a Consulting Forest Geneticist with Isabella Point Forestry Ltd. Salt Spring Island, BC, Canada. ipf@saltspring.com.

COMMITTEE REPORTS
Hazard Tree Committee Report
Chair: Pete Angwin

Pete Angwin opened the breakfast meeting with slides of 2007 Hazard Tree Workshop in Midway,Utah. The Workshop was well attended, with 51 participants (43 paid and 8 waived registrations). The committee meeting started after the slide show. Four items were on the formal agenda:

1) **Pete Angwin** led the discussion on the 2010 Hazard Tree Workshop. Planning for the Workshop will start in late 2008. **Call or e-mail Pete if you'd like to be part of the planning committee.**

Discussion followed regarding the location of 2010 Workshop. Traditionally, the workshop has been held in the western United States, but several locations, including Saint Paul, MN and Asheville, SC, have been suggested.

Should we consider a location outside of the Western US? Holding the workshop outside of the western US would provide an opportunity to have a joint workshop with pathologists and hazard tree specialists from other regions. The first planning committee meeting will be in the Spring of 2009, at which time, meeting location nominations will be accepted and discussed. The final location decision will also be made at the meeting. *Committee members voted that any location nationwide would be acceptable as a nomination.*

2) Judy Adams provided an update on the ITFD (International Tree Failure Database). The ITFD Steering Committee met in May in Chicago. It was a productive and energetic meeting with USDA Forest Service folks and other key stakeholders. The database is working well, as is the program for handheld data recorders. Many pathologists, land managers, arborists, and others nationwide are now trained to enter data.

The next ITFD Steering Committee meeting will be in either Portland or San Diego in the fall of 2008. It will be organized by Paul Ries and will be held jointly with a State/Municipal/Urban meeting.

3) **Brennan Ferguson** next led a discussion on updating the FIDL (Forest Insect and Disease Leaflet) series.
11 FIDLs addressing tree decay fungi were published from 1958-1977, but none have been revised! Brennan will be placing all of them on line soon. Shall we revise any or all of these FIDLs? Are they still relevant? The Forest Health Protection Washington Office (WO) previously assigned people to write or revise these but currently there is no WO leadership to do so. Some revisions could be as simple as new latin names or new color formats.

Several committee members volunteered to lead efforts to revise particular FIDL as follows. No one who volunteered is assumed to be the author but will, at the very least, help coordinate the effort.

Rust red stringy rot – Don Goheen, Greg Filip
Decay and discoloration of aspen – Kelly Burns, Brian Geils, MaryLou Fairweather, Jim Worrall
Heartrot of Engelmann spruce – Jim Worrall, Brian Geils, Kelly Burns
Heart rots of Douglas-fir- Don Goheen, Brennan Ferguson
Red rot – MaryLou Fairweather, Blakey Lochman
Red and white fir – Katy Mallams, Kristen Chadwick, Pete Angwin
Western hemlock – Paul Hennon, Lori Trummer, Greg Filip
White trunk rot of hardwoods – Lori Trummer

4) **Lori Trummer and Jim Worrall** led a discussion on the National Hazard Tree Coordination Initiative. Over the past few months, a small planning group

consisting of Lori Trummer (Region 10), Jim Worrall (Region 2), Joe O'Brien and Jill Pokorny (Northeastern Area), Bill Jones (Region 8), Pete Angwin (Region 5) and Judy Adams (FHTET) produced a draft of the initiative, which was distributed to the committee. The draft addresses the need for more national coordination, funding and improvement of USDA Forest Service hazard tree programs, and makes several suggestions to accomplish this. As the discussion got underway, Jim Hoffman made a touching speech on his "pre-passion" for the initiative. Lori introduced the subject by relating some of the lessons from the 2005 Alaska fatality of a Boy Scout leader in Alaska- that better national coordination of hazard tree programs would allow us to work together better, share resources, and not "reinvent the wheel". Mary Lou Fairweather suggested including the financial costs of accidents (if known) in the Initiative proposal since a Region 3 experience is that one Ranger District's budget is now devoted to paying the costs of a hazard tree-related accident settlement. Bill Woodruff strongly supported that the WO be involved, perhaps even the Chief. Katie Mallams suggested coordinating with recreation, fire, and roadside maintenance and integrating their concerns as we approach the WO. Don Goheen suggested that WIFDWC send a strong resolution to the WO that they fill the National Pathologist position with Hazard Tree leadership as a major issue.

The group was asked if creating a National Hazard Tree Website was in their interests. This exercise could be done in a year and would help support the case for WO recognition for National Hazard Tree programs, and support the future proposal to request that WO dollars be dedicated to Hazard Tree issues.

Committee members agreed to contribute to a National Website with information, posters, brochures, etc. and Regional links to individual programs. Judy Adams agreed to sleuth out a proper location for the Website – likely hosted by FHTET or St. Paul Field Office. She will check with Frank Sapio and others. All regions will be involved with populating the Web page.

Fred Baker proposed that he could contribute to Hazard Tree Training programs online, and John Schwandt suggested increased coordination with State Recreation and Highway departments.

Committee members agreed to have a smaller work group, with all regions represented, develop a proposal to be presented at the Fall 2008 Forest Health Protection Directors meeting, encompassing recognition for a National Hazard Tree Program and requesting dollars to support these efforts. Details will be worked out over the next year thru e-mail and conference calls, and perhaps a face to face meeting.

The following Regional representatives were added to the work group listed above so that all Forest Service Regions now have at least one representative: Region 6: Don Goheen (conference calls ok but no travel) and Greg Filip; Region 3 MaryLou Fairweather; Regions 1&4: John Guyon (Marcus Jackson will serve as ITFD representative). This is in addition to the six current committee members listed above.

Notes taken by Lori Trummer, Jim Worrall and Judy Adams. Edited and submitted by Pete Angwin

Nursery Pathology Committee
Chair: Bob James

Eight people attended the committee meeting. Discussions centered around continuing efforts to find alternatives to methyl bromide soil fumigation in bare root forest nurseries. The major group of pathogens in forest nurseries continues to be *Fusarium* spp., although *Cylindrocarpon* spp. have seemed to increase in importance recently. Molecular genetic characterization of *Fusarium oxysporum* populations associated with forest nurseries has recently commenced at the Rocky Mountain Research Station. Isolates highly virulent to conifer seedlings have been reclassified as *F. commune*; current investigations are underway to determine extent of this new taxon throughout forest nurseries in western North America. No new and unusual diseases were reported.

The chair of the committee was transferred to Katy Marshall [FHP, R-6] because of the upcoming retirement of the current chairman Bob James.

Root Disease Committee Report
Chair: Brennan Ferguson

Attendance: ~40

The Root Disease Committee luncheon in Sedona was well-attended. Several reports and presentations were given followed by individual reports on current root disease work. The following notes, reports, and work summaries were submitted for inclusion in the Committee notes.

Greg Filip: The IUFRO Root & Butt Rot Working Group met in the western United States August 12-19, 2007. The meeting was co-sponsored by the University of California, Berkeley, and the USDA Forest Service, Region 6, Forest Health Protection unit. The meeting began in Berkeley, CA, and ended in Medford, OR. The goal of the meeting was to show participants a sample of the vast diversity of landscapes, forests, and root diseases that occur in the Pacific West, U.S.A. Approximately 70 people from 17 countries traveled over 1200 miles through three national parks and 10 national forests. We saw all of the major root diseases in the Pacific West: *Armillaria, annosus, Phellinus*, and black stain. One participant described the meeting as an "International bus Field trip marathon" = IUFRO. Another said "It was a little short of way too much." Most were very pleased with the meeting and the weather. Thanks to all who helped to plan and facilitate the meeting: University of California Berkeley staff and Forest Health Protection pathologists in Regions 5 and 6. The next IUFRO Root and Butt Rot Working Group meeting will be held in 2011 in northern Italy. Ciao!

Charles "Terry" Shaw: Copies of the 1991 USDA Forest Service Agricultural Handbook "Armillaria Root Disease" are still available. Contact Terry (cgshaw@fs.fed.us) for information.

Aaron Smith: A project to determine the distribution of *Armillaria ostoyae* on the eastern slopes of the Oregon Cascades was started in 2007. Collaborators are John Hanna, Aaron Smith, Helen Maffei, Mee-Sook Kim, and Ned Klopfenstein. The collection of isolates and field data took place in 2007; the next phase of the project will identify Armillaria isolates to species using molecular methods. Sites positive for *A. ostoyae* will be associated with climate data to determine the potential distribution (and disease activity) of *A. ostoyae* within the region. This data can be added to a global *A. ostoyae* dataset that will help develop climate models to predict the future changes in *A. ostoyae* under various climate change scenarios.

Brennan Ferguson: The Montana DNRC Forest Pest Management program will soon have a new "Forest Insect & Disease Leaflet" web page available. All 173 current forest insect and disease titles will be available as PDFs in a consistent format and quality. Various bibliographies, with links to the leaflet PDF files, will be provided. Full citations are provided for copying into working documents as desired. Announcements will be sent to both pathology and entomology list servers when the site is available.

Richard Sniezko: *Phytophthora lateralis* resistance screening in Port-Orford-cedar continues. Some new greenhouse tests show that some seedling families die very quickly (by ~110 days after inoculation), but others seem to have some type of 'slow lateralis' resistance, and some seedlings in those families survive for 2 or even 3 years in the greenhouse after inoculation. This trait is in addition to the other 'high survival' resistance trait in which some families show 50 to 100% survival in the greenhouse even 3 years after inoculation. Over 12,500 parent trees have been screened via the stem dip technique since 1996, and the top 10% of those are being evaluated in a second test using a root dip technique.

Rona Sturrock: Canadian Forest Service, Pacific Forestry Centre – Research summary

Completed Papers

Lim, Y.W., R.N. Sturrock, I. Leal, K. Pellow, T. Yamaguchi and C. Breuil. 2007. Distinguishing homokaryons and heterokaryons in *Phellinus sulphurascens* using pairing tests and ITS poly-morphisms. Antonie van Leeuwenhoek (in press).

Sturrock, R., S. Zeglen, and J. Turner. 2006. British Columbia's coastal forests: Laminated root rot forest health standestablishment decision aid. BC Journal of Ecosystems and Management 7(3): 41-43.

Ongoing Projects

Douglas-fir interactions with *P. sulphurascens* (syn. *P. weirii*), including resistance

Screening of cDNA libraries from two Douglas-fir families proposed to have differential tolerance to the fungus.
Sturrock, R.N., M.A. Islam, and A.K.M. Ekramoddoullah. 2007. Host-pathogen interactions in Douglas-fir seedlings infected by *Phellinus sulphurascens*. Phytopathology - in press.

Islam, M.A., R.N. Sturrock, and A.K.M. Ekramoddoullah. A proteomics approach to investigate proteins differentially expressed in Douglas-fir seedlings infected by *Phellinus sulphurascens*. In preparation for submission.

Islam, M.A., R.N. Sturrock, and A.K.M. Ekramoddoullah. Ultrastructural and immuno-histochemical studies to understand the host-pathogen interaction between Douglas-fir and the fungus *Phellinus sulphurascens*. In preparation for submission.

P. sulphurascens diversity and identification and expression analysis of putative PR genes

Using SSCP and other sequencing techniques to conduct genetic analysis of 72 isolates (including heterokaryons and homokaryons from NA and Eurasia) X 9 genes (including ITS, ML, MS, Act2, rpb1, RPB2, tef1, laccase, and TUB).

Screening of cDNA libraries from *P. sulphurascens* mycelia 'induced/exposed to' Douglas-fir root tissues.

Collecting *P. sulphurascens* isolates from N/S/E/W extremities of its range. Thanks to Ellen M. Goheen, Don Goheen, and Pete ngwin for their efforts in Oregon and California.

REQUEST FOR ASSISTANCE: Can anyone provide isolates from Idaho and/or Montana?

Distribution and impacts of *Phellinus* root disease in the southern interior region of British Columbia

Collaborating with Project Leader Michelle Cleary (BC Ministry of Forests and Range) to better define where DRL occurs in interior BC and what its impact is and to estimate an operational adjustment factor (OAF) to use in timber-supply analyses.

Occurrence and behaviour of *P. weirii* and other decay organisms in western redcedar and yellow cedar

Initial thinking; looking for more isolates and collaborators; gene sequencing as for *P. sulphurascens*; literature synthesis needed.
Climate change syntheses. Overview paper at WIFDWC 2007. Annotated bibliography and other contributions to WWETAC/WIFDWC initiative to look at Climate, Climate Change, and Western Forest Diseases.

Ned Klopfenstein, Paul Zambino, Mee-Sook Kim, Bryce Richardson, Geral McDonald, John Hanna, and James Donley: USDA Forest Service, Rocky Mountain Research Station, Moscow, ID

The Moscow research group (Ned Klopfenstein, Paul Zambino, Mee-Sook Kim, Bryce Richardson, Geral McDonald, John Hanna, and James Donley) continues to work on DNA-based characterization of Armillaria species (Kim et al. 2006) and phylogenetic relationships of *A. ostoyae* across the landscape (Hanna et al. 2007b). Ongoing studies are examining the genetic relationships among *A. ostoyae* isolates from across the Northern Hemisphere. International collaborations have been established, and recent collection trips were made to Alaska, Korea, and Mexico. We are hoping for collaborations among several colleagues who have *A. ostoyae* isolates from known locations. This information will help evaluate potential invasive risks of *A. ostoyae* and lay a framework for predicting potential impacts of climate change on Armillaria root disease. Recently, *A. nabsnona* was reported from Hawaii for the first time (Hanna et al. 2007a).

Manager guidelines are being developed for root disease considerations when implementing fuels treatments (Rippy et al. 2005). In addition, the Armillaria response tool (ART), a web-based tool, was developed for fuels-treatment managers that predicts risks of Armillaria root disease based on habitat type and host trees (McDonald et al. 2007; Armillaria Response Tool

http://forest.moscowfsl.wsu.edu/fuels/tools/art/).

Currently, we are combining DNA-based diagnostics of *A. ostoyae* with precise location data to develop a model to predict *A. ostoyae* distribution based on climate factors. A collaborative effort with Helen Maffei and Aaron Smith is surveying *A. ostoyae* in the east-slope Cascades of Oregon to include in the predictive model.

Molecular diagnostic tools were developed to distinguish *Fusarium commune*, which is highly pathogenic in conifer nurseries, from nonpathogenic *F. oxysporum*, which is morphologically indistinguishable from *F. commune* (Stewart et al. 2006). Ongoing collaborations with Bob James and Kas Dumroese are examining the potential biological control potential of nonpathogenic *F. oxysporum*.

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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 marker. Forest Pathology 36: 145-164.

McDonald, G.I., Tanimoto, P.D., Rice, T.M., Stewart, J.E., Tonn, J.R., Hall, D.E., Zambino, P.J., Klopfenstein, N.B., Kim, M.-S. 2005. Root Disease Analyzer – Armillaria Response Tool (ART). Fuels Planning: Science Synthesis and Integration Environmental Consequences Fact Sheet 13. Research Note RMRS-RN-23-12-WWW. Fort Collins: CO. USDS, Forest Service, Rocky Mountain Research Station. 2 p.

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Stewart, J.W., Kim, M.-S., James, R.L. Dumroese, R.K., Klopfenstein, N.B. 2006. Molecular characterization of *Fusarium oxysporum* and *Fusarium commune* isolates from a conifer nursery. Phytopathology 96: 1124-1133.

Rust Committee Report
Chair: Holly Kearns

The Rust Committee luncheon was held on October 17 with over 40 participants and many interesting reports on recent, current, and pending activities.

REPORTS

Dave Shaw—Along with Matt Betts and Tom Manning investigated Clark's Nutcracker and the population structure of whitebark pine (WBP) along 15 - 5,000 m transects. Eight transects were located within relatively large contiguous pieces of WBP habitat along the Oregon Cascade Crest, and seven in smaller, more isolated areas. The cone year was off, so the fact that we saw fewer birds in the contiguous area than the isolated area can't be really taken to the bank. However, we found interesting differences in population structure of the trees, with a 'normal' reverse J in the isolated populations and a disrupted population structure in the contiguous area. The possibility exists that Clark's Nutcracker might be moving seed from healthy populations of WBP to areas where it has been disrupted. We hope to follow up with more detailed nutcracker studies.

Blakey Lockman and Gregg DeNitto—FHP Missoula, continue to update the Whitebark Limber Pine Information System (WLIS). Data from FIA phase 2 plots from RMRS and PNW have been obtained and are being incorporated into the Level 1 data. A preliminary display of WLIS with this new data was shown and several discrepancies were noted. Blakey and Gregg will work on these discrepancies and make needed corrections. An updated database will be on the R1/4 FHP website after corrections are made plus newly submitted data. Those working on limber pine and whitebark pine are encouraged to input their data into WLIS and submit it to Blakey/Gregg to expand the database.

Richard Reich—I am actively working on two projects involving pine stem rusts of lodgepole pine

in the sub boreal spruce biogeoclimatic zone. I presented a poster on comandra blister rust risk in the poster session. The poster showed preliminary results of an assessment of a comandra resistance trial designed to test the susceptibility of 130 families of lodgepole pine. In addition to testing susceptibility, we mapped the location of both the alternate host for comandra blister rust, (*Geocaulon lividum*) and stalactiform blister rust (*Castelleja* spp, and *Melampyrum linear*). We also created an accurate terrain model using differential GPS. The objective of this alternate host component is to model the risk based on distance to the alternate host and topography.

A second project involves a western gall rust assessment of 3 lodgepole pine clonal seed orchards. This project involves spatial autocorrelation and regression analysis to examine infection gradients and site factors, as well as identify resistant seed lots. This project also involves a foliar disease assessment and analysis to determine the pattern of inheritance between western gall rust and 3 different foliar diseases (*Lophodermella concolor*, *Dothistroma pini*, and *Elytroderma deformans*).

Rich Hunt—I would like people to know what's happening with our programs.

First the interior program is steered through the bureaucracy by BC Forest Service geneticist Mike Carlson. Over the past few years Mike has made full sib crosses among putative resistance traits identified in Idaho using Idaho materials imported into BC several years ago. We have examined data from INOC years 2003 to 2006. No-spots seedlings are lacking, no needles fall off, there are no fungicidal short shoot reactions. The seedlings behave just the same as if their parents came from north of the border; i.e., most are severely cankered, and a few display slow-canker-growth resistance. We have

coned ID survivors from our famous Vlem plantation and inoculated them at the same time as ID seedlings and the 2007 spotting data is following in the same pattern as our published results with BC selections; i.e., they are displaying age related resistance.

Second, the coastal program is steered thru the bureaucracy by BC Forest Service geneticist John King. Over the past several years John has crossed the best parents, i.e., parents whose OP offspring had a high proportion of slow-canker-growth resistance. The seed from the few slow-canker-growth seedlings that matured, and we crossed, was all cooked in a faulty refrigerator. I'd like to say that the parental full sib crosses have yielded families with spectacular levels of slow-canker-growth resistance, but they have not. The resistance levels are extremely poor, being much lower than OP off-spring! On the other hand, so far the field trials are showing encouraging results. Because we did not get a large sample size of slow-canker-growth resistance in the full-sib offspring, our attempts to correlate resistance to levels of PR 10 and a chitanase isoform have been thwarted.

Additionally, we have identified a dozen homozygous Cr2 individuals in plantations established from Champion Mine seed purchased from the Dorena program. Some pollen has been used as boosters in the seed orchards. Unfortunately we are finding a number of plantations suffering red-banded needle disease. One plantation suggests that OR white pine sources are more susceptible to *Dothistroma* than local white pine sources.

Bryce Richardson—The RMRS-Moscow lab is currently working on several projects for two invasive rust pathosystems: white pine blister rust and Ohia rust. For blister rust, recent publications include: assessing genetic diversity among different hosts and the pathogenicity of *C. ribicola* isolates to *Pedicularis racemosa* (Richardson et al. 2007a), a first report of infection on *Pedicularis bracteosa* (Zambino et al. 2007), and the influence of host resistance on genetic diversity of *C. ribicola*

(Richardson et al. in press). Ongoing projects include intercontinental phylogeography of *C. ribicola*. For the hosts, recent publications include bioclimatic modeling of whitebark pine and western white pine (Warwell et al. 2007, Richardson et al. 2007b). Ongoing projects involve range-wide population genetics and genecology of western white pine using molecular and quantitative approaches. Population genetics of Ohia rust (*Puccinia psidii*) is also an ongoing project with collaborators in Region 5, Phil Cannon, and University of Hawai'i..

References:
Richardson, B.A., Zambino, P.J., Klopfenstein, N. B., McDonald, G. I., Carris, L. M. 2007a. Assessing host specialization among aecial and telial hosts of the white pine blister rust fungus, *Cronartium ribicola*. Canadian Journal of Botany. 85: 299-306.

Richardson, B.A., Warwell, M.V. Kim, M-S., Klopfenstein, N. B., McDonald, G. I. 2007b. Integration of population genetic structure and plant response to climate change: sustaining genetic resources through evaluation of projected threats. Proceedings: Advances in threat assessment. Boulder, CO. July, 2006.

Warwell, M.V., Rehfeldt, G.E., Crookston, N.L. 2007. Modeling contemporary climate profiles and predicting their response to global warming for whitebark pine (*Pinus albicaulis*). In EM Goheen and RA Sniezko, tech. coords. Proceedings of the conference whitebark pine: a Pacific Coast perspective. 2006 August 27-31. Ashland, OR. R6-NR-FHP-2007-01. Portland, OR: USDA Forest Service, Pacific Northwest Region, p. 139-142.

Zambino, P.J., Richardson B.A., McDonald, G.I. 2007. First Report of the White Pine Blister Rust Fungus, *Cronartium ribicola*, on *Pedicularis bracteosa*. Plant Disease 91: 467.

Patricia Maloney—Det Vogler and I got funded for a project titled "Natural and anthropogenic threats to white pines from lower montane forests to subalpine woodlands of the Lake Tahoe Basin: An ecological and genetic assessment for conservation, monitoring, and management" But my second comment was the importance of recording detailed information about microhabitat for white pine recruits (e.g., rock, tree, and/or shrub nurse, substrate growing in: exposed

soil, litter and soil, rock, etc..). The last thing was that we have 61 whitebark families from the Lake Tahoe region at IFG with Det and Annie for resistance evaluations and some other analyses as well.

Michelle Cleary—A pre-announcement for an upcoming meeting being organized by Stefan Zeglen and myself entitled: 'Nakusp II: Western White Pine Management Conference' which will be held June 16-19th in Vernon and Nakusp, British Columbia. The meeting will focus on silviculture management of western white pine in B.C, advances in the genetic resistance of western white pine, the status of white pine tree improvement program after two decades and its future in B.C. The meeting will consist of several presentation sessions with invited speakers, an evening poster session, and several field trips to genetic gain resistance field trials, seed orchards, and white pine management areas as we tour from Vernon to Nakusp and back over 3 days. A formal announcement of the meeting will be distributed to the WIFDWC email listing.

Bill Jacobi—Pruning trials at the Sand Dunes National Park and a campground on the Medicine Bow National Forest were revisited after two years. Pruning to remove cankers and to remove lower branches to prevent lower stem infections did not induce Ips or other bark beetle attack. Only one canker scribing attempt failed at the Sand Dunes. We are in the process of assessing the status of the trees in the Medicine Bow National forest but in the first 100 trees there did not appear to be any negative issues associated with pruning.

Amy Ramsey—Washington DNR, 2006-2007: Installed six F3 progeny Western white pine blister rust resistance field trials in western Washington. Continued to monitor 22 F2 progeny Western white pine sites for incidence and severity of white pine blister rust.

John Schwandt—Whitebark Pine (WBP):
WBP Restoration program funded 24 projects for whitebark pine restoration using a total of $250K

FHP funds that were matched with over $290K of other funds (hand out provided with more details). A second year of the program is planned (see John for details).

We installed 120 permanent monitoring plots in the Idaho Panhandle NF on four ridges that had recently been burned (Selkirk Mountains) to improve WBP regeneration. We will monitor the occurrence of natural regeneration over time in different burn intensities; 1/50 ac plots were established in burned, partially burned and unburned areas to provide comparative data. Plans call for revisit every 5 years.

We conducted 12 year re-measurements of 5 areas with permanent WBP regeneration plots on the Idaho Panhandle NF (Lunch Peak, Roman Nose 1&2, Trout Lake, Long Mtn.). We will be doing data analysis over the winter to compare infection levels and mortality between areas over the last 12 years.

WBP seed planting study (Vinegar Hill and Mt Batchelor) in coop with R-6. Two years after planting 700 seed with 5 different treatments to enhance germination or reduce rodent predation, we had a little over 50% germination in seed that had a warm stratification treatment compared to less than 10% in the controls.

We conducted a small sample survey in the Selkirk Mountains of northern Idaho to try to quantify the amount of live WBP remaining after a major MPB outbreak. We found that 90+ % of the mature WBP had been killed by MPB, but there were still nutcrackers around foraging cones on the few remaining mature trees. Only a few natural WBP regeneration were tallied, so it appears that this stand which had been predominantly WBP in the past was being converted to other species.

John Schwandt—Western White Pine (WWP):
We are in the process of analyzing 15 years of data on F2 plantations of WWP to determine rust infection and mortality levels over time (in coop with Brennan Ferguson - IDL)

Amy Eckert finished a MS through OSU that looked at abnormal cankers in F2 stands of WWP. She found that cankers could be classified into 3 groups (normal, abnormal and something in-between) and that the rust girdling rates were significantly different between the groups (or at least 2 of the groups)

F2 canker study - we have been following the annual growth of more than 100 branch cankers on 10 year old F2 stock for the past 4-5 years and have found that the average annual growth rate towards the bole is 2.1 inches, and that most of the cankers >24 inches from the bole had died. Most cankers that were within 6" of the bole grew into the bole.

Bear damage on pruned trees has become a major problem in some F2 plantations of WWP that have been pruned. Some areas have 30-40% of trees damaged and considerable mortality, so we are looking for suggestions to reduce the losses from bears.

White pine gene conservation strategy part of a national effort to develop gene conservation strategies for exotic insects and diseases was the focus of a meeting at the Dorena Genetic Center.

Kelly Burns—reported the following for the Rocky Mountain Region (R2) in 2007

Field work is completed for the Evaluation Monitoring project, Limber Pine Health in the Rocky Mountain Region, a cooperative effort between R1 (Jackson/Walla), R2, RMRS, and CSU. Eighty-three long-term monitoring plots were established throughout the distribution of limber pine in northern CO, WY, central MT, and ND. The objective is to assess the long-term ecological health of limber pine within WPBR-infested and threatened areas of the central Rocky Mountains.

The R2 WPBR Management Guide is complete and is currently being published as a General Technical Report that will be available in 2008 (Burns, KS; Schoettle, AW; Jacobi, WR; Mahalovich, MF. In

Press. Options for the Management of White Pine Blister Rust in the Rocky Mountain Region. RMRS-GTR-xxx).

CSU, R2-FHM, and RMRS are facilitating the collection of cones from phenotypically resistant white pines on the Roosevelt and Medicine Bow NFs for future gene conservation, resistance screening, and restoration planting.

CSU, R2-FHM, Great Sand Dunes National Park and Preserve, and the Medicine Bow NF are working together on a pruning and canker removal project aimed at prolonging the life of high-value limber and bristlecone pines. Field work is complete, data is being analyzed, and a report will be available in 2008.

R2-FHM is involved in several projects with RMRS and Dorena Genetic Resource Center (Sneizko/Kegley) looking at WPBR resistance in limber and bristlecone pine.

> **Bristlecone:** Seeds from 184 families have been collected and grown. Seeds were sown in 9/02 and seedlings inoculated in 9/05. Symptoms are still developing. About half of the seedlings are showing stem symptoms but it is too early to identify family trends.

> **Limber**: (STDP Project R2-2006-02): Seeds were sown in 7/06 and seedlings inoculated in 9/06. One year after inoculation, several families are showing segregation suggestive of an R gene, however, we have not seen the classic hypersensitive reaction that is seen on western white pine, sugar pine, and southwestern white pine. Some of the seedtrees whose progeny have shown canker-free traits in the artificial inoculation study are threatened by mountain pine beetles. We are working with the USFS districts to protect these seedtrees prior to the 2008 beetle flight and until the genotypes have been adequately conserved.

Brennan Ferguson—Continue data collection from permanent plots installed in Idaho Department of Lands (IDL) plantations to monitor performance of rust-resistant western white pine (F2 stock). Currently writing a summary report for the IDL data, and am collaborating with Holly Kearns and John Schwandt on analysis of the combined data set for the IDL and USFS F2 monitoring data.

Based on observations and data from the IDL F2-monitoring plantations that show a high percentage of lethal, or potentially lethal, branch and stem cankers occur in the lower 18-24" of the bole of F2 stock, Holly Kearns and I have proposed a long-term, early-pruning study in F2 stock that will apply various bole-pruning treatments to trees 2-6 feet in height.

John Schwandt and I continue to offer our blister rust workshop, focusing on performance and young-stand management of F2 western white pine in the Inland Empire, as part of the cooperative IDL-USFS annual trainings. This year's workshop was held in September in Coeur d'Alene, Idaho, with 25 students in attendance.

Richard Sniezko—The next meeting of IUFRO 2.02.15 – Breeding and genetic resources of five-needle pines will be Sept. 22-26, 2008 in Korea. Meeting details are available at http://iufro.kfrigene.net/. The Conference welcomes presentations on quantitative and molecular genetics, resistance to diseases and pests, hybridization, selection and improvement, genecology and conservation genetics in all five-needle pines. The conference will comprise presentations on all these aspects as well as excursions to the natural stands of *Pinus Koraiensis* in Sorak and Odae mountains. A visit to the seed orchard of *P. Koraiensis* will be included. A special presentation on the Korean effort to control white pine blister rust by eradication of *Ribes* is also scheduled. Please contact Dr. King, Dr. Noh or myself with any questions. We also welcome your suggestions for the continue working of this IUFRO group.

Business Meeting Minutes
Michael McWilliams

Old Business

The 2006 proceedings was published and distributed, and there were no additional business notes or comments. It was moved by E. Goheen and seconded by Baker to approve proceedings, and it carried.

Kudos were offered to the organizing committee: Jim Worral, MaryLou Fairweather, Bob Mathiasen, and to Dan Rierson on the projector and Bobbie Fitzgibbons and Barb Walters for check-in.

Treasurer's report. John Schwandt
We have been given a new Tax ID number: 35-2307554. Please use this as necessary in the future.

2007 Hazard Tree Conference –June 11-15 Midway, Utah. Balance as of last report (12/31/06): $344.54 Conference Expenses actually exceeded the registration income, but Pete Angwin was able to get his office to help sponsor the conference so we were able to avoid a negative balance. We now have a positive balance of: $971.54

2007 WIFDWC conference - Oct 16-19 Sedona, Arizona. Balance as of last report (12/31/06): $9,857.28
 Expenses $9,431,11
 Registration/Income: $15,458.45
 Interest/Service charges $109.71
 Payoff of remaining balance for Proceedings
  CD's $4,075.00
 Current Balance (12/31/07) $11,899.33

Total amount in Account as of 12/31/2007 = $12,870.87 (Hazard tree + WIFDWC)

Pete reported the Hazard Tree meeting was only $250 short. As an organization, WIFDWC can enter into grants and agreements.

We still owe $4000 for the historical proceedings CDs (out of $16,049 total cost), and we could take WIFDWC money and pay this off. DeNitto moved and Angwin seconded that we do not pay this off yet, and it carried.

The University of Idaho accepted Visa for registration this year, and this cost $40 per registrant ($15 to U of I, and 10% to Visa). Paying by check incurred no extra charges. Federal employees must charge it, and can't put registration on a travel claim. The U of I wired WIFDWC the money, crated a report, and we have the option of staying with them for registration and continuing to use the service. Discussion was generally favorable for the added convenience, even though it made registration more expensive.

Old Business. Giles, Jacobi, and Guyon were tasked with researching whether publication in proceedings inhits publication in peer-reviewed journals. We agreed to defer their report until the next business meeting.

Nominations. Bill Jacobi nominated Fred Baker for secretary, Greg DeNitto for chairman, and Will Littke for interim program chairman. Local arrangements will be handled by Blakey Lockman and Marcus Jackson. Schwandt moved to accept nominations, E. Goheen seconded, and it was carried. Greg DeNitto shared that the meeting would be Sept 15-18 in Missoula at the Holiday Inn Missoula Downtown at the Park, near a trail system and the river.

Discussion On Future Meetings

A protracted discussion ensued about WIFDWC in 2009, where to hold the meeting, and whether to invite the entomologists. Jacobi suggested Colorado, and said he would help but could not commit to a particular place, but could include Cortez, Durango, Pagosa Spinge, the Vail area, or Ft. Collins. Dave Shaw moved and eventually retracted his motion to invite the entomologists in '09. Jacobi moved and Kearns seconded the motion to have the 2009 meeting in Region 2, and the motion carried..

DeNitto moved and D. Shaw seconded a motion to invite the entomologists to meet with us in 2010. Discussion ensued around the fact they have a different organization, prefer concurrent sessions, and WIFDWC attendance is lower at joint meetings. Joint meetings are almost like two independent meetings with a few joint sessions. The motion initially carried 20:18, but there may have been a miscount. Filip suggested Region 6, possibly Wenatchee, as a venue. Michelle Cleary offered an invitation to Canada (Valemount) if the entomologists declined. Terry Shaw moved and Van der Camp seconded a motion to defer decision on

2010 until the 2008 business meeting. The motion failed. A straw poll among attendees carried Vailmont as the 2010 location for the meeting.

Terry Shaw moved and Thies seconded a motion to revote on inviting the entomologists. This motion passed. Van der Kamp motioned to rescind previous motion to invite entomologists, Thies seconded, and this motion passed. Jacobi moved and Blodgett seconded a motion to invite the entomologists to a joint meeting in 2010. Discussion paralleled previous discussion concerning the differences in our orgainizations, the fact we don't want to loose control of our meeting, this is our chance to talk fungi, and that other opportunities exist to talk with the entos. The motion failed.

Terry Shaw moved and Angwin seconded a motion to have the 2010 meeting in Valemount, British Columbia. The motion passed.

Items of interest. -Lockman contacted Dale Berghdahl concerning the WIFDWC listserv. He intends to maintain this list, the secretary needs to send changes in the mailing list, and ask Dale for updates periodically. The directory is of members, and has a category for invited speakers, etc. One can register on the listserv without talking to WIFDWC.

Publication of the proceedings through Utah State costs $1200-$1400 for the whole process. It is up to the secretary whether to avail themselves of this option. A comment was made that regional reports need to be more concise to allow presentation by all organizations.

New Business

Harry Kope made a motion to form a committee for foliar diseases, Cleary seconded. T. Shaw proposed a climate change committee. An additional standing committee would require amendment of the bylaws, Giles commented that the establishment of a committee would need to go to all WIFDWC members. Kope withdrew the motion. There was discussion on the need for an interim foliar and shoot disease committee. The chair proposed appointing an ad hoc committee, and a straw vote carried the creation of a new informal committee on foliar and shoot diseases convened by Kope, and a climate change committee convened by T. Shaw.

Angwin nominated Dave Schultz as a new honorary life member, and this passed. Thies requested inclusion as an honorary life member, and this passed.

Angwin moved that the WIFDWC chair write a letter to the Chief of the Forest Service and Rob Mangold's FHP requesting that the national office fill the national pathologist position ASAP, with a pathologist, and that one of their duties be to coordinate Hazard Tree issues and efforts. Schwandt seconded the motion. T. Shaw commented that the Chief would have to assign additional duties, and Angwin commented that the Hazard Tree effort was not well defined. D. Goheen proposed additional duties concerning introduced pathogens and climated change. The motion was amended to state the letter be sent to the Chief, and that duties include coordination of Hazard Tree issues nationwide, and coordination of introduced diseases and climate change issues. The motion passed.

The Outstanding Achievement Award committee needs a new member, and Ellen Goheen volunteered. A suggestion was made to reprint the bylaws and a list of significant motions in the proceedings.

DeNitto made a motion for adjournment, Little seconded, and the motion passed.

In Memoriam: Lee Paine

Dr. Lee Paine, a retiree of the Pacific S.W. Forest Experiment Station in Albany, California, and past member of WIFDWC, died in Green Valley, Arizona on January 9, 2007.

Lee was born in Kansas City, but moved with his family to Chicago, and later to Evanston, Ind. He went to Northwestern Univ. for a short period of time, but then transferred to the Univ. of Idaho where he received his bachelor's degree in 1943.

He served in the Air Force (OSS) in Italy during the war, and is reputed to have served behind enemy lines. He was fluent in Italian, Spanish, German, and Romanche. These languages enabled him to serve well in the OSS capacity.

Following the war he received his Masters degree from the Univ. of Idaho (1947), and then moved to Zurich, where he earned a Doctor of Science (1951) from the Swiss Federal Institute of Technology. His research there involved the susceptibility of pear trees to penetration and toxic damage by mistletoe (Viscum album).

Lee joined the Forest Service in 1957, where he began the study of decays of conifers. After a few years, Lee left the studies on decays, and began what became a massive research project on Hazardous Tree Failures on Forested Recreation Sites West of the Mississippi. This resulted in an actuarial analysis of the causes and predisposing conditions for virtually every campground-recreational area tree failure for more than a decade.

Lee retired from the Forest Service in the mid 1980s, and continued to live in his home in Richmond, Ca. He loved to travel, particularly to Mexico, and at one point considered living there. However, in 1991 he moved to Green Valley, Arizona, where he lived until he passed away. Lee was never married, and has no surviving heirs to my knowledge.

<div align="right">by Bob Scharf and Nancy Gillette, PSW Station</div>

Standing Committees and Chairs, 1994—2006

Committee	Chairperson	Term
Hazard Trees	J. Pronos	1994-2005
	P. Angwin	2006
Dwarf Mistletoe	R. Mathiasen	1994—2000
	K. Marshall	2001—2003
	F. Baker	2004—2006
Root Disease	G. Filip	1994—1995
	E. Michaels Goheen	1996—2005
	B. Ferguson	2006
Rust	J. Schwandt	1994, 2005
	R. Hunt	1995-2004
	H. Kearns	2006
Disease Control[*]	B. James	1995-2002
Nursery Pathology	B. James	2002-2005
Ad Hoc Committes 2007		
Climate Change	T. Shaw	2007
Foliar and Shoot Diseases	H. Kope	2007

*Disease Control was disbanded and Nursery Pathology established in 2002.

Past Annual Meeting Locations and Officers

Meetings and Officers, 1953—1989

Annual	Year	Location	Chairperson	Secretary-Treasurer	Program Chair	Local Arrangements
1	1953	Victoria, BC	R. Foster			
2	1954	Berkeley, CA	W. Wagener	P. Lightle		
3	1955	Spokane, WA	V. Nordin	C. Leaphart	G. Thomas	
4	1956	El Paso, TX	L. Gill	R. Davidson	V. Nordin	
5	1957	Salem, OR	G. Thomas	T. Childs	R. 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	J. Hadfield
27	1979	Salem, OR	T. Laurent	T. Hinds	B. van der Kamp	L. Weir
28	1980	Pingree Park, CO	R. Gilbertson	O. Dooling	J. Laut	M. Schomaker
29	1981	Vernon, BC	L. Weir	C.G. Shaw III	J. Schwandt	D. Morrison R. Hunt
30	1982	Fallen Leaf Lake, CA	W. Bloomberg	W. Jacobi	E. Hansen	F. Cobb J. Parmeter
31	1983	Coeur d'Alene, ID	J. Laut	S. Dubreuil	D. Johnson	J. Schwandt J. Byler
32	1984	Taos, NM	T. Hinds	R. Hunt	J. Byler	J. Beatty E. Wood
33	1985	Olympia, WA	F. Cobb	W. Thies	R. Edmonds	K. Russell
34	1986	Juneau, AK	K. Russell	S. Cooley	J. Laut	C.G. Shaw III
35	1987	Nanaimo, BC	J. Muir	G. DeNitto	J. Beatty	J. Kumi
36	1988	Park City, UT	J. Byler	B. van der Kamp	J. Pronos	F. Baker
37	1989	Bend, OR	D. Goheen	R. James	E. Hansen	A. Kanaskie

Bylaws were amended in 1989 to split the office of Secretary-Treasurer.

Meetings and Officers, 1990—2007

Annual	Year	Location	Chair-person	Secretary	Treasurer	Program Chair	Local Arrangements	Historian	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—D. Morrison—D. Morrison	J. Adams—J. Adams—J. Adams
47	1999	BreckenridgeCO	F. Baker	E. Michaels Goheen	J. Schwandt	J. Taylor	D. Johnson		
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		

Bylaws passed in 1998 WIFDWC Business Meeting identify officers as chairperson and secretary elected at annual business meeting and treasurer and historian, elected every five years.

WIFDWC MEMBERS

Judy Adams
last attended 2007
USDA, Forest Service
2150 Centre Ave., Bldg A, Suite 331
Fort Collins CO 80526 USA
(970) 295-5846
jadams04@fs.fed.us

Mike Albers
last attended 2004
Minnesota Dept. of Natural Resources
1201 East Hwy. 2
Grand Rapids MN 55744 USA
Mike.albers@dnr.state.mn.us

Stew Alcock
last attended 2006
Integrated ProAction Corp.
P.O. Box 2110
Chetwynd BC V0C 1J0 CANADA
(250) 788-7904
alcock@intpac.ca

Robert Anderson
last attended 2000
University of Hawaii
Pacific Coop. Studies Unit U
3190 Maile Way, Room 159
Honolulu HA 96822 USA
(808) 956-9428
robertan@hawaii.edu

Peter Angwin
last attended 2007
USDA, Forest Service
Shasta-Trinity National Forest
3644 Avtevch Parkway
Redding CA 96002 USA
(530) 226-2436
pangwin@fs.fed.us

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

Sue Askew
last attended 2006
University of British Columbia
534 Bunker Road
Victoria BC V9C 3B7 CANADA
(250) 474-5499
saskew@pfc.forestry.ca

Fred Baker
last attended 2007
Utah State University
Dept of FRWS
5230 Old Main Hall
Logan UT 84322-5230 USA
(435) 797-2550
fred.baker@usu.edu

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

Alan Baxter
last attended 2006
West Fraser Mills Ltd.
P.I.R. Division
P.O. Box 3130
Smithers BC CANADA
(250) 847-2656

Russell Beam
last attended 2006
Colorado State University
Dept. of BSPM, 885 Kline Drive
Lakewood CO 80215 USA
(303) 249-2051
russell.beam@colostate.edu

Jerome Beatty
last attended 2007
USDA, Forest Service PNRS
WWETAC, 3160 N.E. 3rd Street
Prineville OR 97754 USA
(541) 416-6582
jbeatty@fs.fed.us

Miroslava "Mirka" Bednarova
last attended 2006
Mendel University of Agriculture and
Forestry, Department of Forest
Protection and Game Management
Zemedelska 1, 613 00
Brno CZ Czech Republic
svezi.mirka@email.cz

Frank Betlejewski
last attended 2003
USDA, Forest Service
J. Herbert Stone Nursery
2606 Old Stage Road
Central Point OR 97502 USA
(541) 858-6127
fbetlejewski@fs.fed.us

Peter V. Blenis
last attended 1995
University of Alberta
Dept. Renewable Resources
751 General Services Bldg
Edmonton AB T6G 2H1 CANADA
(780) 492-0106
peter.blenis@ualberta.ca

James Blodgett
last attended 2007
USDA, Forest Service
Rapid City Service Center
8221 South Highway 16
Rapid City SD 57702 USA
(605) 716-2783
jblodgett@fs.fed.us

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

Crystal Braun
last attended 2007
University of Northern British Columbia
207-1380 Foothills Blvd
Prince George BC V2M 6V8 CANADA
250-960-5297
braunca@gmail.com

Nicholas Brazee
last attended 2005
University of Washington
1714 E. Spring St
Seattle WA 98122 USA
206-543-1486
nbrazee@u.washington.edu

Kerry Britton
last attended 2005
USDA, Forest Service
kbritton01@fs.fed.us

Pat Brochez
last attended 2006
BC Ministry of Forests, Bag 3500
Burns Lake BC V0J 1E0 CANADA
(250) 692-2225
Pat.Brochez@gov.bc.ca

Anna Brown
last attended 2006
Forest Research Agency
Alice Holt Lodge
Farnham Surrey UK GU10 4LM UK
anna.brown@forestry.gsi.gov.uk

John Browning
last attended 2007
Weyerhaeuser Forestry
P.O. Box 420, 505 N. Pearl St.
Centralia WA 98531 USA
(360) 339-1721
john.browning@weyerhaeuser.c

Lindsay Bulman
last attended 2006
ENSIS
49 Sala Street
Rotorua NZ New Zealand
lindsay.bulman@ensisjv.com

Kelly Burns
last attended 2007
USDA, Forest Service
740 Simms Street
Golden CO 80401 USA
303-375-5070
ksburns@fs.fed.us

Faith Campbell
last attended 2003
American Lands Alliance
726 7th St. SE
Washington DC 20003 USA
(202) 547-9120
phytodoer@aol.com

Sally Campbell
last attended 2007
USDA, Forest Service
620 SW Main St., Suite 400
Portland OR 97205 USA
503-808-2034
scampbell01@fs.fed.us

Philip Cannon
last attended 2007
USDA, Forest Service
1323 Club Road
Vallejo CA 94592 USA
(707) 562-8913
pcannon@fs.fed.us

Kristen Chadwick
last attended 2007
USDA, Forest Service
Deschutes National Forest
1001 SW Emkay
Bend OR 97702 USA
(541) 383-5587
klchadwick@fs.fed.us

Gary Chastagner
last attended 2007
Washington State University
WWREC
7612 Pioneer Way E.
Puyallup WA 98371 USA
(253) 445-4528
chastag@wsu.edu

Michelle Cleary
last attended 2007
BC Ministry of Forests and Range
Southern Interior Forest Region
515 Columbia Street
Kamloops BC V2C 2T7 CANADA
(205) 828-4583
Michelle.Cleary@gov.bc.ca

David Coates
last attended 2006
BC Ministry of Forests
Northern Interior Forest Region
Bag 6000, 3333 Tatlow Road
Smithers BC V0J 2N0 CANADA
(250) 847-6386
Dave.Coates@gov.bc.ca

Brad Collins
last attended 2006
Oregon State University
3405 NW Orchard Avenue, Apt. 127
Corvallis OR 97330 USA
(541) 754-8560
collinb2@science.oregonstate.edu

David Conklin
last attended 2007
USDA, Forestry and Forest Health
333 Broadway Blvd. SE
Albuquerque NM 87102 USA
505-842-3288
daconklin@fs.fed.us

Craig Cootsona
last attended 2005
University of Washington
3700 Crystal Ridge Drive
Puyallup WA 98372 USA
253-848-0214
ccootsona@qwest.net

Mike Cruickshank
last attended 2005
Canadian Forest Service, Pacific
Forestry Centre, 506 W. Burnside Rd.
Victoria BC V82 1M5 CA
250-363-0641
mcruicks@pfc.forestry.ca

Amanda Crump
last attended 2006
Colorado State University, Dept. of
BSPM, Campus Delivery 1177
Ft. Collins CO 80523-1177 USA
(970) 491-4671
amanda.crump@colostate.edu

Angie Dale
last attended 2006
University of Northern British Columbia
3333 University Way
Prince George BC V2N 4Z9 CANADA
(250) 960-6442
dale@unbc.ca

Norm Dart
last attended 2006
Washington State University
Research and Extension Center
7612 Pioneer Way East
Puyallup WA 98371 USA
(253) 445-4596
normdart@wsu.edu

Lance David
last attended 2007
Information Technology Experts
2120 S. College Ave.
Fort Collins CO 80525 USA
970.295.5856
ldavid@fs.fed.us

Tom DeGomez
last attended 2007
University of Arizona
P O Box 15018 NAU
Flagstaff AZ 86011 USA
degomez@ag.arizona.edu

Annette Delfino-Mix
last attended 2007
USDA, Inititute of Forest Genetics
2480 Carson Rd.
Placerville CA 95667 USA
(530) 295-3023
amix@fs.fed.us

Gregg DeNitto
last attended 2007
USDA, Forest Service
Northern Region, Forest Health
Protection, P.O. Box 7669
Missoula MT 59807 USA
406-329-3637
gdenitto@fs.fed.us

Magaret Dick
last attended 2005
New Zealand Forest Research Institute
Private Bag 3020
Sala St. Rotorua NZ
0064 7 343 5531
margaret.dick@ensisjv.com

Ron Diprose
last attended 2006
BC Ministry of Forests and Range
Coast Forest District
7077 Duncan Street
Powell River BC V8A 1W1 CANADA
604-485-0723
Ron.Diprose@gov.bc.ca

G. Keith Douce
last attended
University of Georgia
Dept of Entomology, P.O. Box 748
Tifton GA 31793 USA
(229) 386-3298
kdouce@uga.edu

Marla C. Downing
last attended 2007
USDA, Forest Service
Forest Health Portection, FHET
2150 Center Ave., Bldg, A, Suite 331
Fort Collins CO 80526 USA
(970) 295-5843
mdowning@fs.fed.us

Amy Eckert
last attended 2005
Oregon State University
College of Forestry
RH 321
Corvallis OR 97331 USA
541-760-3039
amy.eckert@oregonstate.edu

Robert Edmonds
last attended 2006
University of Washington
College of Forest Resources
Box 352100
Seattle WA 98195 USA
(206) 685-0953
bobe@u.washington.edu

Jim Ellenwood
last attended 2004
USDA, Forest Service
2150 Centre Ave., Bldg A, Suite 331
Fort Collins CO 80526
jellenwood@fs.fed.us

Marianne Elliott
last attended 2005
Washington State University
Puyallup Res. and Ext. Center
Puyallup WA USA
melliott@puy.ad.wsu.edu

Melissa Erickson
last attended 2004
USDA, Forest Service, PSRS
P.O. Box 245
Berkeley CA 94701
(510) 559-6470
melissaerickson@fs.fed.us

Mary Lou Fairweather
last attended 2007
USDA, Forest Service
Southwest Forest Sciences Complex
2500 S. Pine Knoll Rd.
Flagstaff AZ 86001 USA
(928) 556-2075
mfairweather@fs.fed.us

Brennan Ferguson
last attended 2007
Ferguson Forest Pathology
P.O. Box 2127
Missoula MT 59806-2127 USA
(406) 239-7761
brennan@fergusonforestpatholo

Gregory Filip
last attended 2007
USDA, Forest Service
P.O. Box 3623
Portland OR 97203 USA
(503) 808-2997
gmfilip@fs.fed.us

Bobbe Fitzgibbon
last attended 2007
USDA, Forest Service
2500 South Pine Knoll Drive
Flagstaff AZ 86001 USA
928.556.2072
bfitzgibbon@fs.fed.us

Susan Frankel
last attended 2007
USDA, Forest Service, PSRS
800 Buchanan Street
Albany CA 94710 USA
510-559-6472
sfrankel@fs.fed.us

Christine Friedrichsmeier
last attended 2006
BC Ministry of Forests
Vanderhoof Forest District
P.O. Box 190, 1522 Highway 16 East
Vanderhoof BC V0J 3A0 CANADA
(250) 567-6425

Christine.Friedrichsmeier@gov.b
Amy Gannon
last attended 2007
Montana DNRC
2705 Spurgin Road
Missoula MT 59804 USA
406-542-4283
agannon@mt.gov

Brian W. Geils
last attended 2007
USDA, Forest Service
Rocky Mountain Research Station
2500 S. Pine Knoll Dr.
Flagstaff AZ 86001 USA
(928) 556-2076
bgeils@fs.fed.us

Daniel Gilmore
last attended 2004
University of Minnesota
1861 Highway 169 East
Grand Rapids MN 55744
dgilmore@umn.edu

John Goetz III
last attended 2005
Washington State University
Dept. Plant Pathology
Pullman WA 99164 USA
208-883-2310
JohnGoetzIII@hotmail.com

Ellen Goheen
last attended 2007
USDA, Forest Service
Southwest Oregon FID Service Centre,
2606 Old Stage Road
Central Point OR 97502 USA
(541) 858-6126
egoheen@fs.fed.us

Donald Goheen
last attended 2007
USDA, Forest Service
Southwest Oregon FID Service Centre,
2606 Old Stage Road
Central Point OR 97502 USA
(541) 858-6125
dgoheen@fs.fed.us

Betsy Goodrich
last attended 2007
Colorado State University
Dept. of Bioagr. Sciences and Pest
Mgmt., 1177 Campus Delivery
Fort Collins CO 80523-1177 USA
970-491-4671
betsy.goodrich@colostate.edu

Tom Gordon
last attended 2003
University of California, Davis
Department of Plant Pathology
University of California, Davis
Davis CA 95616 USA
trgordon@ucdavis.edu

Jesus Guerra-Santos
last attended 2004
Universidad Nacional Autonoma de
Mexico, Km 2.5 Carr. Cuautitlan-
Teoloyucan, San Sebastian Xhala
Cuautitlan Izcalli Mexico c.ps4700
Mexico
52 (55) 56231834

John Guyon
last attended 2007
USDA, Forest Service
Ogden Field Office
4746 South 1900 East
Ogden UT 84403 USA
801-476-4420
jguyon@fs.fed.us

Jim Hadfield
last attended
USDA, Forest Science Lab
1133 N. Western Ave.
Wenatchee WA 98801 USA
(509) 664-2777
jshadfield@fs.fed.us

Leila Hadj-Chikh
last attended 2004
WCS, 3985 18th St.
San Francisco CA 94114
lhadjchikh@wcs.org

Susan Hagle
last attended 2003
USDA, Lochsa Ranger Station
Rte 1 Box 398
Kooskia ID 83539 USA
(208) 926-6416
shagle@fs.fed.us

Erin Hall
last attended 2006
BC Ministry of Forests
Northern Interior Forest Region
Bag 6000, 3333 Tatlow Road
Smithers BC V0J 2N0 CANADA
(250) 847-6300
Erin.Hall@gov.bc.ca

John Hanna
last attended 2005
USDA, Forest Service, RMRS
1221 S. Main St.
Moscow ID 83843 USA
(208) 883-2354
jhanna@fs.fed.us

Paul Hanna
last attended 2006
Silverwood Consulting
4443 Keith Avenue
Terrace BC V8G 1J7 CANADA
(250) 635-0766
Silverwood.Consulting@telus.n

Everett Hansen
last attended 2006
Oregon State University
Dept of Botany and Plant Pathology,
Cordley Hall
Corvallis OR 97331 USA
(541)-737-5243
hansene@science.oregonstate.edu

Kevin Hardy
last attended 2006
BC Ministry of Forests
Coast Forest Region
2100 Labieux Road
Nanaimo BC V9T 6E9 CANADA
(250) 751-7093
Kevin.Hardy@gov.bc.ca

Jeri Lyn Harris
last attended 2005
USDA, Forest Service
740 Simms St.
Golden CO 80401 USA
303-236-3760
jharris@fs.fed.us

Erin I. Havard
last attended 2006
BC Ministry of Forests
Northern Interior Forest Region
Bag 6000, 3333 Tatlow Road
Smithers BC V0J 2N0 CANADA
(250) 847-6327
Erin.I.Havard@gov.bc.ca

Paul Hennon
last attended 2007
USDA, Forest Service
2770 Sherwood Lane, Suite 2A
Juneau AK 99801 USA
907-586-8769
phennon@fs.fed.us

Tom Heutte
last attended 2004
USDA, Forest Service
2770 Sherwood Lane, Suite 2A
Juneau AK 99801 USA
theutte@fs.fed.us

Diane Hildebrand
last attended 2005
USDA, Forest Service
Westside Service Center
16400 Champion Way
Sandy OR 97055 USA
(503) 668-1474
dhildebrand@fs.fed.us

Janice Hodge
last attended 2006
JCH Forest Pest Management
182 Horner Road
Lumby BC V0E 2G7 CANADA
(250) 547-6452
jch@cablelan.net

Robert Hodgkinson
last attended 2006
BC Ministry of Forests
Northern Interior Region
1011 4th Avenue
Prince George BC V2L 3H9 CANADA
(250) 565-6122
Robert.Hodgkinson@gov.bc.ca

James Hoffman
last attended 2007
USDA, Forest Service
1249 S. Vinnell Way
Boise ID 83703 USA
208-373-4221
jthoffman@fs.fed.us

Edward Hogg
last attended 2007
Canadian Forest Service
Northern Forestry Centre
5320-122 Street
Edmonton AB T6H 3S5 Canada
780-435-7225
thogg@nrcan.gc.ca

Bruce Hostetler
last attended 2003
USDA, Westside Service Center
16400 Champion Way
Sandy OR 97055 USA
(503) 668-1475
bhostetler@fs.fed.us

Brian Howell
last attended 2007
USDA, Forest Service
740 Simms Street
Golden CO 80401 USA
303-375-5070
bhowell@fs.fed.us

Sue Hoyles
last attended 2006
BC Ministry of Forests
Northern Interior Forest Region
1011 4th Avenue
Prince George BC V2L 3H9 CANADA
(250) 565-6214
Susan.Hoyles@gov.bc.ca

Barbara Illman
last attended 2004
USDA, Forest Products Lab
One Gifford Pinchot Dr.
Madison WI 53705-2398 USA
608-231-9269
billman@wisc.edu

Marcus Jackson
last attended 2007
USDA, Forest Service
FHP Missoula Field Office
200 E Broadway
Missoula MT 59807 USA
(406) 329-3282
mbjackson@fs.fed.us

William R. Jacobi
last attended 2007
Colorado State University
Dept. of BSPM
Fort Collins CO 80523-1177 USA
(970) 491-6927
william.jacobi@colostate.edu

Robert James
last attended 2007
USDA, Forest Service
Idaho Panhandle National Forests
3815 Schreiber Way
Coeur d'Alene ID 83815 USA
208-765-7421
rjames@fs.fed.us

Steve Jeffers
last attended 2004
118 Long Hall
Box 340315
Clemson SC 29643 USA
sjffrs@clemson.edu

William Jones
last attended 2007
USDA, Forest Service
200 WT Weaver Blvd.
Asheville NC 28802 USA
8282590526
wejones@fs.fed.us

Alan Kanaskie
last attended 2006
Oregon Dept. of Forestry
2600 State St.
Salem OR 97310 USA
(503) 945-7397
akanaskie@odf.state.or.us

Amy Kearney
last attended 2005
Montana DNRC
2705 Spurgin Rd.
Missoula MT 59804 USA
406-542-4283
akearney@mt.gov

Holly Kearnslast attended 2007
USDA, Forest Service
Panhandle National Forests
3815 Schreiber Way
Coeur d'Alene ID 83815 USA
(208) 765-7493
hkearns@fs.fed.us

Angelia Kegley
last attended 2003
USDA, Forest Service
Dorena Tree Improvement Center
34963 Shoreview Rd.
Cottage Grove OR 97424 USA
(541) 767-5703
akegley@fs.fed.us

Mee-Sook Kim
last attended 2007
USDA, Forest Service
Rocky Mountain Research Station
1221 S. Main Street
Moscow ID 83843 USA
(208) 883-2362
mkim@fs.fed.us

Ned Klopfenstein
last attended 2007
USDA, Forest Service
Rocky Mountain Research Station
1221 S. Main St.
Moscow ID 83843 USA
(208) 883-2310
nklopfenstein@fs.fed.us

Jennifer Klutsch
last attended 2007
Colorado State University
Dept. of BSPM,
C129 Plant Science Bldg.
Fort Collins CO 80523-1177 USA
(970) 491-4671
Jennifer.Klutsch@colostate.edu

Scott Kolpak
last attended 2003
USDA, Forest Service
Dorena Tree Improvement Center
34963 Shoreview Rd.
Cottage Grove OR 97424 USA
(541) 767-5717
sekolpak@fs.fed.us

Andrea Koonce
last attended 2007
USDA, Forest Service
602 S. Tippecanoe Ave.
San Bernardno CA 92408 USA
(909) 382 2673
akoonce@fs.fed.us

Harry Kope
last attended 2007
BC Ministry of Forests and Range
Forest Practices Branch
727 Fisgard Street
Victoria BC V8W 9C2 CANADA
250-387-1946
Harry.Kope@gov.bc.ca

Thomas Kubisiak
last attended 2003
USDA, Southern Research Station
23332 Highway 67
Saucier MS 39574 USA
(228) 832-2747
tkubisiak@fs.fed.us

Dominique Lejour
last attended 2002
Canadian Forest Service, Pacific
Forestry Centre, 506 W. Burnside Rd.
Victoria BC V8Z 1M5 CANADA
250-363-3751
dlejour@pfc.forestry.ca

Kathy Lewis
last attended 2006
University of Northern British Columbia
3333 University Way
Prince George BC V2N 4Z9 CANADA
(250) 960-6659
lewis@unbc.ca

Lisa Lielich
last attended 2006
BC Ministry of Forests
Prince George Tree Improvement
Station, 17000 Domano Blvd.
Prince George BC V2N 5M2 CANADA
(250) 963-8416

Dreena Lindstrom
last attended 2007
University of British Columbia
702-5639 Hampton Place
Vancouver BC V6T 2H6 CANADA
778-688-5642
dreena50@interchange.ubc.ca

Willis Littke
last attended 2007
Weyerhaeuser Forestry, WTC 1A5
32901 Weyerhaeuser Way S
Federal Way WA 98063-9777 USA
(253) 924-6995
will.littke@weyerhaeuser.com

Blakey Lockman
last attended 2007
USDA, Forest Service
Northern Region, Forest Health
Protection
P.O. Box 7669
Missoula MT 59807 USA
(406) 329-3189
blockman@fs.fed.us

Martin MacKenzie
last attended 2007
USDA, Forest Service
Stanislaus National Forest
19777 Greenley Road
Sonora CA 95370 USA
209 532 3671
mmackenzie@fs.fed.us

Helen Maffei
last attended 2007
USDA, Forest Service
Deschutes National Forest
1001 SW Emkay
Bend OR 97702 USA
541-383-5591
hmaffei@fs.fed.us

Katy Mallams
last attended 2007
USDA, Forest Service
Southwest Oregon FID Service Centre
2606 Old Stage Road
Central Point OR 97502 USA
(541) 858-6124
kmallams@fs.fed.us

Patricia Maloney
last attended 2007
University of California, Davis
Dept. Plant Pathology
PO Box 2572
Kings CA USA
530-546-3014
patricia-maloney@sbcglobal.net

Danielle Martin
last attended 2007
Oregon State University
925 NW Beca Ave
Corvallis OR 97330 USA
541-401-7835
dkhm22@yahoo.com

Roy Mask
last attended 2007
USDA, Gunnison Service Center
216 N Colorodo
Gunnison CO 81230 USA
(970) 642-1133
rmaks@fs.fed.us

Robert Mathiasen
last attended 2007
Northern Arizona University
School of Forestry
P.O. Box 15018
Flagstaff AZ 86011 USA
(928) 523-0882
robert.mathiasen@nau.edu

Andy Matysiak
last attended 2006
McElhanney Consulting Services Ltd.
P.O. Box 787
Smithers BC V0J 2N0 CANADA
(250) 847-4040
amatysiak@mcelhanney.com

Michael McWilliams
last attended 2007
Oregon Dept. of Forestry
2600 State St.
Salem OR 97310 USA
(503) 945-7395
mmcwilliams@odf.state.or.us

Jessie Micales-Glaeser
last attended 2005
USDA, Forest Products Laboratory
One Gifford Pinchot Dr.
Madison WI 53711 USA
608-231-9215
jmicales@fs.fed.us

Robert Mitchell
last attended 2006
BC Ministry of Forests
Skeena-Stikine Forest District
Bag 6000, 3333 Tatlow Road
Smithers BC V0J 2N0 CANADA
(250) 847-6300
Robert.D.Mitchell@gov.bc.ca

Phil Mocettini
last attended 2004
USDA, Boise Field Office
1249 S. Vinnell Way, Suite 200
Boise ID 83703 USA
(208) 373-4223
pmocettini@fs.fed.us

Ron Neilson
last attended 2003
USDA, Forest Service
Pacific Northwest Research Station
Corvallis OR 97330 USA
(541) 750-7303
rneilson@fs.fed.us

Maria Newcomb
last attended 2002
University of Wisconsin
Department of Plant Pathology
1630 Linden Drive
Madison WI 53706 USA
(608) 213-3941
msn@plantpath.wisc.edu

Danny Norlander
last attended 2007
Oregon State University
218 NW 35th St
Corvallis OR 97330 USA
541-829-0046
Daniel.norlander@oregonstate.e

Eun-Sung Oh
last attended 2005
Oregon State University
Dept. of Botany and Plant Pathology
2082 Cordley Hall
Corvallis OR 97331 USA
(541) 737-5242
ohe@science.oregonstate.edu

Daniel Omdal
last attended 2007
Washington Dept. of Natural Resources
WDNR Resource Protection
P.O. Box 47037
Olympia WA 98504-7037 USA
360-902-1692
dan.omdal@wadnr.gov

Hideji Ono
last attended 2007
Alberta Sustainable Resource
Development
8th Floor, 9920-108 Street
Edmonton AB T5K 2M4 CANADA
(780) 422-8801
Hideji.Ono@gov.ab.ca

Michael Ostry
last attended 2004
USDA, Forest Service
Northern Research Station
1561 Lindig Ave.
St. Paul MN 55108 USA
mostry@fs.fed.us

William Otrosina
last attended 2005
USDA, Forest Service
Southern Research Station
320 Green St.
Athens GA 30602 USA
(706) 559-4295
wotrosina@fs.fed.us

Jennifer L. Parke
last attended
Oregon State University
Dept of Crop and Soil Science ALS
3017
Oregon State University
Corvallis OR 97331 USA
(541) 737-8170
jennifer.parke@oregonstate.edu

Catherine Parks
last attended 2000
USDA, Forest Service
Pacific Northwest Research Station
1401 Gekeler Lane
LaGrande OR 97850 USA
541-962-6531
cparks01@fs.fed.us

Ian Patchett
last attended 2006
BC Ministry of Forests
Northern Interior Forest Region
1011 4th Avenue
Prince George BC V2L 3H9 CANADA
(250) 565-4440
Ian.Patchett@gov.bc.ca

Fred Peet
last attended 2003
Canadian Forest Service
Pacific Forestry Centre
506 West Burnside Rd.
Victoria BC V8Z 1M5 CANADA
(250) 363-0780
fpeet@pfc.forestry.ca

Jennifer Plummer
last attended 2006
West Fraser Mills Ltd.
P.I.R. Division
P.O. Box 3130
Smithers BC V0J 2N0 CANADA
(250) 847-2656
jennifer.plummer@westfraser.com

John Popp
last attended 2005
USDA, Forest Service
Rocky Mountain Research Station
240 W. Prospect Rd.
Fort Collins CO 80526 USA
970-498-1269
jpopp@fs.fed.us

Amy Ramsey
last attended 2007
Washington Dept. of Natural Resources
Resource Protection Division
P.O. Box 47037
Olympia WA 98504-7037 USA
360-902-1309
amy.ramsey@dnr.wa.gov

Tod Ramsfield
last attended 2001
Forest Research, Private Bag 3020
Rotorua NEW ZEALAND
64-7-343-5534
tod.ramsfield@forestresearch.co.nz

Richard Reich
last attended 2007
BC Ministry of Forests and Range
Northern Interior Forest Region
1011 4th Avenue
Prince George BC V2L 3H9 CANADA
250-565-6203
richard.reich@gov.bc.ca

Bryce Richardson
last attended 2007
USDA, Forest Service
1221 South Main Street
Moscow ID 83843 USA
208-883-2311
brichardson02@fs.fed.us

Melanie Kallas Ricklefs
last attended 2004
Washington Dept. of Natural Resources
P.O. Box 47037
Olympia WA 98504 USA
360-902-1395
melanie.kallas@wadnr.gov

Kathy Riley
last attended 2003
Washington State University
WWREC
7612 Pioneer Way E
Puyallup WA 98372 USA
(253) 445-4625
klriley@wsu.edu

Raini Rippy
last attended 2003
USDA, Forest Service
Rocky Mountain Research Station
1221 S Main St.
Moscow ID 83843 USA
(208) 883-2360
rrippy@fs.fed.us

David Rizzo
last attended 2003
University of California, Davis
Dept. of Plant Pathology
Davis CA 95616 USA
(530) 754-9255
dmrizzo@ucdavis.edu

Donald Robinson
last attended 2002
ESSA Technologies Ltd.
1765 West 8th Avenue, Suite 300
Vancouver BC V6J 5C6 CANADA
604-733-2996
drobinson@essa.com

Gary Roke
last attended 2002
Canadian Forest Service, Pacific
Forestry Centre, 506 W. Burnside Rd.
Victoria BC V8Z 1M5 CANADA
250-363-3868
groke@pfc.forestry.ca

David Rusch
last attended 2002
Rot Rooters Forest Consulting
302 Dogwood Street
Parksville BC CANADA
250-951-0305
rusch@bcsupernet.com

Dave Russell
last attended 2007
USDI, Bureau of Land Management
14171 Williams Hwy
Grants Pass OR 97527 USA
(541) 846-7296
Dave_Russell@or.blm.gov

Daniel Ryerson
last attended 2007
USDA, Forest Service
333 Broadway Boulevard SE
Albuquerque NM 87102 USA
505-842-3285
dryerson@fs.fed.us

Angel Saavedra
last attended 2006
Wenatchee Forest Sciences Lab
1133 N. Western Avenue
Wenatchee WA 98801 USA
(509) 664-9223
alsaavedra@fs.fed.us

Shiroma Sathyapala
last attended 2004
Forest Biosecurity
101-103 The Terrace
P.O. Box 2526
Wellington New Zealand
sathyapalas@maf.govt.nz

Craig Schmitt
last attended 2005
USDA, Pacific Northwest Reserch
Station, 1401 Gekeler Lane
LaGrande OR 97850 USA
541-962-6544
clschmitt@fs.fed.us

Scott Scholefield
last attended 2006
University of Northern British Columbia
18115 Walnut Road
Prince George BC V2K 5V5 CANADA
(250) 971-2472
scott.scholefield@gmail.com

Bob Schroeter
last attended 2003
USDA, Forest Service
J. Herbert Stone Nursery
2606 Old Stage Road
Central Point OR 97502 USA
(541) 858-6123
rschroeter@fs.fed.us

John Schwandt
last attended 2007
USDA, Forest Service
Idaho Panhandle National Forests
3815 Schreiber Way
Coeur d'Alene ID 83815 USA
(208) 765-7415
jschwandt@fs.fed.us

Simon Shamoun
last attended 2002
Canadian Forest Service
Pacific Forestry Centre
506 W. Burnside Rd.
Victoria BC V8Z lM5 CANADA
250-363-0766
sshamoun@pfc.forestry.ca

John Shaw
last attended 2004
USDA, Forest Service
Rocky Mountain Research Station, FIA
507 25th Street
Ogden UT 84401
jdshaw@fs.fed.us

David Shaw
last attended 2007
Oregon State University
Dept. Forest Science
321 Richardson Hall
Corvallis OR 97331 USA
541-737-2845
dave.shaw@oregonstate.edu

Charles "Terry" Shaw
last attended 2007
USDA, Forest Service
Pacific Northwest Research Station,
WWETAC, 3160 NE 3rd Street
Prineville OR 97754 USA
(541) 416-6600
cgshaw@fs.fed.us

Aaron Smith
last attended 2007
USDA, Forest Service
Deschutes National Forest
1001 SW Emkay Dr.
Bend OR 97702 USA
541-383-5771
alsmith@fs.fed.us

Thomas Smith
last attended 2007
California Dept. of Forestry & Fire
Protection, 1416 9th Street
Sacramento CA 95814 USA
916-599-6882
tom.smith@fire.ca.gov

Eric L. Smith
last attended 2005
USDA, Forest Service
2150 Centre Ave., Bldg A, Suite 331
Fort Collins CO 80526-8121 USA
(970)-295-5841
elsmith@fs.fed.us

Richard Sniezko
last attended 2007
USDA, Forest Service
Dorena Genetic Resource Center
34963 Shoreview Rd.
Cottage Grove OR 97424 USA
(541) 767-5716
rsniezko@fs.fed.us

Sharon Stanton
last attended 2003
Portland State University
Dept. of Biology
PO Box 751
Portland OR 97207 USA
(503) 281-3582
sstanton@pdx.edu

Jeff Stone
last attended 2006
Oregon State University
Dept. of Botany and Plant Pathology
2082 Cordley Hall
Corvallis OR 97331-2902 USA
(541) 737-5260
stonej@science.oregonstate.edu

Rona Sturrock
last attended 2007
Canadian Forest Service
Pacific Forestry Centre
506 W. Burnside Rd.
Victoria BC V8Z 1M5 CANADA
(250) 363-0789
rsturrock@pfc.forestry.ca

Wendy Sutton
last attended 2002
Oregon State University
Dept. of Botany and Plant Pathology
2082 Cordley Hall
Corvallis OR USA
541-737-5242
suttonw@bcc.orst.edu

Dave Thomas
last attended 2004
USDA, Forest Health Protection
1601 N. Kent St.
Arlington VA 22209 USA
(703) 605-5342
dthomas06@fs.fed.us

Alan Thomson
last attended 2002
Canadian Forest Service
Pacific Forestry Centre
506 West Burnside Road
Victoria BC CANADA
250-363-0632
athomson@pfc.forestry.ca

Borys Tkacz
last attended 2005
USDA, Forest Health Protection
1601 N. Kent St. RPC7- FHP
Arlington VA 22209 USA
(703) 605-5343
btkacz@fs.fed.us

Loreen Trummer
last attended 2007
USDA, Forest Service
3301 C St., Suite 202
Anchorage AK 99503-3956 USA
907-743-9460
ltrummer@fs.fed.us

Detlev R. Vogler
last attended 2007
USDA, Forest Service
Institute of Forest Genetics
2480 Carson Road
Placerville CA 95667-5107 USA
(530) 621-6881
dvogler@fs.fed.us

Jim Walla
last attended 2005
North Dakota State University
Plant Pathology Department
Fargo ND 58105 USA
701-231-7069
j.walla@ndsu.edu

Dave Weaver
last attended 2006
BC Ministry of Forests
Skeena-Stikine Forest District, Bag 6000
Smithers BC V0J 2N0 Canada
250 847 6334
Dave.Weaver@gov.bc.ca

Joan Webber
last attended 2003
Forest Research Agency
Alice Holt Lodge
Farnham Surrey GU10 4LH UK
44 1420 526241
joan.webber@forestry.gsi.gov.uk

Cedar Welsh
last attended 2006
University of Northern British
Columbia, 3333 University Way
Prince George BC V2N 4Z9 CANADA,
(250) 960-6807
welsh@unbc.ca

Ken White
last attended 2006
BC Ministry of Forests
Northern Interior Forest Region
Bag 6000, 3333 Tatlow Road
Smithers BC V0J 2N0 CANADA
(250) 847-6383
Ken.J.White@gov.ca

Teresa White
last attended 2006
Cedrus Consulting
P.O. Box 579
Smithers BC V0J 2N0 CANADA
250 847 2289
cedrus@telus.net

Alan Wiensczyk
last attended 2006
FORREX, 400 1488 4th Avenue
Prince George BC V2L 4Y2 CANADA,
(250) 614-4354
alan.wiensczyk@forrex.org

Beth Willhite
last attended 2003
USDA, Forest Service
Mt. Hood National Forest
16400 Champion Way
Sandy OR 97044 USA
(503) 668-1477
bwillhite@fs.fed.us

Lori Winton
last attended 2001
Oregon State University
Dept. of Botany and Plant Pathology,
Cordley 2082
Corvallis OR 97331 USA
541-737-5242
wintonl@bcc.orst.edu

William Woodruff
last attended 2007
USDA, Forest Service
NE CA Shared Service Area
2550 Riverside Dr.
Susanville CA 96130 USA
(530) 252-6680
wwoodruff@fs.fed.us

Alex Woods
last attended 2007
BC Ministry of Forests and Range
Northern Interior Forest Region
Bag 6000, 3333 Tatlow Road
Smithers BC V0J 2N0 CANADA
(250) 847-6382
alex.woods@gov.bc.ca

Jim Worrall
last attended 2007
USDA, Forest Service
Gunnison Service Center
216 N Colorado St.
Gunnison CO 81230 USA
(970) 642-1166
jworrall@fs.fed.us

Arezoo Zamani
last attended 2002
Canadian Forest Service
Pacific Forestry Centre
506 W. Burnside Rd.
Victoria BC V8Z 1M5 CANADA
250-363-0619
azamani@pfc.forestry.ca

Paul Zambino
last attended 2003
USDA, Forest Service
San Bernardo National Forest
1824 South Commercenter Circle
San Bernadino CA 92408 USA
(909) 382-2727
pzambino@fs.fed.us

Stefan Zeglen
last attended 2007
BC Ministry of Forests and Range
Coast Forest Region,
2100 Labieux Road
Nanaimo BC V9T 6E9 CANADA
(250) 751-7108
stefan.zeglen@gov.bc.ca

RETIRED MEMBERS

Norm Alexander
last attended 1996
5972 Glendale Drive
Chilliwack BC V2R 3A5 CANADA
(604) 824-2156
normalex@telus.net

Ed Andrews
last attended
Richard T. "Dick" Bingham
last attended 1975
1127 American Ridge Dr.
Kendrick ID 83537-9504 USA

Clive Brasier
last attended 2003
Forest Research Agency
Alice Holt Lodge
Farnham Surrey GU10 4LH UK
44 1420 526240
clive.brasier@forestry.gsi.gov.uk

James Byler
last attended 2005
1523 E. Woodland Dr.
Dalton Gardens ID 83815 USA
208-772-7442
jjbyler@aol.com

Fields Cobb
last attended 1996
4492 Lakeshore Dr.
Sagle ID 83860 USA
208-265-1513
fieldscobb@hotmail.com

Charles H. Driver
last attended 1996
2019 Edith Ave
Enumclaw WA 98022
(360) 802-3083

David Etheridge
last attended 1975
3941 Oakdale Place
Victoria BC V8N 3B6 CANADA
250-477-5726

Lowell Farmer
last attended 1965

Mike Finnis
last attended 1984
1888 Gonzales
Victoria BC V8Z 1M5 CANADA

Alvin Funk
last attended 1989
6819 Jedora Drive
Brentwood Bay BC V0S 1A0 CANADA

Robert Gilbertson
last attended 2007
University of Arizona
4321 Vereda Rosada
Tucson AZ 85750 USA
520-539-4340
gilbertson4340@msn.com

Linnea Gillman
last attended 1980
3024 S. Winona Ct.
Denver CO 80236 USA

James Ginns
last attended 1996
1970 Sutherland Road
Penticton BC V2A 8T8 CANADA
250-492-9610

Donald Graham
last attended 2003
5702 NE 88th Court
Vancouver WA 98662 USA
206-892-8811
dongram@pacifier.com

John Hart
last attended 2005
Hartwood Natural Resource Consultants
1390 Curt Gowdy Dr.
Cheyenne WY 82009 USA
307-778-3993
huntwyoming@aol.com

Alan Harvey
last attended 1998

Ray Hoff
last attended 1995
907 East 7th Street
Moscow ID 83843 USA

John Hopkins
last attended

Richard Hunt
last attended 2002
Canadian Forest Service
Pacific Forestry Centre
506 West Burnside Rd.
Victoria BC V8Z 1M5 CANADA
250-363-0640
rhunt@pfc.forestry.ca

David Johnson
last attended 2001
12851 W. Asbury Place
Lakewood CO 80228 USA

John Kliejunas
last attended 2007
5305 Lightwood Drive
Concord CA 94521 USA
925.682.4825
kliejunas@comcast.net

Leon Lamadeleine
last attended 1993
Thomas Laurent
last attended 2004
P.O. Box 240130
Douglas AK 99824-0130 USA
907-364-2435

John Laut
last attended 1999
4700 E. Main St.
Space 382
Mesa AZ 85205 USA
480-981-6957

Paul Lightle
last attended

Otis Maloy
last attended 1991
1036 Wallen Rd.
Moscow ID 83843 USA
omaloy@moscow.com

Neil Martin
last attended 1989
514 South Howard
Moscow ID 83843 USA
jandnmart@moscow.com

Arthur McCain
last attended 1998
1 Hilldale Rd.
Lafayette CA 94549-2803 USA
925-284-9632
mccain@nature.berkeley.edu

Tim McConnell
last attended 2004

Geral McDonald
last attended 2004
553 Old Moscow Rd.
Pullman WA 99163 USA
208-883-2343
gimcdonald@fs.fed.us

Tom McGrath
last attended 2007
216 N. Mound St.
Nacogdoches TX 75961 USA
smokeytom@suddenlink.net

Hadrian Merler
last attended 2004
 VIT 8Z1 CANADA

Alex Molnar
last attended 2002
2085 St Andrews Way
Courteney BC VGN NV5 CANADA
250-334-0365

Duncan Morrison
last attended 2005
1487 Stellys Cross Road
Saanichton BC V8M 1S8 CANADA
250-652-3281
armillaria@shaw.ca

John Muir
last attended 2003
2031 Casa Marcia Crescent
Victoria BC V8N 2X5 CANADA
(250) 477-1805
johnmuir@consultant.com

Earl Nelson
last attended 1993
2175 Condor Dr.
Redmond OR 97756 USA
541-504-0685
bigearl35@aol.com

Tom Nicholls
last attended 2005
Nature Education Cntr, W7283 Walnut
St., P.O Box 63
Fifield WI 54524-0063 USA
715-762-3076
nicho002@umn.edu

Vidar Nordin
last attended 1997
P.O. Box 2368, Station D
340 Laurier Ave. West
Ottawa ON K1P 5W5 CANADA
613-234-7478
vidar.nordin@sympatico.ca

Don Norris
last attended 1995
643 Rossland Ave.
Trail BC V1R 3N2 CANADA
(25) 368-6647

John "Dick" Parmeter
last attended 2002
04837 Oceana Drive
Florence OR 97439 USA
541-997-1692
jrpakp@presys.com

Art "Doc" Partridge
last attended

Roger Peterson
last attended 1999
1750 Camino Corrales
Santa Fe NM 87505 USA
505-983-7559
rogpete@aol.com

Glenn W. Peterson
last attended
3817 Dudley
Lincoln NE 68503 USA
402-464-3696

John Pronos
last attended 2005
jpronos@hughes.net

Jerry Riffle
last attended 1986
6086 E. George St.
Syracuse IN 46567 USA
jerry.riffle@kconline.com

Jack Roff
last attended

Lewis Roth
last attended 2003
4798 Becker Circle SE
Albany OR 97321 USA
541-926-6068

Kenelm Russell
last attended 2002
Forest and Health Tree Services
8143 Evergreen Dr. NE
Olympia WA 98506 USA
360-943-8199
fishtrap1@aol.com

Robert Scharpf
last attended 2007
Quartz Hill Vineyard
8548 Mosquito Road
Placerville CA 95667 USA
(530) 622-8315
qtzhill@d-web.com

Mike Schomaker
last attended 2005
5400 Vardon Way
Fort Collins CO 80528 USA
(970) 223-1929
Michael.Schomaker@colostate.e

E. Mike Sharon
last attended

Pritam Singh
last attended 1997
1135 St. Jovite Ridge
Orleans ON K1C 1Y6 CANADA

Richard B. Smith
last attended 2002
7797 16 th St.
P.O. Box 622
Grand Forks BC V0H 1H0 CANADA
250-442-2419

Richard S. Smith
last attended
643 Amberwood Way
Livermore CA 94550 USA

Michael Srago
last attended 2005
7006 Potrero Ave.
El Cerrito CA 94530 USA
510-232-7092
msrago@comcast.net

James L. Stewart
last attended
3028 Covington St.
Fairfax VA 22031-2011 USA

Jack Sutherland
last attended 1996
1963 St. Ann Street
Victoria BC V8R 5V9 CANADA
250-598-4033
jsuther@islandnet.com

Al Tegethoff
last attended 1996
11750 E. Sneller Vista Dr.
Tucson AZ 85749 USA

Walter Thies
last attended 2007
USDA, Forest Service
Pacific Northwest Research Station
3317 NW Firwood Drive
Corvallis OR 97330 USA
(541) 752-5214
wgthies@msn.com

Robert Tinnin
last attended 2003
8876 SW Edgewood St.
Tagard OR 97223 USA
(530) 620-2470
bob.tinnin@verizon.net

Jim Trappe
last attended

Eugene P. Van Arsdel
last attended 2007
62 Lagarto
Tijeras NM 87059 USA
(505) 286-4116
epvan@highfiber.com

Bart van der Kamp
last attended 2007
University of British Columbia
Faculty of Forestry, Univ. of BC
3042-2424 Main Mall
Vancouver BC V6T IZ4 CANADA
(604) 822-2728
vdkamp@interchg.ubc.ca

Allen Van Sickle
last attended 1997
4436 Rangemont Place
Victoria BC V8N 5L6 CANADA
vansickl@islandnet.com

Gordon Wallis
last attended 1983
4720 Spring Rd., RR #3
Victoria BC V8X 3X1 CANADA

Conrad Wessela
last attended

Roy Whitney
last attended 1990
47 Cumberland Dr. NW
Calgary AB T2K 1S8 CANADA

Stuart Whitney
last attended 1991
5033 Ayum Road
Sooke BC V0S 1N0 CANADA

Ed Wicker
last attended 1991
1240 Thatuna Ave.
Moscow ID 83843 USA

Ralph Williams
last attended 1994
9650 S. Powerline Rd
Nampa ID 83686-9408 USA

Ed Wood
last attended

Bratislav Zak
last attended

DECEASED MEMBERS

Stuart "Stuie" Andrews	Oscar Dooling	Tommy Hinds	Lee Paine
Jesse Bedwell	Norm Engelhart	Brenton Howard	Clarence Quick
Robert Bega	Ray Foster	John Hunt	Dave Schultz
Warren Benedict	Dave French	Paul Keener	Charles G. Shaw
John Bier	Lake S. Gill	James Kimmey	Albert Slipp
Bill Bloomberg	Clarence "Clancy" Gordon	Don Leaphart	Willhelm Solheim
Roy Bloomstrom	John Gynn	Neil McGregor	Phil Thomas
Thomas "Buck" Buchanan	John Hansbrough	Jim Mielke	Willis Wagener
Don Buckland	Hans Hansen	D. Reed Miller	Charles "Doc" Waters
Hubert "Hart" Bynum	Homer Hartman	Vergil Moss	Larry Weir
Elmer Canfield	George Harvey	Harold Offord	John Woo
Toby Childs	Frank G. Hawksworth	Nagy Oshima	Ernest Wright
Ross Davidson	Dwight Hester	John Palmer	Wolf Ziller

Back Row: Bart Van der Kamp, Terry Shaw, Aaron Smith, Bob James, Jerry Beatty
Front Row: Walt Thies, Jim Hoffman, Alex Woods, Jim Blodgett, Tom McGrath

Back Row: Greg Filip, Michael McWilliams, Paul Hennon, Dave Russell, Gary Chestagner, Roy Mask
Front Row: Richard Sniezko, Don Goheen, Fred Baker, John Kleijunas, Bill Woodruff, Lori Trummer

Left to Right: Bryce Richardson, Ned Klopfenstien, Amy Gannon, MaryLou Fairweather, Andi Koonce, Amy Ramsey, Katy Mallams, Bill Jacobi, Mee-Sook Kim, Brian Howell, Holly Kearns, Martin MacKenzie

Left to Right: Gregg DeNitto, Brian Geils, Will Littke, Patricia Maloney, Det Vogler, Annette Delfino-Mix, Hideji Ono, Crystal Braun, Ted Hogg, Susan Frankel, John Browning, Stefan Zeglen, Sally Campbell

Back Row: Danny Norland, Brennan Ferguson, Ellen Goheen, Mike McWilliams, Blakey Lockman, Aaron Smith, Jennifer Klutsch, Dan Omdal, Betsy Goodrich, Phil Cannon, Marcus Jackson
Front Row: Danni Martin, Dave Shaw, Kristin Chadwick

Left to Right: Harry Kope, Richard Reich, Gene Van Arsdel, Marla Downing, Michelle Cleary, Lance David, John Schwandt

Back Row: Bob Gilbertson, Pete Angwin, Bob Mathiasen, Rona Sturrock, Jim Worrall, Dreena Lindstrom, Judy Adams, John Guyon, Bob Scharpf, Margo Scharpf
Front Row: Kelly Burns, Helen Maffei, Gail Thies, Tom DeGomez

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