

Proceedings of the 63rd Annual Western International Forest Disease Work Conference

**September 21-25, 2015
Newport, Oregon**



***Proceedings of the 63rd Annual Western
International Forest Disease Work Conference***

*Best Western Agate Beach Inn
Newport, Oregon, U.S.
September 21-25, 2015*

Compiled by:

Amy Ramsey
Washington Department of Natural Resources
Olympia, Washington

and

Patsy Palacios
S.J. and Jessie E. Quinney Natural Resource Research Library
Quinney College of Natural Resources
Utah State University, Logan, Utah

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Papers are formatted and have minor editing for language, and style, but otherwise are printed as they were submitted. The authors are responsible for content.

Special Thanks for Photos go to:

Kristen Chadwick

Christy Cleaver

David Hunter

Alan Kanaskie

Willis Littke

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AGENDA

Monday, September 21

- 3:00 - 5:00 pm **Registration**
- 3:30 - 5:00 pm **Nursery Committee Meeting**
Anna Leon, Committee Chair
- 5:00 - 7:00 pm **Welcome Social**
-

Tuesday, September 22

- 7:00 - 8:30 am **Registration**
- 7:00 - 8:30 am **Rust Committee Meeting**
Helen Maffei, Committee Chair
- 8:30 - 9:00 am **Welcome and Introductions 2015 WIFDWC Chair**
Alan Kanaskie, Forest Pathologist, Oregon Department of Forestry, Salem
- 9:00 - 9:30 am **Keynote Address: Ecology of the Oregon Coast Range**
Steve Acker, Zone Ecologist, USDA Forest Service, Pacific Northwest Region, Willamette National Forest, Eugene, Oregon
- 9:30 - 9:40 am **2015 Outstanding Achievement Award Presentation**
Kathy Lewis: 2015 Committee Chair
- 9:40 - 10:00 am **Outstanding Achievement Award Address**
Terry Shaw, 2014 Award Recipient
- 10:00 - 10:30 am Break
- 10:30 - 11:40 am **Graduate Student Flash and Dash**
Betsy Goodrich: Moderator
- Zolton Bair, Oregon State University*
Using transcriptomics to identify candidate genes associated with blister rust resistance in whitebark pine
- Patrick Bennett, Oregon State University*
Genetic diversity and population structure of *Phaeocryptopus gaeumannii* in the Pacific Northwest
- Christina Benemann, Oregon State University*
Determining the relative amount of soil borne inoculum of *Phytophthora ramorum* within an Oregon tanoak forest
- Yung-Hsiang Lan (Sky), Oregon State University*
Relationship between canopy structure, microclimate, and Swiss needle cast severity among different ages of Douglas-fir forests
- Nicholas Wilhelmi, Oregon State University*
The effect of seed source and planting environment on Douglas-fir foliage diseases
- Saeideh Jafarpoor, University of Tehran, Karaj, Iran (w/ University of Idaho)*
Armillaria surveys in Iran

Megan Dudley, Colorado State University
An initial phylogeny of cytopora species on quaking aspen

Kelsey Dannel, Oregon State University
Understanding the *Populus: Sphaerulina musiva* pathosystem

Brandon Alveshire, Oregon State University
Distribution and ecology of *Armillaria* species in riparian areas of the Northern Great Plains

Natália Risso Fonseca, Universidade Federal de Viçosa, Brazil
Ongoing molecular studies of eucalyptus powdery mildew in Brazil

Dixie Daniels, Oregon State University
Phylogenetic identification of entomopathogenic and endophytic fungal populations in west coast Douglas-fir foliage identified as *Phaeocryptopus gaemannii* infected

11:40 -12:00 am **Outstanding Achievement Award Address**
Will Littke, 2014 Award Recipient

12:00 -1:30 pm Lunch

12:00 -1:30 pm **Root Disease Committee Meeting**
Blakey Lockman, Committee Chair

1:30 - 2:00 pm **Meet the Faculty**
Alan Kanaskie: Moderator
Jared LeBoldus, Oregon State University
Jane Stewart, Colorado State University
Jim Kiser, Oregon State University

2:00 - 5:00 pm **Drought and Other Factors as Contributors to Tree Mortality**

Part 1: Behavior at the Tree Level

Susan Frankel: Moderator

Tree response to water and heat stress

Frederick (Rick) Meinzer, Senior Research Ecologist, USDA Forest Service, Pacific Northwest Research Station, Corvallis Forestry Sciences Laboratory; Corvallis, Oregon

Structural mechanisms trees use to deal with water stress

Barbara Lachenbruch, Professor, Ecophysiology, Department of Forest Ecosystems and Society, Oregon State University, Corvallis, Oregon

3:00 - 3:30 pm Break

3:30 - 5:00 pm **Drought and Other Factors as Contributors to Tree Mortality**

Part 2. Disease and Drought Interactions

Alex Woods: Moderator

Tree drought stress and insect and pathogen incidence in western yellow pine

Nancy Grulke, Center Director, USDA Forest Service, Western Wildland Environmental Threat Assessment Center, Prineville, Oregon

Annual trends of Armillaria root disease, mortality, and climatic factors in a tree plantation of southeast British Columbia

Michael Murray, Forest Pathologist, BC Ministry of Forests, Lands and Natural Resource Operations, Nelson, BC, Canada

A forest pathology for global-change associated tree mortality

Richard Cobb, Researcher, UC Davis, Department of Plant Pathology, Davis, California

Discussion

5:00 pm

Adjourn

7:00 -9:00 pm

Poster Session / Silent Auction / Photo Contest / Ice Cream Social

Josh Bronson, Brent Oblinger, and Ellen Goheen: Moderators

Wednesday. September 23

8:00 am - 6:30 pm

Field Trip

Forty Years of Forest Pathology in Northwest Oregon

Dave Shaw, Alan Kanaskie, Greg Filip, Amy Ramsey, and Everett Hansen

6:30 - 8:00 pm

Climate Change Committee Meeting

Alex Woods, Committee CoChair

Thursday. September 24

7:00 -8:30 am

Foliage and Twig Disease Committee Meeting

Stefan Zeglan, Acting Committee Chair

8:30 -10:00 am

Emerging Pathogens: Conifer Phytophthoras

Ellen Michaels Goheen: Moderator

Phytophthora pluvialis causing red needle cast of *Pinus radiata* in New Zealand

Nari Williams, Forest Pathologist, Scion Research, Rotorua, New Zealand

Phytophthoras on conifers in the UK - a paradigm shift?

Joan Webber, Forest Pathologist, Forest Research, United Kingdom

Challenges for western North America

Everett Hansen, Emeritus Professor, Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon, USA

Discussion

10:00 -10:30 am

Break

10:30 -12:00 am

Needle Pathogen Outbreaks: Why There? Why Then?

Gabriela Ritokova: Moderator

Dothistroma needle blight, weather and possible climatic triggers behind the disease's recent emergence

Alex Woods, Forest Pathologist, Forest Pathologist, BC Ministry of Forests, Lands and Natural Resource Operations, Smithers, BC, Canada

Regional climate models for predicting the distribution and severity of Swiss needle cast

Jeff Stone, Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon

Tree-ring history of Swiss needle cast impact on Douglas-fir growth in western Oregon: Correlations with climatic variables
Henry Lee, US Environmental Protection Agency, National Health and Environmental Effects Research Laboratory, Western Ecology Division, Corvallis, Oregon, USA

Discussion

12:00 -1:30 pm Lunch

12:00 -1:30 pm **Hazard Tree Committee Meeting**
Alan Kanaskie, Acting Committee Chair

1:30 - 5:00 pm **Field Trip**
Topless Conifers: Coastal Forest Ecology on the Hemlock Annosus Trail
Dave Shaw

6:00 pm **Dinner with the Sharks:** Banquet at the Oregon Coast Aquarium
Bonfire on the Beach

Friday September 25

7:00 -8:30 am **Dwarf Mistletoe Committee Meeting**
Dave Shaw, Committee Chair

8:30 -10:30 am **Special papers**
Ellen Michaels Goheen: Moderator

Chalara in the UK, three years down the line
Joan Webber, Forest Pathologist, Forest Research, United Kingdom

Identifying fungi associated with the walnut twig beetle, *Pityophthorus juglandis*, in black walnut
Dixie Ann Daniels, Graduate Student, Oregon State University, Corvallis, Oregon

Limber Pine Conservation Strategy for Rocky Mountain National Park
Christy Cleaver, Forest Pathologist, USDA Forest Service Northern Region, Forest Health Protection, Coeur d'Alene, Idaho

Dothistroma needle disease in the Prairie Provinces, understanding the risk
Tod Ramsfield, Research Scientist Forest Pathology, Canadian Forest Service, Northern Forestry Centre, Edmonton, Alberta, Canada

Temporal epidemiology of sudden oak death in SW Oregon: prioritizing our monitoring efforts to manage EU1 in Curry County
Ebba Peterson, Post Doctoral Research Associate, Oregon State University, Corvallis, Oregon

Determining the efficacy of steam as a mitigation for *Phytophthora* species in soil
Sarah Navarro, Plant Disease Program Specialist, Oregon Department of Agriculture, Salem, Oregon

10:30 -11:30 am **Business Meeting**
Alan Kanaskie, 2015 WIFDWC Chair.

11:30 am **Close and Adjourn**
Alan Kanaskie, 2015 WIFDWC Chair

2015 OUTSTANDING ACHIEVEMENT AWARD: BRIAN GEILS

The Outstanding Achievement Award (OAA) Committee: *Mike Cruickshank, Ellen Goheen, and Kathy Lewis*

We are very pleased to announce that the recipient of the WIFDWC Outstanding Achievement Award for 2015 is Brian Geils.

Without exception, the nomination letter and letters in support of the nomination described a creative scientist with a broad range of interests, a high level of enthusiasm and curiosity, and a great guy to be with in the field.

Brian started his career with an undergraduate degree in wildlife biology, but after working in forest pest management in New Mexico, Brian saw the light and went on to a masters degree in forest science, where he and Art Partridge became the dynamite duo – blasting stumps out of the ground to study root disease (oh the good old days...). Brian tried consulting for a while, but nothing could compare to blowing up stumps, so he went on to a PhD at Colorado State working with Bill Jacobi on blister rust in the central Rocky Mountains. In 1984, he started work with the Rocky Mountain Research Station in Fort Collins, which was the beginning of a highly productive collaboration with the renowned mistletoe expert, Frank Hawksworth.

Everyone who supported Brian's nomination mentioned his work on the 1996 Handbook on the Biology, Pathology and Systematics of Dwarf Mistletoes by Hawksworth and Wiens. Without Brian's exceptionally diligent work on preparation of tables and figures, references, appendices, proofreading and many other tasks associated with publishing a book, the book would never have seen the light of day. Brian also led development of a book on Mistletoes of North American Conifers, with colleagues from Mexico and Canada.

Apparently Brian applied some of his creative genius in "transactions" necessary to get cartons of the books past Mexican customs agents.

Rust diseases of forest trees was another area where Brian had significant impact, including over 45 publications on tree rusts, and co-editing of the 2010 Special Issue of the Journal of Forest Pathology dedicated to White Pines, Ribes and Blister Rust.

Several nominators spoke of Brian's contributions "behind the scenes" which demonstrate that Brian's focus was on having impact and making important contributions to our knowledge of tree pathogens, and not on self-promotion. He served as curator of the Forest Service's mistletoe herbarium and the online mistletoe literature database of over 13,000 records. He was also "Scientist in Charge" of the Fort Valley Experimental Forest, and technical editor on the Assessment of Forest Ecosystem Health in the Southwest. All of these are examples of often thankless tasks that are of importance to the scientific community, but bring little recognition to those doing them.

Words used by his nominators to describe Brian include: "attention to detail", "willingness to share", "unforgettable", "curious", and "delightful friend". The OAA committee is privileged to have had the opportunity to read the letters of support for Brian's nomination, and was in unanimous agreement with awarding him the 2015 Outstanding Achievement Award.

We would like to thank each of you who wrote a letter of support for the nomination of a forest pathologist for this award.



2014 OUTSTANDING ACHIEVEMENT AWARD: RECIPIENT WILLIS LITTKE - THESE ROOTS RUN DEEP: REFLECTIONS ON 37 YEARS WITH WIFDWC

I am honored to receive the WIFDWC outstanding achievement award and especially honored to share this moment with Terry Shaw. WIFDWC is a unique organization bringing all the best qualities and experiences in forest pathology and entomology, forest management and nursery science to the forefront. The strength of this organization is our personal interactions and mentoring opportunities as we meet or interact professionally. The uniqueness in this organization is founded in the pathways which have brought us together.

For many, a direct pathway can be charted from classical forest pathology curriculum to a current employment situation. However, my background like others passed first through classical mycological taxonomy and thence to forestry. One now feels dated as many hours of tedious nights in dark hallways of Johnson Hall (UW) with a microscope lacto-phenol and Meltzer's reagent as companions, have now been largely replaced by PCR equipment and whirling centrifuges.

My family's involvement in the PNW dates to the late 1840's with great-great grandfather being a mountain man and granny an interpreter for Ft. Vancouver. Subsequent generations also worked in the woods. My interest in forestry began as a Boy Scout earning my first merit badge in forestry. Our scoutmaster was one of the last back-country rangers in the Forest Service. His tales of "high adventure" traveling alone by horse all summer checking on fire conditions in the North Cascades sealed the deal for me. As break from college I joined the Mt. Baker-Snoqualmie ranger district in 1967 as member of a fast-attack fire crew. Years later sitting in a fire ecology class I had to laugh about descriptions of what a crown fire was like, having survived several encounters myself.

Along the way in post-graduate school, there were professors of note; Bob Edmonds, Charlie Driver,

Dan Stuntz, Joe Ammirati and others. The comradery of graduate students (many still active in WIFDWC or retired) all working on aspects of forest pathology was a great learning and teaching experience.

Shortly after entering the UW I was attended my first WIFDWC (Victoria BC), and was in awe of the breadth of forest health experience represented in the room. I was also struck on the openness of the members to share thoughts and discussions on topics at hand. This is the "hidden secret" of WIFDWC—the one on one interactions with fellow professionals. My message to younger people is to take advantage of the opportunity to engage other members during the meeting.

During the last 35 years WIFDWC has evolved to its present state. One great reminder of this change is embodied in a late WIFDWC proceeding touting a graph of "Ties and Skirts" over time. So too, the continued representation of Forest Service, Ministry of Forests, Canada Forests and State and Private forestry personnel—and yes a small but vocal contingent of industry forest health professionals.

What will the future bring for WIFDWC? That outcome will be decided by you attending this and future meetings. It will be encumbered on those of us who are retired but still attend and contribute to discussions and paper reviews. It will come down to learning and teaching new skills to unlock complex disease interactions. I will predict the "disease triangle" will be replaced with something that resembles more the "cats-cradle" string game we played as kids. Resolve to begin today on making new contacts, mentoring a new attendee, or talking with retirees. There is an old adage: Some watch it happen; some let it happen; and some make it happen—which will you do?



MEET THE FACULTY

Jim Kiser, Instructor, Oregon State University

Department of Forest Engineering, Resources and Management, Corvallis, Oregon

Education:

Ph.D. Forest Science, 2009. Oregon State University.

M.S. Forest Management (Integrated Minor in Statistics), 1992. Oregon State University.

B.S. Forest Management, 1982. Humboldt State University.

Areas of Interest:

- Ecology of Fungi and Forest Trees, Molecular Genetics of Forest Fungi, Physiological Responses of Forest Trees to Fungi.
- Surveying Technology in Forest Environments and Forest Harvesting, Technological Efficiencies for Forest Operations.
- Statistical Processes in Forest Biometrics, Efficiencies in Timber Cruising Design and Sampling. Biometric Responses of

Forest Trees to Damage, Modeling tree Growth Responses to Damage.



Photo from www.forestry.oregonstate.edu.

Jared M. LeBoldus, Assistant Professor in Forest Pathology, Oregon State University

Department of Botany and Plant Pathology and Department of Forest Engineering, Resources and Management, Corvallis, Oregon

Dr. Jared M. LeBoldus joined the department of Botany and Plant Pathology and the Forest Engineering and Resources Management in the fall of 2015. His research interests lie in the area of forest pathology. His basic research explores the interactions between host and pathogen populations in natural ecosystems and his applied research is focused on developing management strategies for a variety of forest diseases. Dr. LeBoldus received a B.Sc. (2003) in Forest Science from the University of British Columbia. He received his M.Sc. (2006) and Ph.D. (2010) from the University of Alberta in the area of Forest Biology and Management.



Jane E. Stewart, Assistant Professor in Plant Pathology, Colorado State University

Department of Bioagricultural Sciences and Pest Management, Ft. Collins, Colorado

Jane joined BSPM at Colorado State University in 2015. Jane, originally from Oregon, received her BS in biology from the University of Oregon. After a summer of fieldwork in Oregon coast forests with Oregon State University, she went to the east coast and received a master's degree in Forest Pathology from the University of Vermont with Dale Bergdahl. Her work was focused on insect vectoring potential of the butternut canker pathogen. She was then traveled back to the west and was a biological science technician with Mee-Sook Kim and Ned Klopfenstein at the USDA FS-RMRS in Moscow, Idaho. Her work was focused on molecular diagnostics and management of *Armillaria* and *Fusarium* pathogens. Jane then started a Ph.D. in Plant Pathology with Tobin Peever from Washington State University. Her work was focused on the fungal genetics, genomics, and species concepts of a citrus pathogen, *Alternaria alternata*. After graduating, Jane, traveled back to Corvallis, Oregon and worked with Niklaus Grünwald, as a postdoctoral scholar, at the USDA ARS Horticultural Research Laboratory on *Phytophthora* species. She then began postdoctoral researcher position at the University of Georgia, where she examined mechanisms that drive emerging pathogens with Marin Talbot Brewer. Her work focused on a blueberry pathogen, *Exobasidium maculosum*. Jane has

published over 20 research articles in forest pathology, fungal genetics and genomics and fungal biology. Her research program at CSU is focused on tree pathogens in Colorado and worldwide to better understand the biology, ecology and genetics, and management of emerging tree/plant pathogenic fungi.



STUDENT PAPERS



DISTRIBUTION AND ECOLOGY OF ARMILLARIA SPECIES IN RIPARIAN AREAS OF THE NORTHERN GREAT PLAINS

Brandon Alveshere¹, James T. Blodgett², Jeri Lyn Harris³, and Jared M. LeBoldus^{1,4}

ABSTRACT

This study aims to collect baseline data on species of Armillaria present in the Northern Great Plains region. In addition to identifying the species of Armillaria present in riparian forests of the region, the researchers intend to map species distributions, determine host range relationships, and evaluate pathogenicity among the different species. This involves the temporary establishment of Armillaria survey sites along forested riparian areas in North Dakota, South Dakota, and Nebraska. Samples of Armillaria are collected at each survey site where Armillaria is found, cultured in the lab, and identified using molecular techniques. Measured parameters and collection methods for each survey site are based on those of Blodgett and Worrall (1992a, 1992b). In the first field season of this study (2015), Armillaria was found at 42 of 47 sites surveyed. Armillaria was associated with root rot at 23 sites and butt rot at 4 sites; it was found only in a saprophytic and/or epiphytic capacity at all other sites where it was recorded. Root rot was observed on 9 hardwood tree species, which included *Populus deltoides*, *Fraxinus pennsylvanica*, *Quercus macrocarpa*, *Acer negundo*, *Ulmus americana*, *Acer saccharinum*, *Salix*, *Morus alba*, and *Gleditsia triacanthos*. Butt rot was observed only in 3 species, *Populus deltoides*, *Fraxinus pennsylvanica*, and *Morus alba*.

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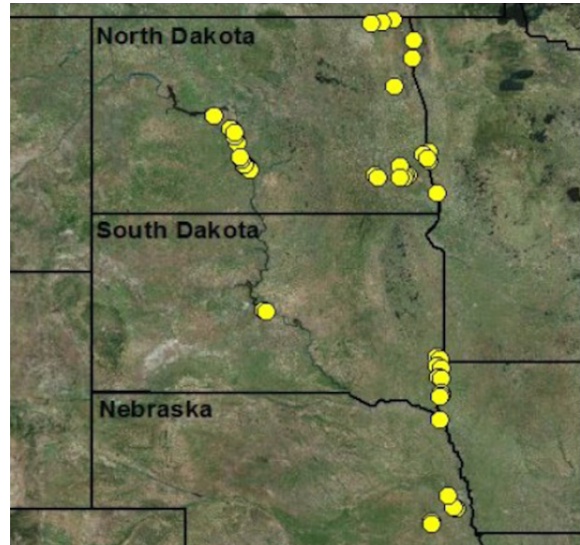


Figure 1. 2015 Distribution map for survey sites.



Figure 2. *Armillaria mycelium*.



Figure 3. *Armillaria* rhizomorphs in culture.

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Table 1. 2015 results summary table.

	Host Condition			
	Root Rot	Butt Rot	Epiphytic Association	Snag
<i>Populus deltoids</i>	5	1	1	3
<i>Frazinus pennsylvanica</i>	7	2	3	3
<i>Quercus macrocarpa</i>	1	0	0	1
<i>Acer negundo</i>	2	0	2	2
<i>Tilia americana</i>	0	0	0	1
<i>Ulmus americana</i>	2	0	0	1
<i>Acer saccharinum</i>	3	0	0	0
<i>Ulmus rubra</i>	0	0	0	1
<i>Salix</i> spp	1	0	0	0
<i>Morus alba</i>	1	1	0	0
<i>Gleditsia triacanthos</i>	1	0	0	0

USING TRANSCRIPTOMICS TO IDENTIFY CANDIDATE GENES ASSOCIATED WITH BLISTER RUST RESISTANCE IN WHITEBARK PINE (*PINUS ALBICAULIS*)

Zolton Bair¹, Richard Sniezko², Jill Wegrzyn³, Heather Lintz⁴, and Jeff Stone¹

ABSTRACT

Whitebark pine (*Pinus albicaulis*) is an iconic high-elevation tree species currently threatened by climate change, mountain pine beetle, and white pine blister rust, a lethal disease caused by the non-native fungal pathogen *Cronartium ribicola* that afflicts all North American white pines. One major goal in managing pine populations vulnerable to blister rust is to identify sources of heritable resistance that can be used in operational breeding programs (Schoettle *et al.* 2007). While USDA Forest Service breeding programs have identified major resistance genes in four other *Pinus* spp (Kinloch and Dupper 2002; Schoettle *et al.* 2014), no such R-gene is evident in whitebark pine. However, genetic variation in susceptibility to blister rust has been observed in naturally occurring populations. Although resistance phenotypes of whitebark pine differ from those in other pine species, certain seed parents are known to produce offspring that consistently develop fewer rust cankers compared to other seed parents under the same disease pressure, suggesting a genetic basis for partial resistance (King *et al.* 2010). By using transcriptomics to assess differential expression between these resistant and susceptible trees, we can detect genes that are differentially expressed in resistant individuals.

In collaboration with Dorena Genetic Resource Center, germplasm was collected from over 50 mother trees, representing whitebark pine popula-

tions throughout the Pacific Northwest. Three days after inoculation with blister rust, secondary needles were sampled and flash-frozen to preserve each individual's RNA expression profile. Half of the seedlings from each family served as a control group and were only inoculated after needles were collected. This sampling strategy permits comparisons between both experimental conditions, while still revealing the resistance phenotype of each individual tree seedling.

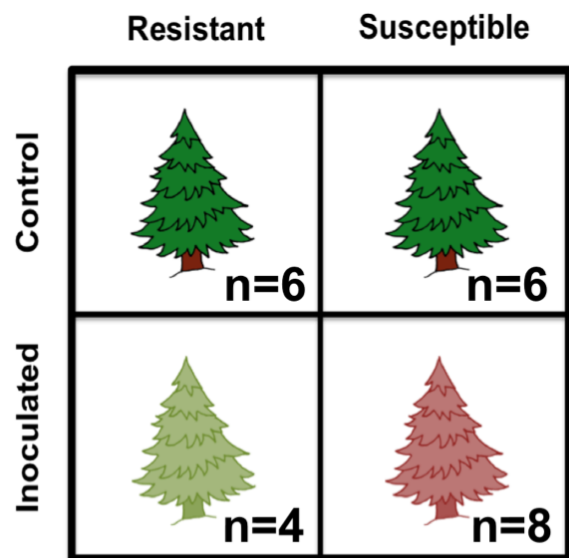


Figure 1. Sampling design permits transcriptome comparisons based on inoculation treatment and resistance phenotype.

Certain progeny of the Mt. Rainier half-sibling family ‘Shadow Lake 39’ exhibit partial resistance and were the primary focus of our bioinformatic investigation. Four representative seedlings from this family were deeply sequenced using RNA-Seq (Wang *et al.* 2009) and assembled *de novo* using Trinity (Haas *et al.* 2013). These assembled sequences were annotated with inferred transcript function, gene ontology terms, and predicted

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proteins to create a reference transcriptome. To identify genes associated with resistance, individual libraries were created from 24 seedlings were sequenced to yield mRNA expression data. Sequenced reads were aligned to the reference transcriptome before calculated gene expression counts using Corset (Davidson and Oshlack 2014). Comparative analyses of transcriptomes were conducted using edgeR (Robinson *et al.* 2010; McCarthy *et al.* 2012) to reveal differentially expressed genes. This comprehensive list was curated to reveal the top candidate genes associated with blister rust resistance in whitebark pine. As in

the case of *Pinus monticola*, these candidates can be used to develop gene-specific markers, permitting breeders to quickly screen individual whitebark pines for blister rust resistance using qRT-PCR (Liu *et al.* 2013). Following validation, these markers will help to expedite and economize resistance breeding programs and restoration efforts. Utilizing resistance trials, needle histology, and transcriptomic analyses in concert, this study has identified candidate genes associated with blister rust resistance in whitebark pine and has suggested potential mechanisms of partial resistance.

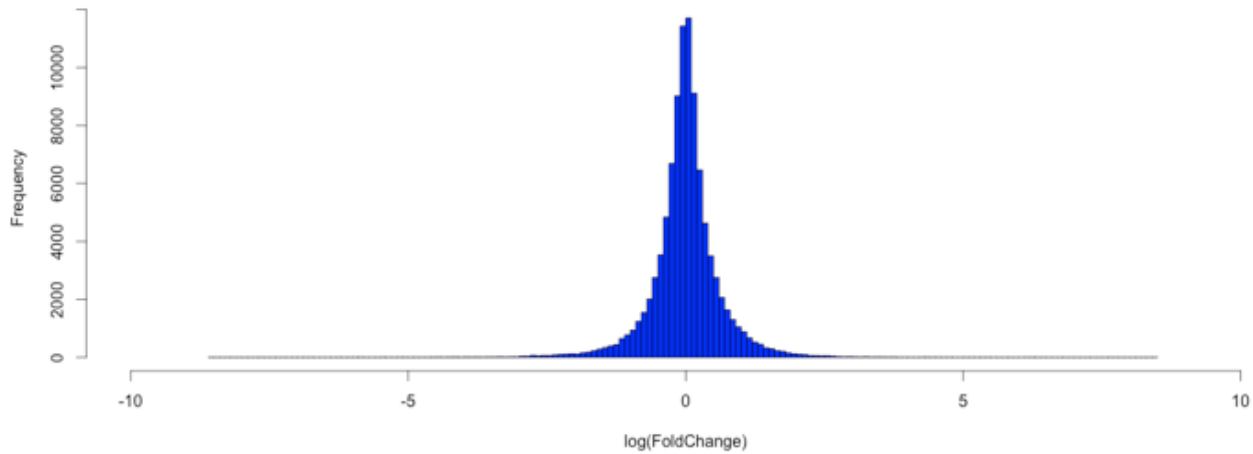


Figure 2. Comparative analysis demonstrates that the vast majority of genes are not differentially expressed between resistant and susceptible individuals.

ACKNOWLEDGEMENTS

This inoculation experiment was designed and executed in cooperation with Dorena Genetic Resource Center. The RNA extraction protocol and sequencing design were developed in conjunction with the Neale Lab at UC Davis. Library preparation and sequencing were conducted at the Center for Genome Research and Biocomputing. This study was funded by the Forest Service’s Special Technology Development Program (R6-2012), with additional contributions from the Whitebark Pine Ecosystem Foundation and a WIFDWC student travel award.

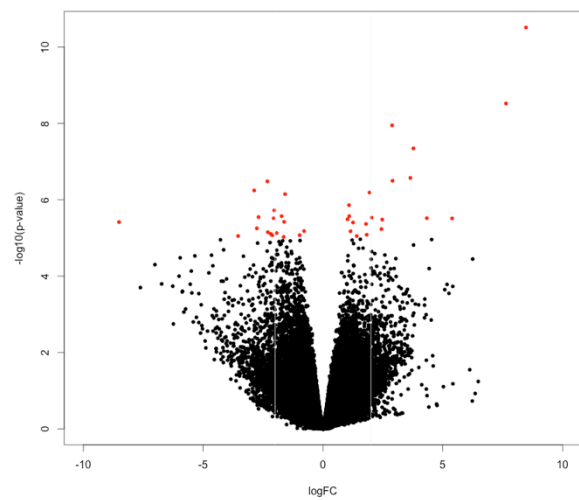


Figure 3. Volcano plot reveals genes that are most differentially expressed in resistant individuals ($p < 0.00001$).

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POPULATION GENETICS OF *PHAEOCRYPTOPUS GAEUMANNII*

*Patrick Bennett*¹

INTRODUCTION

The focus of my research is a Douglas-fir foliage disease dubbed Swiss Needle Cast (SNC) due to its discovery in Switzerland in the 1920s (Boyce 1940). The causal organism *Phaeocryptopus geummannii*, an ascomycete fungus, is one of the most common and abundant fungi in western Oregon. In the SNC pathosystem, disease is caused by the physical blockage of stomata by fruiting structures (pseudothecia) of the fungus (Manter *et al.* 2000). The main symptoms are chlorosis, premature foliage loss, and growth reductions (Hansen *et al.* 2000).

Previous studies have shown that there are two lineages, or subpopulations, of this fungus that co-exist in varying abundances across the landscape, and that these lineages appear to be reproductively isolated (Winton *et al.* 2006). The lineages can only be differentiated using molecular techniques. Inoculation studies suggested that these lineages might also differ in virulence (Winton 2001). It has also been established that climate variables such as winter temperature at spring/summer dew-point deficit are significant predictors of disease severity in the field, though the mechanisms of climate influences are not understood (Manter *et al.* 2005, Stone *et al.* 2007, Stone *et al.* 2008).

OBJECTIVES

For my thesis research I am employing DNA microsatellites (repetitive regions of the genome with highly conserved flanking regions) to further investigate the distribution of these two lineages

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across a latitudinal gradient from the northern border of California to British Columbia. Given that there are differences in the distributions of these lineages, and that they may differ in virulence, I hypothesize that there will be a distinct relationship between the relative proportions of these lineages present and disease severity at each of the study sites.

I am also interested in describing the genetic structure of these populations by analyzing multilocus microsatellite genotypes. My hypothesis is that the lineages will be strongly and significantly differentiated, and I expect to find evidence that confirms their reproductive isolation. This may lead to the determination that the lineages constitute separate species.

In addition to the population genetics investigations, my thesis research aims to determine the relative severity of the symptoms caused by each of the lineages through a series of controlled inoculation experiments with several distinct genotypes of each lineage. I will choose a subset of these inoculated individuals with which to directly assess the influence of climate variables such as winter temperature on disease development.

CONCLUSIONS

These findings will provide insights into the mechanisms of fungal speciation in sympatry, and may contribute to a more thorough understanding of the dynamics of this disease. A better understanding of the distribution of the two lineages and their influences on disease will inform forest health and land management decisions, but will also allow for effective prediction of disease risk and appropriate mitigation strategies. By determining the role of climate in this disease, forest pathologists may gain a better understanding

of potential future outbreaks of fungal diseases in a changing climate.

ACKNOWLEDGEMENTS

Members of the Hansen Lab at Oregon State University, Paul Reeser and Wendy Sutton, provided invaluable support, assistance, and advice on the laboratory techniques and protocols used in this study. The Swiss Needle Cast Cooperative, The Portland Garden Club, Cascade Mycological Society, and the US Forest Service have provided funding for this research. Their contributions are gratefully acknowledged. I would also like to acknowledge the Department of Botany and Plant Pathology and the Graduate School at Oregon State University for their generous contributions to student travel.

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DETERMINING THE RELATIVE AMOUNT OF SOIL-BORNE INOCULUM OF *PHYTOPHTHORA RAMORUM* WITHIN AN OREGON TANOAK FOREST

Christina Benemann¹ and Jennifer Parke¹

ABSTRACT

Phytophthora ramorum, the causal agent of Sudden Oak Death (SOD), is an invasive phytopathogen that has caused extensive mortality of oaks and tanoaks in California and southern Oregon. Rain readily spreads the pathogen down through the canopy, causing infection of lower parts of the tree and neighboring plants.

Although this top-down aspect of the disease cycle is well understood, the importance of soilborne inoculum as a contributing factor to the spread of the disease remains unclear. Our study aims to: (i) compare the amount of inoculum washed down through the canopy to that coming from the soil and (ii) to detect and quantify inoculum in relation to

soil depth. Throughout the rainy season, both rainwater and soil samples were periodically collected from within the Generally Infested Area in Brookings, Oregon. A tiered bucket design was used to capture throughfall and splash-up from the soil surface during rain events. Detection of the pathogen in both rainwater and soil was done culturally through established baiting techniques. Quantification of the pathogen will be achieved utilizing quantitative PCR, which should detect measurable differences that will indicate how inoculum from infected canopies and soil contribute to the total inoculum pool.

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PHTYOGENETIC IDENTIFICATION OF ENTOMOPATHOGENIC AND ENDOPHYTIC FUNGAL POPULATIONS IN WEST COAST DOUGLAS-FIR FOLIAGE IDENTIFIED AS *PHAEOCRYPTOPUS GAEUMANNII*-INFECTED

Dixie Daniels¹ and Jim Kiser¹

ABSTRACT

Defoliation of coastal coniferous forests in the Pacific Northwest has been previously linked to Swiss Needle Cast disease caused by the fungus *Phaeocryptopus gaeumannii*. Current affected areas are in excess of half a million acres along the coast range of Oregon. Research will focus on the mechanistic ecology of endophytes, including potential alternate vectors for defoliation. One pathogen in particular, *Sydowia polyspora* has been implicated as a needle cast pathogen in a number of studies in Canada and Europe in conifer species including pines, firs, hemlocks, and others. Research goals are to provide a current baseline for endophytes in coastal Oregon conifers for: three age-classes of needles, three vertical strata within the tree, along different geographic coastal zones within Oregon. These baselines will help to characterize fungal communities in association with *Phaeocryptopus gaeumannii* that may be additionally implicated in Douglas-fir needle-cast (*S. polyspora* and *Rhabdocline* spp for example). Coastal Oregon forests provide a number of important interregional ecological and economic benefits. In addition to the timber growth potential, coastal forests produce abundant biodiversity and

provide the economic viability for the coastal communities. Oregon wood production occurs on about 36% of the timber land base and accounts for approximately 17% of the U.S. lumber output with an estimated value of 1.9 billion dollars on 4.2 billion board feet. Employment related to wood production accounts for approximately 60,000 jobs in the state. The additional 64% of the timberland base supports water resources, recreation, and wildlife habitat. Longer term silvicultural management of coastal forests will benefit from knowledge gained from this project. A better understanding of endophyte interactions will enhance opportunities to understand and implement various mixed-species plantings. Current strategies are to replace monoculture stands with mixed species plantings of primarily western red cedar and western hemlock. It is vital to understand if we are mitigating what we believe to be a single-species pathogen, or proving new vector-targets for the complex of pathogens involved.

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CAPACITY OF OLD SUPPRESSED BEECH TREES TO ADJUST XYLEM ANATOMY AND THEORETICAL HYDRAULIC PROPERTIES AFTER THINNING

Estelle Noyer^{1,2}, *Barbara Lachenbruch*^{3,4}, *Jana Dlouha*^{1,2}, *Catherine Collet*^{1,2}, *François Ningre*^{1,2}, and *Meriem Fournier*^{1,2}

INTRODUCTION

Canopy opening increases resource availability around trees. However it also induces an increase of mechanical loads applied on newly isolated tree, and an increase of soil and air temperatures (Aussenac 2000). Changes in foliar and wood traits (Caquet *et al.* 2009) are generally observed and interpreted as an adaptation to the new microclimatic conditions. Here, we will focus on the trunk growth and xylem anatomy adaptation of two different developmental stages of beech trees after thinning.

MATERIALS AND METHODS

Two samples were used for the study. Sample 1 included 42 old suppressed beeches (60 to 100-years-old). Half of those were thinned during the winter 2007-08 and all trees were harvested in 2013. For the sake of anatomical comparison, we sampled 5 dominant mature beeches of similar age in the same stand. Vessel anatomy of seven rings before and six rings after the thinning was analyzed. Sample 2 included 15 dominant beech saplings (11 to 18-years-old), five saplings were not released, five saplings were thinned in 2007 and five saplings were released in 2011. All trees were harvested in 2013.

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We analyzed vessels features, namely vessel diameter and frequency. Then, we computed the hydraulic diameter D_{HP} (mean of the power four of diameter relevant for hydraulic conductivity), the theoretical specific conductivity K_s and the theoretical stem specific conductivity K_T following the work of Steppe and Lemeur (2007).

HYPOTHESIS TO TEST

1. What is the hydraulic demand in suppressed and dominant trees and the corresponding xylem anatomy?

While dominant trees have to manage with the high and variable evapotranspiration demand, the micro-climate of suppressed trees is buffered by the forest cover (Aussenac 2000). This difference is reflected in the xylem anatomy (Gebauer *et al.* 2014).

- **H1a:** Canopy dominant trees are in a less buffered environment than suppressed trees. To maintain the same performance, dominant trees will need to have higher hydraulic safety.
- **H1b:** Because of a less variable environment, suppressed trees have more constant hydraulic properties from year to year than trees of the same age that are in the canopy.

2. Which reaction is expected in suppressed trees after thinning?

Suppressed trees grow up under water- and light-limited condition resulting in a slow growth rate. After canopy opening, the radial growth increases

with resource availability (Collet, Lanter, and Pardos 2001). Larger xylem area induced by thinning increases potential transpiration rates and sap flow allowing hydraulic shoot performance adjustments to water demand of the crown. But conductivity capacity of functional wood is different, following ring structure (McCulloh *et al.* 2010). If the individuals are diffuse porous, we expect them to have the same hydraulic performances (K_s) regardless of radial increment. If they are ring porous, we expect at higher radial growth rate, that the K_s will decrease because the earlywood band will stay the same width and latewood will make up the rest of the growth ring. Beech trees are usually seen as diffuse porous but it has tendency to be semi-porous (Gasson 1985). We can expect that at slow growth rate, we will have a relatively uniform wood, but at faster growth rate also having a band of latewood, made of smaller-diameter vessels at lower frequency.

- **H2:** Face to a demanding environment, released trees need to be risk averse. We expect wider rings to have a broader distribution of vessels diameters with more vessels in the small-diameter class.

3. Comparison thinning reaction of saplings and old suppressed beech trees.

A previous study on beech suppressed saplings shows hydraulic performances increase strongly after thinning (Caquet *et al.* 2009). In young sapling (small trunk diameter), we expect the ring width increase to have an important effect on xylem hydraulic properties. In large trees, speeding up of the growth will impact the available conductive area more than in small trees with small circumference and so limited absolute increase of conductive area. Comparing response of old suppressed beech and dominant beech sapling, we will test the last hypothesis:

- **H3:** In sapling, we expect a great effect on K_s after thinning while in large trees we

expect K_h to be mainly ensured by increased growth rate.

CONCLUSIONS

We hope this study will allow us to better understand the mechanism of the tree hydraulic adaptations after thinning and its variability in function of the tree social status and developmental stage.

FINANCIAL SUPPORT

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THE EFFECTS OF SEED SOURCE AND PLANTING ENVIRONMENT ON DOUGLAS-FIR FOLIAGE DISEASES

Nicholas Wilhelmi¹, Dave Shaw¹, Connie Harrington², Brad St. Clair³, and Lisa Ganio⁴

ABSTRACT

Severe impacts due to pathogens and insects are commonly associated with maladapted Douglas-fir (*Pseudotsuga menziesii*) populations. The foliar pathogens *Phaeocryptopus gaeumannii*, the causal agent of Swiss Needle Cast, and *Rhabdocline* spp, the causal agent of *Rhabdocline* needle cast, are two very important Douglas-fir pathogens. These pathogens have been shown to disproportionately affect genetically maladapted seed sources, causing serious growth impacts and sometimes mortality. The relationship between the levels of susceptibility/tolerance to these foliar pathogens and the climate of the seed source is a key component in the identification of proper seed sources for reforestation. Understanding the variation in susceptibility/tolerance to Swiss needle cast and *Rhabdocline* spp will be influential in the modification of current seed zones and the successful movement of seeds to novel locations.

This study is part of the Douglas-fir Seed Source Movement Trials, a large scale common garden, reciprocal transplant study. The study is comprised of 12 diverse west side Douglas-fir (*Pseudotsuga menziesii* var. *menziesii*) seed sources, ranging from northern California to southern Washington. These seed sources are planted in nine diverse planting environments ranging from southern Oregon to southern Washington, from the high elevation to the coast.

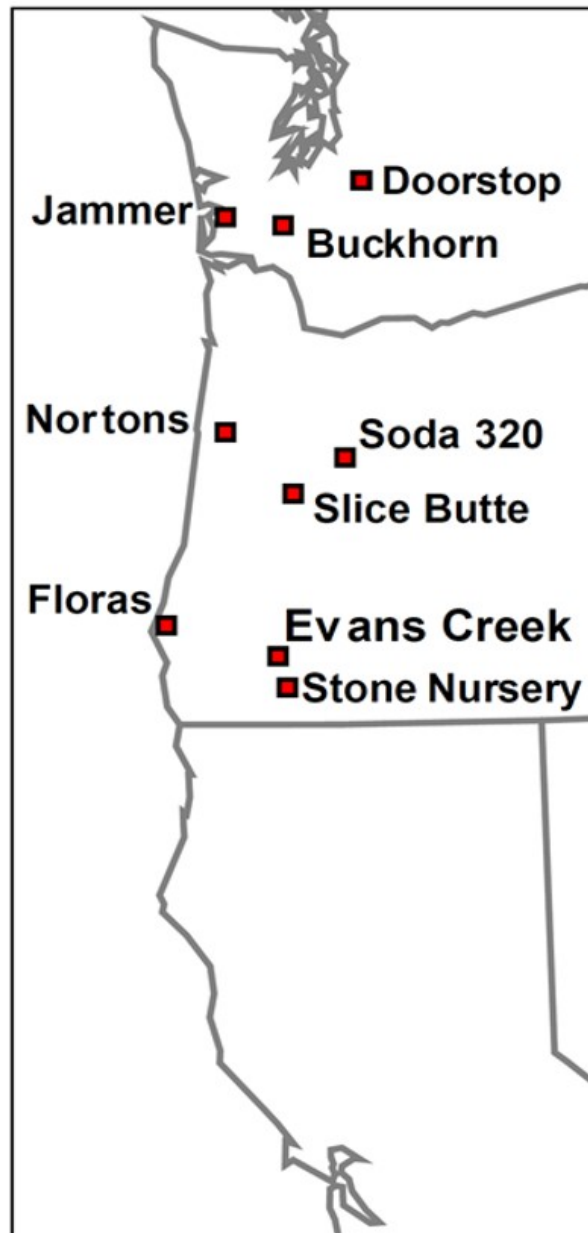
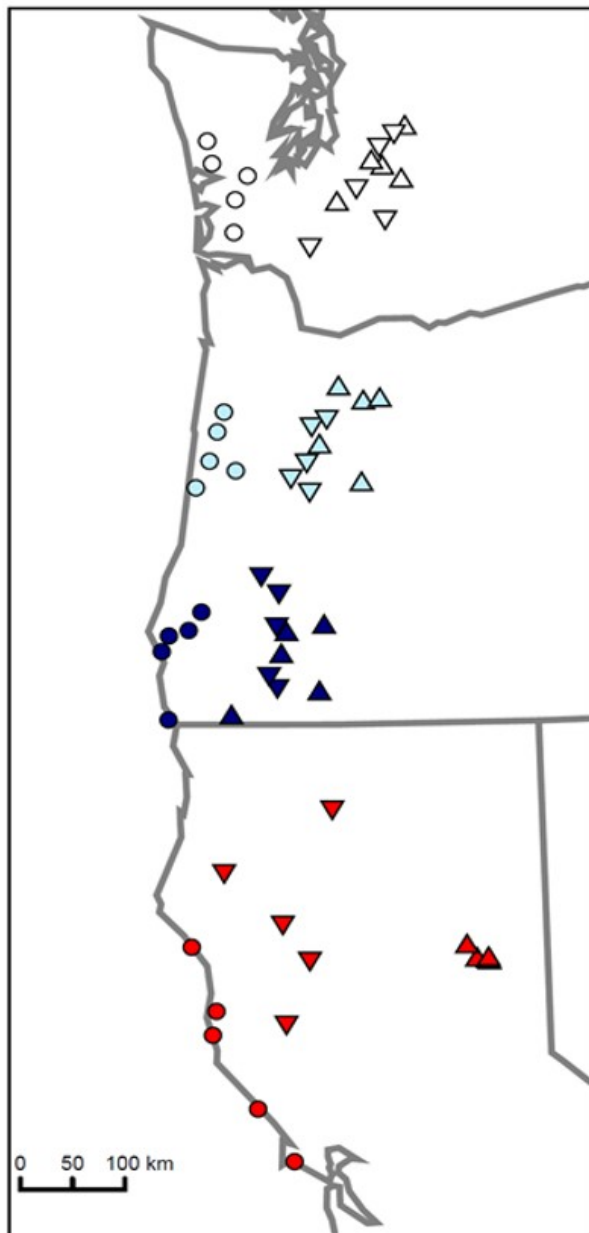
These sites and seed sources were chosen to represent the wide variation in temperature and precipitation of western Oregon and Washington and offer an incredible opportunity to assess the influence *P. gaeumannii*, and *Rhabdocline* spp will have on these seed sources under various climate scenarios. Similar studies have compared inland Douglas-fir (var. *glauca*) to west side Douglas-fir (var. *menziesii*), whereas this study is composed strictly of west side Douglas-fir (var. *menziesii*). Every tree in this study was assessed for the presence and impacts of *P. gaeumannii* and *Rhabdocline* spp. We rated infection levels, needle retention, crown color and crown density. Our objectives are to: 1.) Identify variation in the incidence and impact of these pathogens among these different seed sources. 2.) Compare climatic variables between locations of seed sources and test sites to identify patterns in susceptibility/tolerance related to climate. We hypothesize that: 1.) Susceptibility/tolerance to *Rhabdocline* spp and *P. gaeumannii* is a function of climatic differences between the seed source climate and the planting environment. 2.) Seed sources from regions of high spring/early summer precipitation, low continentality index, and high mean winter temperatures are least affected by *P. gaeumannii*. and *Rhabdocline* spp.

Through this project we will provide a better understanding of the impact *P. gaeumannii*, and *Rhabdocline* spp will have on west side Douglas-fir populations under different climatic conditions. An increased understanding of the varying levels of susceptibility/tolerance to these foliar pathogens will provide land managers valuable information to assist in the identification of proper seed sources.

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60 Populations

9 Planting Locations



- | | |
|----------------------|-------------------------------------|
| ○ Washington Coast | △ Washington Cascade High Elevation |
| ● Oregon Coast North | ▽ Washington Cascade Low Elevation |
| ● Oregon Coast South | △ Oregon Cascade High Elevation |
| ● California Coast | ▽ Oregon Cascade Low Elevation |
| ▲ California Sierra | ▲ Oregon Siskiyou High Elevation |
| ▼ California Klamath | ▼ Oregon Siskiyou Low Elevation |

Figure 1. Seed Source and Test Site Locations of the Douglas-fir Seed Source Movement Trials.

**Panel: Drought and Other Factors as
Contributors to Tree Mortality**

STRUCTURAL MECHANISMS USED BY WOODY PLANTS TO DEAL WITH WATER STRESS

*Barbara Lachenbruch*¹

Water availability is vital to many plant functions. Even though trees can go dormant during periods of water scarcity, unlike annuals, long-lived plants cannot simply go to seed and wait it out. Therefore, it is not surprising that a region needs at least a threshold amount of water to support a forest, as shown by superimposing a precipitation map of the contiguous states with a map of the current potential of forest distribution.

If woody plants have insufficient water, they are likely to have stunted growth, disease, and dieback. Plants need water 1) for photosynthesis (as a hydrogen and electron donor for chemical reactions, to lose by passive diffusion in exchange for CO₂ that comes into leaves through open stomata, and for evaporative cooling). If the water supply is insufficient for chemical reactions or for passive loss during periods in which CO₂ is diffusing in, plants may have too little carbohydrate for growth, defense and putatively for embolism (air bubble) removal. Moreover, they may overheat, which can cause proteins to denature and processes to occur at non-adaptive rates. Plants need water 2) to generate turgor pressure (to give mechanical support, to allow some movements such as opening and closing guard cells of stomata, and to allow cell expansion during growth). Without this water, the parts of plants with little secondary xylem for stiffness are likely to wilt (e.g., foliage, shoot tips), and growth will decline and/or be abnormal with cells and their features abnormally small. Plants need water 3) to serve as a carrier to move minerals and organic compounds into and within plants of short and long distances. Without sufficient carrier water, growth and function will be limited by the least available factor.

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A plant with insufficient water may simply live with that stress. The first reactions to stress are its detection and then production of mechanisms for protection and repair at a higher level of stress or a prolonged stress, the plant begins to repair, acclimate, and/or adapt. However, at extreme levels of stress, the plant will fail, at a plant part (e.g., dieback), an entire plant, and even at the level of an entire population.

To understand the structural mechanism plants use to deal with this water stress, it is helpful to first review the mechanism by which water ascends trees, called the cohesion-tension mechanism. Water moves as one continuous mass from the soil all the way up through the stomata. Because water has such strong cohesive forces, when it evaporates out at the leaf, it pulls the water up through the stem. If air gets into the pathway, the water column will break, and the plant will have some degree of 'hydraulic dysfunction,' and thus it is very important to plants to have mechanisms to avoid breaking the water column.

The amount of embolism sustained without going into extreme stress depend on the plant's 'strategy', and can be discussed as its 'hydraulic risk'. The focus of this contribution is on the situations in which plants lower their hydraulic risk through its structure (e.g., architecture, allocation pattern, anatomy), but they can also lower risk through phenology, stomatal behavior, and chemistry (e.g., osmotic regulation).

Plants can be aided to resist a drought through structural changes at the cell, tissue, and organ levels at the cell level, an increase in pitting from the conduits (vessels or tracheids) to the parenchyma could aid the plant in recovery from embolism. Changes in conduit length and diameter, and in pit geometry and frequency, can both increase xylem conductivity, which if all things are

equal, will allow plants to have less negative water potentials and thus avoid embolism in certain circumstances. If the inter-conduit pit geometry changes, or the location and extent of xylem blockages (like tyloses or embolisms) changes, then again, conductivity can be altered and the water potential at which embolism occur can be changed. Structural changes to the individual cell (such as bark or cuticle) a change plant water loss, and changes to guard cell geometry can increase the water use efficiency of foliage.

At the tissue level, structural changes that could alter xylem recovery from embolism and/or capacitance would include increasing the proportion of xylem parenchyma, frequency of rays, and the proximity of the ray and other xylem parenchyma to conduits. Xylem efficiency could be altered with a change in the proportion of earlywood, sapwood width, or with the use of sectoriality, which is the favoring of one part of a canopy by a particular segment of sapwood, or with spiral grain, which could distribute water all around the canopy. The vulnerability to embolism could be altered by changing earlywood or fiber proportions. Water loss could be decreased by decreasing the frequency of stomata and/or lenticels. Lastly, foliage water use efficiency can be altered at the tissue level by changes in leaf mesophyll characteristics.

At the organ or whole-plant level, structural changes that could alter xylem recovery from embolism include changing the allocation to root tissue, particularly if the root has a higher proportion of available carbohydrates (for embolism reversal) as suggested by recent research. A stem's conductive efficiency can be altered by changing the amount of sapwood, by the plant being shorter (rather than taller), and by having a higher proportion of xylem that has an old cambial age (because of the phenomenon that outer

wood has higher conductivity than corewood). Xylem vulnerability to embolism could be decreased by increasing the root/shoot ratio and/or the sapwood/leaf area ratio, both of which could be accomplished through growth or through shedding of parts. Root efficiency at harvesting water can be changed with an increase in root system size, spread, and depth, and plant water loss can be decreased by a plant assuming a more compact growth form, thus increasing the boundary layer, and retarding evaporation.

There are still many issues we have yet to understand about how structure influence a plant's water relations. A few examples that may prove interesting are the role of needle insertion depth. (from which growth rings is a long-lived needle withdrawing water when it transpires?), how widespread is 'sectoriality' (the restriction of axial water transport to a subset of the cross-section for water moving from one height in a tree to another), the role of latewood (and other tissues that have low resistance to embolism) as a locus for short-term water storage, and the extent to which the relative amount of wood of different cambial age pre-adapts a tree for survival in a drought environment. Although wood density is sometimes used as a proxy for drought resistance, I caution that such correlations only work in very restricted circumstances.

In conclusion, plant structure can play key roles in the ability of a plant to handle drought. That fact should mean that managers should be able to monitor structure and then use it to gauge the potential for individuals (and species) to handle different degrees of drought stress. Such work may be helpful for understanding plant performance at different ages, for selective thinning regimes designed to decrease the stand's drought-vulnerability, and for selecting species, genotype, and nursery stock for marginal sites.

TRACKING ANNUAL ARMILLARIA ROOT DISEASE ASSOCIATED MORTALITY IN A YOUNG PLANTATION

M.P. Murray¹ and A. Leslie²

INTRODUCTION

Tree root disease caused by pathogen, *Armillaria ostoyae*, has notable influence on ecological and economic systems of southern British Columbia (BC) and the northwestern United States. The economic importance of *Armillaria* is closely tied to timber production. This disease impacts sustainable forest management due to its ability to increase following logging, reduce tree growth rates, and cause mortality, especially in young regeneration (Cruickshank 2000; Morrison 2000). The earliest stages of plantation development may be especially vulnerable due to the tendency for *Armillaria* to cause heavy mortality during the first 5-20 years of a rotation (Morrison and Pellow 1994; Morrison and others 1988). Insight regarding early spread and mortality can be useful for guiding reforestation practices.

Several field trials have quantified the temporal progression of *Armillaria* within young plantations of southern interior BC. These trials are characterized by stump removal treatments for assessing root disease control and were sampled every 2-10 years spanning 11 to 35-year periods.

We are investigating annual infection and death trends of regeneration associated with *Armillaria* during the first 21 years of a plantation. Our dendrochronological approach is focused on three tree species commonly used for re-planting harvested sites in the Province: Douglas-fir, western larch (*Larix occidentalis* Nutt.), and lodgepole pine (*Pinus contorta* Dougl. ex Loud. var. *latifolia* Engelm. ex S. Wats.). We are also

examining potential climatic influences which may impact host radial growth and contribute to the timing of death.

METHODS

The research trial is a 40.8 hectare plantation that was formerly dominated by lodgepole pine and larch. It is located near Knappen Creek, approximately 46 km north of Grand Forks, BC near the U.S. Border. In 1988 and 1989 all merchantable trees were removed, then replanted with larch and lodgepole pine in 1991. Prior to planting, root disease centres were mapped and high infection was observed. The site was fill planted in 1995 with lodgepole pine in areas where cattle and rodent damage had occurred. Naturally regenerated Douglas-fir is common throughout the site, and there are small amounts of naturally regenerated western redcedar (*Thuja plicata*) and western white pine (*Pinus monticola*). We confirmed the root disease species as *A. ostoyae* using a PCR (polymerase chain reaction) test coupled with sequencing.

In 1997, twenty plots, each 31.6 meters by 31.6 meters were randomly positioned throughout the research trial area. Within each plot, all trees were located on an X-Y axis, mapped, and tagged (3,219 trees). The species of each tree was recorded and measured for height, diameter at 1.3 meters (dbh), leader growth in 1995 and 1996, origin (planted or natural regeneration), health and damage.

In October 2011, a total of 2,997 tagged trees were found and re-assessed for forest health agents and growth. For dead trees, the cause of death was determined wherever possible. All dead and dying trees were examined for evidence of mycelial fans indicating *Armillaria* infection under the bark at the root collar or on exposed roots. A total of 329 trees were dead. Of these, 207 trees were found to have *Armillaria*. In October 2012, all dead trees with *Armillaria* evidence were sampled by sawing two cross-section discs from each tree. One was

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collected from 92 sound wood nearest the root collar, and the second was taken at 1.3 meter height. Cross-sections were scanned using a flat-bed scanner at a minimum of 1200 dpi, then analyzed and recorded using Coorecorder 7.6 (Cybis Elektronik 2015). Knowing the year of planting enabled estimates of death dates. For naturally regenerated trees, we relied on earlier surveys to make estimates.



Figure 1a (top) and **1b** (bottom). Sampled cross-sections from a single Douglas-fir tree infected by *Armillaria* illustrate reduction of radial growth at 1.3 meters (1a) and lack of ring symmetry characteristic at soil line (1b).

To reduce potential errors in date estimations, cross-dating was performed between each dead tree and a master chronology based on healthy live trees sampled at the site. We used the skeleton plot method (Stokes and Smiley 1996) which was computed with CDendro (Cybis Elektronik 2015) based on Cropper (1979). For each tree, we compared our original estimation to the skeleton plot match. Where disagreements of >1 year occurred, the tree series was discarded. Otherwise, we chose the skeleton plot date. Years of *Armillaria* infection were indicated by a one or more consecutive narrow growth rings immediately preceding death. The first year of infection was determined by the existence of a 50% or more reduction in width compared to preceding rings followed by uninterrupted reduced growth until death. Cross-sections taken at breast height provided better uniformity and symmetry of ring growth patterns (Figure 1). Figure 1a (top) and 1b (bottom). Sampled cross-sections from a single Douglas-fir tree infected by *Armillaria* illustrate reduction of radial growth at 1.3 meters (1a) and lack of ring symmetry characteristic at soil line (1b).

RESULTS

From the 207 dead trees associated with *Armillaria*, 53 had been damaged by wood boring insects or too rotten with unreadable complete tree ring series. An additional 57 trees did not cross-date due insufficient ring numbers for comparison or original date estimates were no within a single year of our cross-dating. Thus, a total of 97 trees were used for analysis: (Douglas-fir (46), larch (17), and lodgepole pine (34). The first *Armillaria* infection was evident in 1999 with mortality beginning in 2000 (Figure 2). New infections increased overall until 2006 followed by a decline. The average number of years from initial infection to death was 2.3, 3.6, and 2.7 for each species respectively. All deaths occurred consecutively during a 13-year period, with 41% of deaths during 2007-2008.

DISCUSSION

The trend we observed roughly mirrors two bell-shaped curve. The infection curve precedes the mortality curve along the observed time sequence (Figure 2). A similar bell-shape curve has been reported by Morrison and Pellow (1994) where the mortality peaked at 10 years. Peet and others (1996) documented peaks in the number of infected trees at 14 and 19 years for two plantations. Both of these studies indicated peaks were followed by a less pronounced decreasing trend extending to plantation ages of 22-27 years. *Armillaria ostoyae* spreads slowly belowground and therefore is not associated with epidemics where large numbers of hosts become infected and die within a time

window of several years or less. Instead, owing to the gradual growth of rhizomorphs and roots, *Armillaria* spread may not exceed much more than 1-1.3 m/yr within tree plantations of the Pacific Northwest.

Most trees that died in 2007 had been infected no more than a single year. This may indicate another influence on the high mortality count. We suspect that the extremely warm and dry year of 2007 contributed. We further considered stocking densities, plot locations, frost rings, and daily weather extremes but no patterns or relationships were readily apparent. Further data analysis may better reveal relationships between mortality and climatic factors.

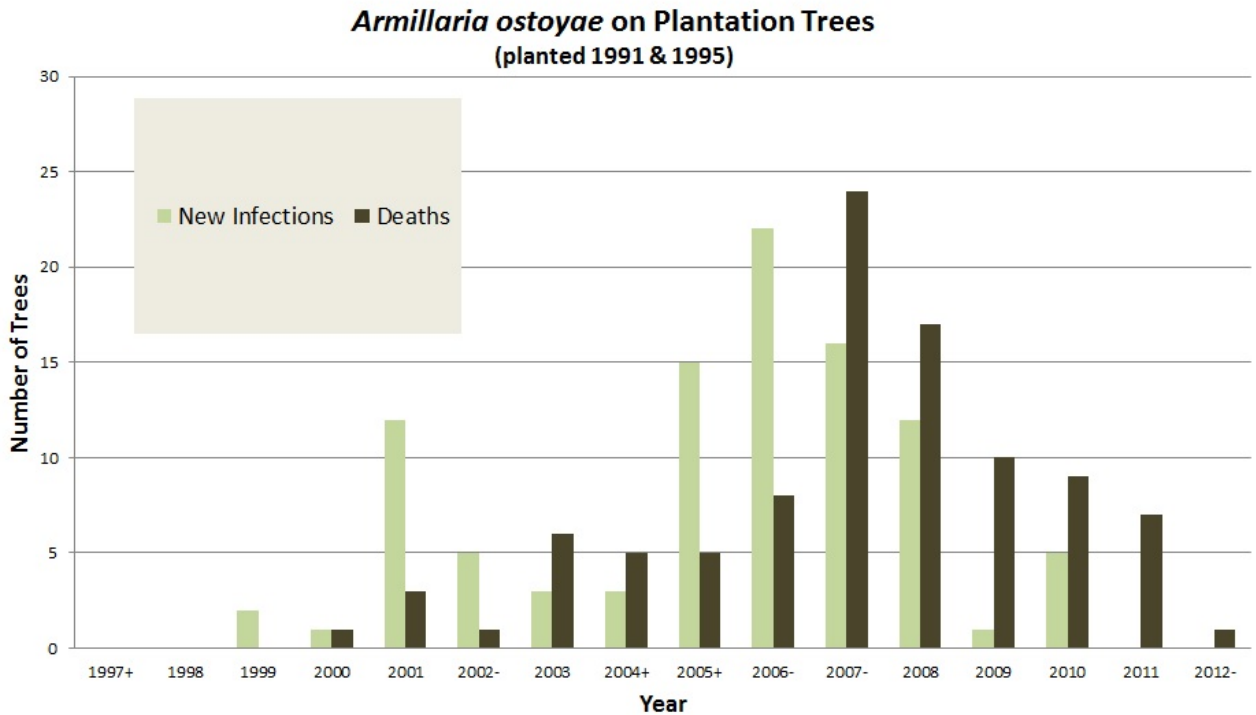


Figure 2. Time sequence of associated with *Armillaria* root disease.

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WORKING PAPER: ISSUES CHALLENGING THE INTEGRATION OF FOREST PATHOLOGY WITH CLIMATE CHANGE SCIENCE

*Richard C. Cobb*¹

The rapid increase in peer reviewed analysis examining the causes of recent mortality events is an exciting opportunity for forest pathology. Much of this literature has focused on tree-level processes and characteristics with a substantial emphasis on ecophysiological dynamics (Adams *et al.* 2009, Anderegg *et al.* 2012, Hartmann *et al.* 2013, Allen *et al.* 2015). The scope and emphasis of the collaborations driving this research are rapidly expanding beyond the individual tree level to include the consequences of these events with generalized predictions of forest function under climate change (Allen *et al.* 2010, Millar and Stephenson 2015). However, prediction of the timing, extent, and intensity of tree mortality has been a persistent and difficult problem that these collective efforts have been unable to address. There is a clear role of Forest Pathology in improving these predictions, particularly in using knowledge of primary and secondary pathological agents of mortality to improve forest mortality forecasts.

Ample reason also exists for concern, alarm, and annoyance among forest pathologists in regards to this recent expansion of the literature. Past high profile forest health issues have transposed cause and effect (Skelly and Innes 1994) and the lack of integration of well over a century of basic and applied research on forest pathology in forest mortality-climate change analysis is certain to create poorly underpinned predictions that in turn could lead to poor application of management resources. Forest pathology has a clear opportunity to intercede and avoid these errors when mortality events center on the role of pathogens but, accom-

plishing this important task is dependent on communicating existing knowledge to the body of researchers working on global change tree mortality issues. As this group is largely ecologists and plant physiologists, much of the detailed knowledge of individual pathogens, their epidemiology and ecology, as well as forest management practices which influence the emergence of disease are largely absent from recent analysis (Allen *et al.* 2015, Millar and Stephenson 2015). Communicating this knowledge and adapting it to ecological approaches to forest mortality will aid in this effort. Additionally, the field must adapt protocols and identify opportunities to integrate climate change driven problems with those caused by pathogens.

Integrating Forest Pathology Data with Broader Forest Health Issues: The Basics

Ample common ground exists among ecologists and forest pathology practitioners. This common ground is that almost any individual working on wildland and managed forest pathogens is also an ecologist studying the interaction of individual pathogen species with their hosts and the environment. Although not all pathogens are microbes, the importance of microbial pathogens to forest disease creates another filter and potential challenge to collaboration and communication: most forest pathologists are also microbiologists, a field that changes at a pace equal to the rapid development of molecular tools to identify pathogens, genes that confer pathogenicity and pathogen biology. Of course, pathologists also use these tools on the host side of disease problems (Bostock *et al.* 2014) which provides the toe-hold needed to begin an integration of approaches.

Forest pathologists have the most clear cut and needed role in integrating molecular approaches in climate change issues of forest health. Doing so

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will also require the field to vet and develop common, transferable approaches to integrating molecular tools with ecological approaches. What methods should be applied to what problems? While the practicality of this question is compelling, it is wholly the wrong point of departure. Given the range, cost, and limitations of molecular methods in Forest Pathology, "What is the objective?" is a more useful place to begin. Tree health, infection, and mortality are fundamental components of forest disease at stand and individual levels. However, I would wager that 'infection' is the only of these terms with an agreed upon definition among pathologists and ecophysiologists. It is also the state and process where molecular methods have the most application in identifying and confirming the presence of particular pathogens within symptomatic plant tissues. While ITS-type confirmatory analysis is a reliable and low-cost method of describing well known pathogens, these methods are limited in ability to describe entire microbial communities or monitor inoculum levels (c.f. Strickland *et al.* 2009). The rapid development of new sequencing approaches has revolutionized the identification and description of microbial communities, but use of these techniques in monitoring inoculum remains problematic.

It may give the readers of this paper pause when I state that 'mortality' does not have an agreed upon definition. This perspective is colored by a problem in plant physiology; similarly to the often invoked definition of pornography, you know a dead tree when you see one. But identifying thresholds of plant resources that determine when an individual plant can no longer recover from an environmentally or biologically induced stress has been surprisingly difficult to identify (Anderegg and Callaway 2012). Identifying when green, physiologically active plants reach a point where mortality is inevitable is critically important to development of early warning systems, such as monitoring based on remote sensing data. Collaborative work between plant physiologists and pathologists could help address this problem.

Given that different pathogens attack and interfere with different plant parts and functions (Oliva *et al.* 2014), experimental manipulation of pathogens and environmental stress on trees could yield variable impacts to non-structural carbohydrates (NSC - soluble carbon plant resources) vs plant vasculature and water relations (c.f. Hartmann *et al.* 2013). For monitoring of mortality at broader scales, such as entire stands and forested regions, classifying mortality based on leaf/needle coloration (e.g. green vs brown/red/grey/etc) will be adequate.

Plant health ratings are arguably the most subjective forest health data routinely collected by forest pathologists and ecologists alike. Rating the relative health of a plant given the amount of potential canopy, frequency of damage to leaves, twigs, or roots, or suppression by neighboring trees is simple and useful field data. Subjectivity among observers is a constant concern with these types of data which places an important burden on pathologists and ecologists alike to develop common goals and guidelines for these data. While individual plant health data is generally avoided in meta analysis of insect and pathogen impacts to plants (Desprez-Loustau *et al.* 2006, Jactel *et al.* 2012), constant re-evaluation of these protocols with ecologists may improve their application.

More Difficult Problems: Doubly Censored Data

Data censoring is a term from statistics developed to describe several special cases of incomplete observation. Incomplete information is common in public health, individuals in a study fail to complete surveys or annual clinical visits, individuals die, or they simply survive without infection or mortality within the timeframe of the study (Militino and Ugarte 1999, Pan *et al.* 2014). This creates data where the individual has not been observed over an interval, where no further data can be collected, or where an event of interest has not yet occurred (respectively). In each of these cases, well tested statistical methods are available to analyze datasets with these types of partial

information. For example, infection and mortality rates of tanoak (*Notholithocarpus densiflorus*), where estimated from datasets where individual trees were observed every year, two years, three years, or up to once in six years (Cobb *et al.* 2012). Forest pathologists are increasingly faced with the task of associating environmental conditions with the prevalence and impact of individual pathogens (Woods *et al.* 2005, Sturrock *et al.* 2011, Weed *et al.* 2013). The combination of censored datasets along with the clear need to identify climate-pathogen relationships creates a unique challenge to statistical analysis of forest pathology datasets (Figure 1). How does one associate climate conditions to an individual infection or mortality event that occurred during a window of 2-10 years? Forest inventory and analysis data (FIA data) is arguably the most comprehensive and most broad-scale data available on forest health trends in the United States, yet the data are collected every 10 years - plots are censused every 10 years on a rolling basis (Bechtold and Patterson 2005). How can the climate conditions in any one year be associated with changes between observations?

Two approaches are available for this problem, 1) aggregate climate data according to the censoring interval or 2) matching of event frequency or intensity within censoring periods. The first is much simpler, and would be highly appropriate to the moving window of surveys that occurs for FIA data. When the survey year is determined randomly, nearby plots may be surveyed on different interval windows such as the first year of each decade, or the fifth year of each decade. An aggregation of climate data would provide insight into changes at the decadal scale. Care must be taken when considering how to aggregate data and these decisions must be driven by the individual data application or informed by the biology of a particular pathogen. Averages may be sufficient for some applications while event frequency or severity may be needed for others. The second approach holds potential to gather more information on the influence of inter annual climate variability on pathogen dynamics and impacts. But

technical aspects of how these data are treated in statistical models are a more difficult problem (Sun 2007). A potential approach is to break time into intervals. This is analogous to the use of covariates with temporally variable effects in survival models (Kleinbaum and Klein 1996). Survival analysis, and its component families of survival models, is a branch of statistics which deals with 'time to event' problems. Estimation of infection and mortality rates can be gained from time to event data, such as estimation of the time a tree survives with infection (analogous to mortality rate) or the time a tree remains infection free. Most survival models assume covariates have a time-invariant effect on these rates, but the effect of many covariates changes over time (Kleinbaum and Klein 1996). One approach to dealing with covariates which violate this assumption is to break the variable up into distinct time periods that have specific effects on the outcome. In this case of doubly censored data, a new covariate designating the censoring interval would articulate interval censored tree data to specific time periods (Figure 1). Caution must be taken as the metric of climate effects becomes even more important and potentially sensitive to assumptions underpinned by aggregation of climate data. Formal model synthesis and testing is needed before these tools can be applied to forest pathology data.

Data censoring issues relate back to overall goals of monitoring programs and data collection associated with field experiments. What are the goals of the analysis? What frequency of surveys can be provided by available funding? Where development of climate-disease relationships are the goal, censoring issues must be taken into account prior to initiation of the study. Ideally, a statistical framework which addresses censoring in the context of specific hypotheses would be identified prior to the initiation of any study. However, changes in funding and other unforeseen challenges almost inevitably occur over a multi-year project meaning that various cases of censoring are likely to challenge individual analysis.

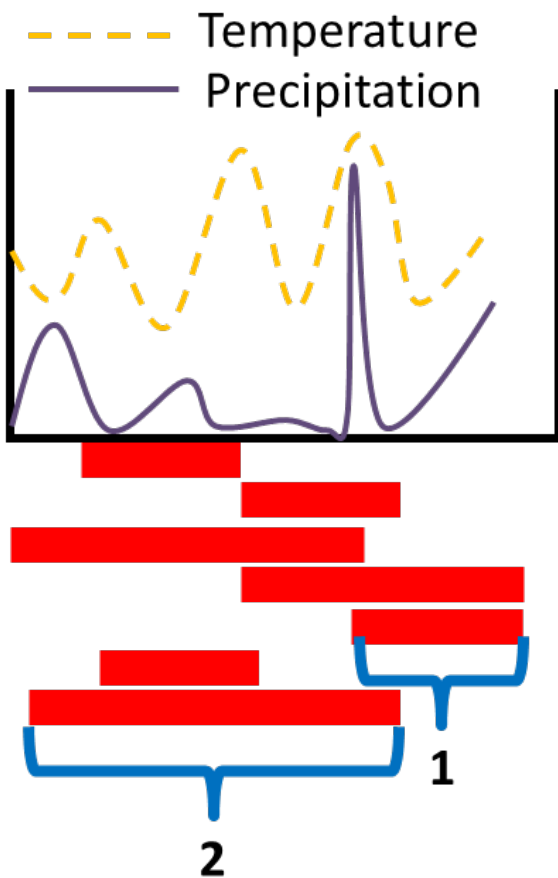


Figure 1. Examples of censoring intervals which span multiple years of distinct climate conditions. Intervals can span different lengths of time and partially overlap (compare 1 and 2).

Integrate Modelers from the Start

Modeling is not the answer. Rather, modeling is a goal oriented exercise undertaken to gain insight into a tricky problem or test a specific hypothesis (Madden *et al.* 2007, Gilligan 2008, Cunniffe *et al.* 2014). In this respect, modeling is no different than the empirical field and lab approaches of traditional Forest Pathology. Many useful models are written from existing datasets (Madden *et al.* 2007). But, where the goal is to test a set of expected disease outcomes given a dataset of observed or forecasted climate parameters, integrating modelers as part of a research team from the beginning can greatly increase the

efficiency and power of an analysis. Identifying the assumptions and limitations of a set of models preferred by an individual investigator can greatly aid what and how data are collected. Any collaboration with a modeling component should be prefaced by a conversation that identifies what each team member needs to complete a task. What data are needed to inform a model? When a model integrates processes that cannot be observed directly, what proxy measurements are appropriate substitutes and how does this change the interpretation of model results? Perhaps of greater importance is to revisit the overall objectives of a project in light of specific model limitations and assumptions to ensure that an integrated data collection and monitoring effort will be successful.

Identifying whether a modeling effort is aimed at testing existing models or making inference on datasets is an important first step. Field experiments, regardless of spatial extent, typically produce large and complex datasets where statistical models will need to account for sources of bias such as blocking and other random factors. These factors can be used to condition alternative hypotheses of the experiment and again revisit the limitations to inference as well as interpretation (Madden *et al.* 2007). Models designed to create forecasts are often built from existing datasets but assumptions about parameters is often inevitable (Meentemeyer *et al.* 2011). In this case, sensitivity analysis of a model can help identify data collection needs, particularly when these parameters represent relatively easy to measure characteristics. In contrast to statistical modeling where careful planning and data collection eases modeling at the end of a project, here models are used at the inception of a project to direct field efforts and experimental design. The most abstract modeling exercises take a much broader view and will often aim to identify conditions that lead to emergent properties such as disease or population equilibrium (Hastings 2010).

Close collaboration with modelers is also needed to collect and analyze data derived from or linked

with large-scale observation networks, such as FIA. Given the spatial and temporal scope of FIA datasets and similar networks much of the structure and original assumptions cannot be avoided. For example, double interval censoring for climate analysis is an unavoidable characteristic of many datasets including FIA. The burden of overcoming these challenges will often rest with quantitative scientists such as ecological modelers, statisticians, and basic mathematicians. Complex data analysis problems may further require these branches of quantitative sciences to work together on specific problems inherent to observational networks. However, these efforts are only likely to gain traction and usage among field ecologists and forest pathologists if they are accessible and applicable to individuals working within these fields. Mathematics and basic statistics journals have notoriously low impact factors because the topics are understood and appreciated by a relatively group of researchers. Yet, applied scientists rely on these advances to overcome specific challenges to data analysis and study design. Clearly, individual researchers who can communicate applied problems and translate quantitative solutions will play a critical role in overcoming technical limitations to analysis of disease in complex ecological systems.

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**Panel: Emerging Pathogens: Conifer
Phytophthoras**

PHYTOPHTHORAS ON CONIFERS IN THE UK – A PARADIGM SHIFT

Joan F. Webber¹

Since their emergence over the last decade, several highly invasive pathogens have emerged to threaten the survival and productivity of UK trees and forests. A significant number of these are species of *Phytophthora* and include *Phytophthora ramorum*, *P. kernoviae*, *P. pseudosyringae*, *P. lateralis* and *P. austrocedri* All, with the possible exception of *P. pseudosyringae*, appear to have been introduced probably during the past 20-25 years. Arguably the best known is *P. ramorum*, which was first found in the UK in 2002 and which continues to pose a number of challenges dictated by its quarantine status, behaviour and host range.

The initial impacts of *P. ramorum* in the UK occurred on ornamental plants in nurseries, then valuable heritage plants and broadleaved trees in public gardens. As findings were gradually made in woodlands between 2003 and 2008, the host most affected turned out to be the understory shrub *Rhododendron ponticum*, a non-native invasive that has become widespread in many woodlands particularly in western Britain. Once infected, this rhododendron species also acted as a sporulating host of *P. ramorum*, allowing the disease to spread to broadleaved hosts which then developed extensive bole cankers. However, despite the prevalence of infected rhododendron in woodlands in south west England, the number of reported tree infections remained low at <100 and largely consisted of native beech (*Fagus sylvatica*) and non-native oak species such as *Quercus cerris*, whilst native oaks such as *Q. robur* and *Q. petraea* remained largely unaffected.

But in 2009, *P. ramorum* unexpectedly spread to plantation grown Japanese larch (*Larix kaempferi*), causing increasingly heavy mortality, and

endangering other plant and trees species due to the prolific sporulation on infected larch needles (Webber *et al.*, 2010). The levels of sporulation on larch far exceeded those on rhododendron and frequently led to branch and bole cankers on other plantation grown conifers such as hemlock (*Tsuga heterophylla*), true firs (*Abies*) and Douglas fir (*Pseudotsuga menziesii*) growing in close proximity to infected larch. Rain trap data monitored in stands of naturally infected larch revealed that sporulation levels peaked in October just before and during larch needle abscission, although lab assays suggested that sporulation by *P. ramorum* can also occur on larch foliage in spring and summer. Between 2010 and the end of 2015, the combined area of affected larch in England, Scotland and Wales rose from 2,000 ha to around 20,000 ha, and not only Japanese larch but also European (*L. deciduas*) and hybrid larch (*L. x eurolepis*) have been found to be both bole and sporulating hosts.

Many millions of larch trees have been felled to curtail sporulation and limit the spread of *P. ramorum*. Widespread mortality of larch with heavy crown symptoms in 2013 was partly ascribed to the cool, wet summer and fall conditions of the previous year considered conducive to sporulation and dispersal of *P. ramorum*. However, the behaviour of the recently characterised EU2 lineage (van Poucke *et al.*, 2012) may also account for exceptionally enhanced disease levels in Scotland. The EU2 has proved to be a much more effective coloniser of larch bark than the more widespread EU1 lineage, although possibly not such a prolific sporulator (Harris, 2015). It also appears that the more aggressive colonizing ability of the EU2, compared with the EU1, may be specific to larch and not replicated in other hosts. Currently, evidence suggests that the ranges of the EU1 and EU2 lineages do not overlap but are rapidly converging in south west Scotland. This raises the likelihood of mixed lineage populations of *P. ramorum* affecting larch forests with further potential consequences.

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Apart from the challenge of controlling ramorum disease on larch, it can also be difficult to confirm infection. Since the first findings on plantation grown larch, thousands of samples have come into the Forest Research laboratories for diagnosis. If *P. ramorum* is confirmed, woodland owners must fell affected trees in line with the measures applied to *P. ramorum* as a quarantine plant pathogen, so correct identification is critical. Even with selective medium, isolation of *P. ramorum* from infected bark tissue has a relatively low success rate (15-20% depending on tissue and season), so real-time PCR diagnosis is an essential part of the process to avoid false negatives. However, occasionally larch samples with resinous lesions and bark cankers consistent with those incited by *P. ramorum* and giving a strong positive with a Phytophthora field test assay (Pocket Diagnostics®) cannot be confirmed as *P. ramorum*, either through isolation or with real-time PCR. This raised the possibility that other *Phytophthora* spp were regularly infecting larch bark at low levels that had not been detected previously.

Using end point PCR and sequencing, coupled with intense efforts at isolation, at least two other *Phytophthora* spp – *P. pseudosyringae* and *P. gonapodyides* have now been confirmed as new causal agents of larch bark cankers. The lesions caused by *P. pseudosyringae* and *P. gonapodyides* are usually smaller than those incited by *P. ramorum*, tend to occur on branches at least 2-3 m above ground level and are discrete aerial infections. This raises the possibility that they may also infect and sporulate on larch needles (as *P. ramorum* does) thereby providing the inoculum for the aerial bark infections.

Since the host jump of *P. ramorum* to larch other conifer-Phytophthora pathosystems have now become familiar in the UK including diseases caused by *P. lateralis* on Port Orford Cedar (*Chamaecyparis lawsoniana*) and *P. austrocedri* on native juniper (*Juniperus communis*). *Phytophthora austrocedri* (formerly *P. austrocerae*) is perhaps better known as the cause of a lethal disease of the native conifer species. *Austrocedrus chilensis* in southern Argentina but is

now causing mortality to juniper in more than a 100 locations in England and Scotland (Green *et al.*, 2015). Work is ongoing to compare isolates of *P. austrocedri* from Britain and Argentina and both genetic and phenotype differences are becoming apparent *Phytophthora austrocedri* is even more challenging to isolate than *P. ramorum* and illustrates many of the difficulties of diagnosing *Phytophthora* diseases of conifers.

Overall, the experience in Britain is that the diagnosis of *Phytophthora* infection in conifers can be problematic. Poor isolation success and the difficulties of DNA extraction from infected tissues that can be highly resinous and rich in inhibitory extractives have very likely contributed to this issue. It now seems likely that the frequency with which Phytophthoras infect conifers may have been underestimated in the past and the focus on broadleaved species as the most common *Phytophthora* hosts needs reconsideration. A lack of knowledge about how widespread *Phytophthora* infection of conifers can be, as well as the species involved, could help us anticipate in future the extent to which some conifers are likely to be at risk from new *Phytophthora* pathogens.

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**Panel: Needle Pathogen Outbreaks: Why
There? Why Then?**

DOTHISTROMA NEEDLE BLIGHT, WEATHER AND POSSIBLE CLIMATIC TRIGGERS FOR THE DISEASE'S RECENT EMERGENCE

Alex J. Woods¹, J. Martín-García^{2,3}, L. Bulman⁴, M. W. Vasconcelos⁵, J. Boberg⁶, N. La Porta⁷, H. Peredo⁸, G. Vergara⁹, R. Ahumada¹⁰, A. Brown¹¹ and J. J. Diez^{2,3}

ABSTRACT

Dothistroma needle blight (DNB), caused by the two fungi *Dothistroma septosporum* and *D. pini*, is a major disease of pines with a worldwide distribution. DNB has been reported from more than 63 countries, infecting over 82 different species of pine and several other non-pine species (Barnes *et al.* 2014). DNB infects needles of all ages, causing pre-mature leaf mortality and reduced photosynthetic capacity (Bradshaw 2004). *Dothistroma* spreads primarily by means of splash-dispersed asexual conidia (Gibson *et al.* 1964) which may be released and germinate any time temperatures are above 5° C and moisture is available (Sinclair *et al.* 1987).

Recognition of increases in the severity of disease in areas where DNB has long been established and notable range expansions (eg. Drenkhan and Hanso 2009) have resulted in the creation of the

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International Dothistroma Alliance (IDA) in 2006 and the subsequent EU COST Action FP1102 DIAROD (Determining Invasiveness And Risk Of Dothistroma), that now includes members from 35 countries. This report is a product of collaboration fostered by DIAROD and its aim was to assess the relationship between DNB, weather factors and climate to better understand possible underlying causes of this recent intensification in disease. A substantial body of literature shows that the life cycles of the fungi are closely related to weather factors such as precipitation and temperature (Gadgil 1967, Peterson 1973). Total accumulations of summer rainfall of more than 100 mm/month are consistently associated with DNB outbreaks (Murray and Batko 1962, Dubin 1967, Peterson 1973, Marks and Hepworth 1986) but the distribution of precipitation events is perhaps more important (Gadgil 1977). Increasing trends in summer precipitation, particularly in the Northern Hemisphere, have been linked to increased DNB activity in both Europe and North America (Brown *et al.* 2003, Woods *et al.* 2005). Optimal daytime temperatures for DNB are 18-20° C while optimal minimums are 10-12° C (Gadgil 1974). When these temperatures are combined with favourable amounts of precipitation DNB can quickly take advantage.

Given the rapid response of DNB to favourable weather conditions it seems plausible that changes in disease behaviour could be due to changes in climate. If a climate fingerprint was to be found linking a forest pathogen to global climate variability one of the best candidates would be DNB given its global distribution (Barnes *et al.* 2014), known rapid response to favourable weather conditions (Peterson 1973) and worldwide recognition as a major disease of pines over the past six decades (Bradshaw 2004). The recurrent El

Niño-Southern oscillation (ENSO) phenomenon influences patterns of temperature and precipitation in many regions of the world, often resulting in warmer and wetter conditions than normal (Zebiak *et al.* 2014). We found that since the 1950s, four of the past five strong El Niño events appear to have coincided with reports of increased DNB activity on an intercontinental scale (Figure 1).

The lack of long term standardized data records limits our ability to fully interpret this relationship but the projected future climatic conditions in the Northern Hemisphere appear to be increasingly favourable for the disease. It has been suggested that ENSO and the associated elevated climate

extremes could serve as an analogue for assessing the impacts of long-term climate change (Coakley *et al.* 1999). Perhaps any possible link to ENSO as a trigger for DNB epidemics may be being superseded by a larger climatic trend. Still, other areas of the world may become less favourable for DNB. Desprez-Loustau *et al.* (2007) hypothesised that the favourable effect of warming for DNB could be counterbalanced by the negative effect of a decrease in summer rainfall, leading to a stable or decreased impact of these pathogens by the end of the century in some regions. Further research is required to be able to accurately predict DNB outbreaks and their impact on pine forests in the future.

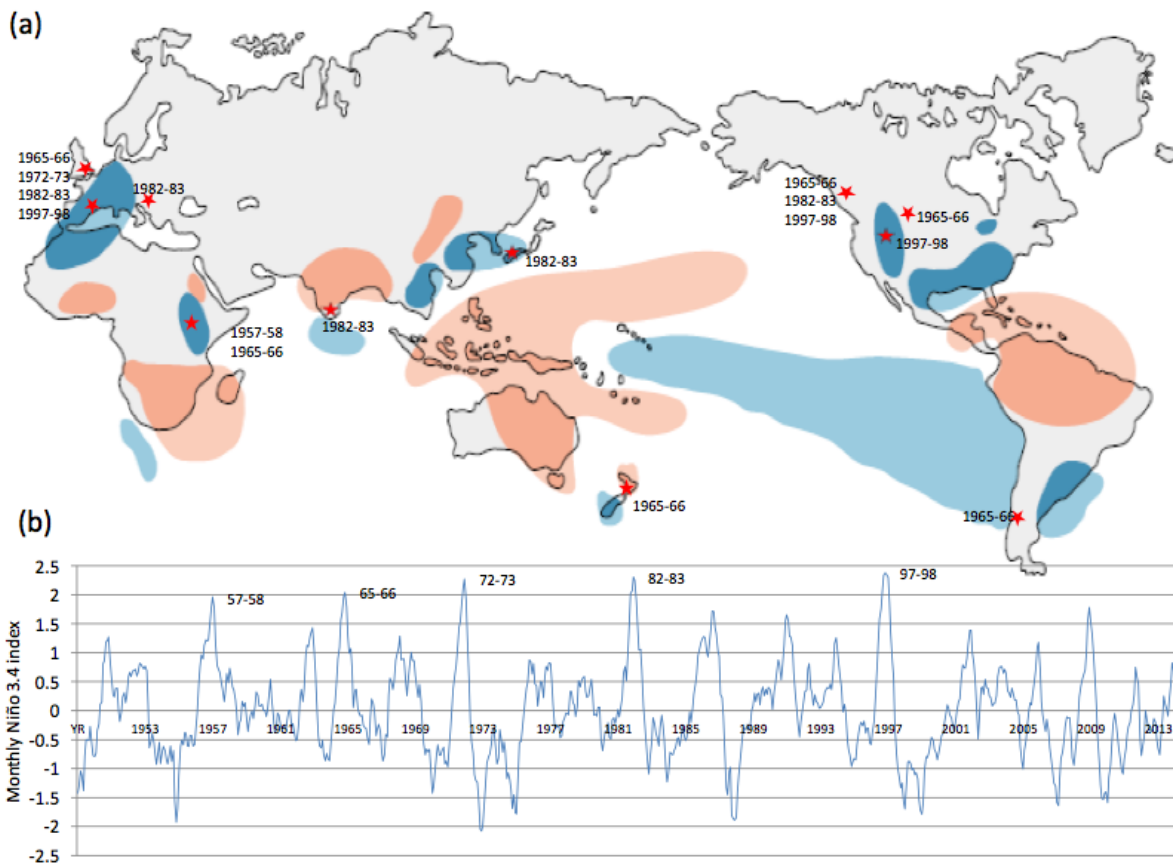


Figure 1 (a) Regions showing increased precipitation (blue) and drier conditions (orange) during El Niño events* and the dates of documented *Dothistroma needle blight* outbreaks and their general locations (red stars) that appear to coincide with the timing of strong El Niños. (b) Monthly Niño-3.4 Oceanic Niño Index (ONI) values for the period 1950-2014 with very strong El Niño events (those ≥ 2.0) identified with the two year period on which they occurred (http://www.cpc.noaa.gov/products/analysis_monitoring/ensostuff/ensoyears.shtml). *Modified with permission from Allan *et al.* (1996) and Holmgren *et al.* (2001).

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TREE-RING HISTORY OF SWISS NEEDLE CAST IMPACT ON DOUGLAS-FIR GROWTH IN WESTERN OREGON: CORRELATIONS WITH CLIMATIC VARIABLES

*E. Henry Lee*¹, *Peter A. Beedlow*¹, *Ronald S. Waschmann*¹, *Steve Cline*¹, *Michael Bollman*¹, *Charlotte Wickham*², and *Nicholas Testa*³

ABSTRACT

The fungal pathogen, *Phaeocryptopus gaeumannii*, occurs wherever Douglas-fir is found but disease damage is believed to be limited to the Coast Range and is of no concern outside the coastal fog zone (Shaw *et al.*, 2011). However, knowledge remains limited on the history and spatial distribution of Swiss Needle Cast (SNC) impacts in the Pacific Northwest (PNW). We reconstructed the history of SNC impacts on mature Douglas-fir trees based on tree ring width chronologies from the west slope of the Coast Range to the high Cascades of Oregon. Our findings show that SNC impacts on growth occur wherever Douglas-fir is found in western Oregon and is not limited to the coastal fog zone. The spatiotemporal patterns of growth impact from SNC disease were synchronous across the region, displayed periodicities of 25-30 years, strongly correlated with winter and summer temperatures and summer precipitation, and matched the patterns of enriched cellulosic stable carbon isotope indicative of physiological stress. While winter and summer temperature and summer precipitation influenced pathogen dynamics at all sites, the primary climatic factor of these three limiting factors varied spatially by location, topography, and elevation. In the 20th century, SNC impacts at low- to mid-elevations were least severe during the warm phase of the Pacific Decadal Oscillation (PDO, 1924-1945) and most severe in 1984-1986, following the cool phase of the PDO

(1945-1977). At high elevations on the west slope of the Cascade Mountains, SNC impacts were the greatest in the 1990s and 2000s, a period of warmer winter temperatures associated with climate change. Warmer winters will likely continue to increase SNC severity at higher elevations, north along the coast from northern Oregon to British Columbia, and inland where low winter temperatures currently limit growth of the pathogen. Surprisingly, tree-ring records of ancient Douglas-fir logs dated ~53K radioactive years B.P. from Eddyville, Oregon displayed 7.5- and 20-year periodicities of low growth, similar to those found in modern day coastal Douglas-fir tree-ring records which we interpret as being due to cyclic fluctuations in SNC severity. Our findings indicate that SNC has persisted for as long as its host, and as a result of changing climate, may become a significant forest health problem in areas of the PNW beyond the coastal fog zone.

INTRODUCTION

Swiss needle cast (SNC) is an economically important disease of most forms of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco, *P. menziesii* var. *glauca* [Beissn.] Franco, and *Pseudotsuga macrocarpa* (Vasey) Mayr) (Boyce 1940; Gadgil 2005). SNC is caused by the fungus *Phaeocryptopus gaeumannii* (Rhode) Petrak and occurs wherever its host is found, but historically has been of minor importance in western North American forests (Boyce 1940; Hansen *et al.* 2000). Epidemic outbreaks of SNC have been reported in coastal Oregon, Washington, and British Columbia and have steadily increased in severity since ~1984 (Hansen *et al.* 2000; Omdal and Ramsey-Kroll 2010; Black *et al.*, 2010; Shaw *et al.* 2011). Disease is most severe in forests and

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plantations on the western slopes of the Oregon Coast range within the coastal fog zone (Hansen *et al.*, 2000). The affected area with visible SNC symptoms — chlorosis and premature needle loss — seen from annual aerial surveys of coastal Oregon have set new record highs each of the last six years (Kanaskie *et al.*, 2015). Evidence suggests that SNC is affected by climate (Manter *et al.* 2005; Stone *et al.* 2008b), alone or in combination with forestry practices of the (later) 20th century. Possible causes for the current increase in SNC severity include: climate warming and the introduction of Christmas tree plantations in the mid-1970s interacting with high soil nitrogen (Hadfield and Douglass 1982; Michaels and Chastagner 1984), or genetic changes in the pathogen (Winton *et al.* 2006). There is mounting concern that SNC is increasing in severity, frequency and range in association with rising winter temperatures (and spring precipitation) and will continue to intensify over the 21st century due to climate change (Zhao *et al.* 2011; Watt *et al.* 2011).

While the epidemiology of SNC and mechanisms of pathogenicity of *P. gaeumannii* on Douglas-fir have been well studied in young plantations, knowledge remains limited on the history and spatial distribution of SNC impacts on mature and older tree growth in the Pacific Northwest (PNW). *P. gaeumannii* is indigenous in western North America and has long been believed to have been pervasive but innocuous in Douglas-fir forests prior to 1950 (Boyce 1940; Peace 1962; Hood 1982). Increased severity since ~1950 is thought to be at least in part, climate mediated because the causal fungus is sensitive to small differences in temperature and moisture (Manter *et al.* 2005; Stone *et al.* 2008b; Black *et al.*, 2010).

A recent dendrochronological study indicates that SNC has affected Douglas-fir growth at least back to 1592, which was the earliest of the available tree-ring records (Lee *et al.*, 2013). Furthermore, the spatiotemporal patterns of decreased annual ring width associated with SNC are synchronous

across six coastal sites in Oregon representing a latitudinal transect at varying elevations, display periodicities of 25-30 years, and are strongly correlated with winter and summer temperatures and summer precipitation. SNC impacts as measured by tree ring width in coastal Oregon peaked in 1984-1986 —thought to be a period when the fungal population reached epidemic levels following several decades of environmental conditions favorable to growth and reproduction of *P. gaeumannii* (Lee *et al.*, 2013).

Growth reduction of Douglas-fir due to SNC in the PNW is symptomatic in the Coast Range of Oregon and Washington, primarily, and is of limited concern outside this region (Shaw *et al.*, 2011). There has been no reported evidence of SNC impacts on growth of inland Douglas-fir although symptoms are often noted in plantations in Northern Idaho and Western Montana (Hagle *et al.*, 2003). A broad scale study involving 59 young Douglas-fir stands (10-23 years) found no growth reductions in the Oregon Cascades during a SNC outbreak between 2001 and 2006 (Filip *et al.*, 2007). To date, there have been no dendrochronological studies to reconstruct the history of SNC impacts on Douglas-fir growth in mature forest stands outside of the Coast Range of Oregon and Washington.

Here, we extend the dendrochronological findings of Lee *et al.* (2013) to examine the history and spatial extent of the cyclical pattern of SNC outbreaks in association with the seasonal climate factors and address the three questions in the panel titled, “Where? Why there? Why now?” We conducted a dendrochronological study to test the following hypotheses:

- (1) *P. gaeumannii* is an ancient tree pathogen that affects Douglas-fir growth as far back as its earliest known existence in the Pacific Northwest;
- (2) SNC is ubiquitous and affects Douglas-fir growth across western Oregon from the Coast Range to the Cascade Range;
- (3) SNC is sensitive to winter and summer temperature, and summer precipitation, and so,

spatial variability in SNC severity can be attributed to variations in site conditions and location. We developed new master chronologies of ancient and modern Douglas-fir ring width from the west slopes of the Coast Range to the west slopes of the Cascade Range of Oregon. We examined the spatial distribution of SNC impacts on mature Douglas-fir trees using time series intervention analysis of intra-annual tree ring width chronologies to reconstruct the history of SNC impacts by site.

The Disease Cycle

The key growth pattern in tree-ring records of coastal Douglas-fir is a sinusoidal cycle of anomalously low growth having a primary periodicity of ~25-30 years and a harmonic periodicity of ~4 years associated with SNC (Lee *et al.*, 2013). The cyclical patterns of SNC impact on Douglas-fir growth occur throughout the life of the tree and because of the effects synoptic seasonal weather patterns on fungal growth, are synchronous across coastal Oregon. We combined our dendrochronological findings with the epidemiology of SNC to develop a conceptual model of the disease cycle driven by needle retention and fungal fruiting body abundance which have routinely been used as indices of disease severity (Hood 1982; Michaels and Chastagner 1984; Hansen *et al.* 2000; Manter *et al.* 2005). SNC reduces assimilation of carbon and tree diameter by stomatal occlusion and early needle abscission (Manter *et al.* 2000; Hansen *et al.* 2000).

Consequently, yearly changes in SNC impacts depend upon inoculum abundance, ascospore germination, and pathogen colonization in association with climatic conditions which affect the proportion of stomata occluded and needle retention Douglas-fir trees on the coast typically retain up to four years of needles but may only have current and 1-year-old foliage due to premature needle abscission in severely affected plantations (Hansen *et al.* 2000; Maguire *et al.* 2002; Zhao *et al.* 2011).

In our conceptual model, the disease cycle begins when pathogen abundance is at epidemic levels, resulting in loss of 2-year-old and older needles and a significant reduction in stem growth (Figure 1). The pathogen population will be reduced due to premature needle abscission resulting in fewer infected needles and a reduction in inoculum. Peak SNC outbreaks reduce tree growth for several consecutive years because photosynthetic capacity is restored to normal only after all needle classes have formed (Saffell *et al.*, 2014).

A delay of several years between inoculation and growth of the fungus and tree growth reduction is expected because the pathogen infects only the newly emerged needles (Hood and Kershaw 1975; Stone *et al.* 2008b). This lagged growth response to SNC is represented by a 4-year periodicity in disease impacts (Figure 1). The slow buildup of pathogen abundance from endemic to epidemic levels over several generations is represented by a 20-year periodicity. The dominant periodicity of 20 years varies by site and is as low as 6 years at Tillamook where more favorable climatic conditions allow the fungus to develop faster (Stone *et al.* 2008b; Lee *et al.*, 2013).

Pseudothecia can be commonly found on 4 to 7-year-old needles in the Cascade Range of Oregon and Washington, and on 1 to 2-year-old needles in some areas of the Coast Range where pathogen dynamics are enhanced by more favorable climatic conditions (Stone *et al.* 2008b). Pathogen abundance is not reset to endemic levels by abscission of 2-year-old and older needles in areas where disease is constantly severe as indicated by a <10-year disease cycle and the presence of pseudothecia on 1 to 2-year-old needles.

Epidemiology of SNC and Climate Relations

Three major phases of the infection cycle of *P. gaumannii* are relevant to the climate-growth relation (Manter *et al.* 2005): (1) the fungus reproduces only sexually and pseudothecia (i.e., fruiting bodies) develop in winter and can begin

plugging stomata as early as December; (2) sporulation and initial infection of needles occur from May to July; and (3) needle colonization by internal hyphal growth occurs year round following initial infection (Figure 2). Wet needles in late spring and early summer are necessary for spore dispersal and initial infection via the stomata (Capitano 1999; Stone *et al.* 2008b). Mild winters, spring precipitation, and moderate summer temperatures at a coastal site on west slopes of the Coast Range are highly favorable conditions for *P. gaeumannii* (Figure 2A). While precipitation is steadily decreasing during sporulation in the

summer, fog frequency is steadily increasing (Figure 2B). Needle wetness is maintained by coastal fog in late summer and is less a limiting factor of fungal development along the coast (Figure 3). SNC impacts on Douglas-fir growth are most severe along the coast where winter daily maximum temperatures are above 7°C (Stone *et al.*, 2008b), summer temperatures range between the temperature optima for germination (18°C) and growth (22°C) (Capitano, 1999), and summer needle wetness is adequate for fungal colonization of needles.

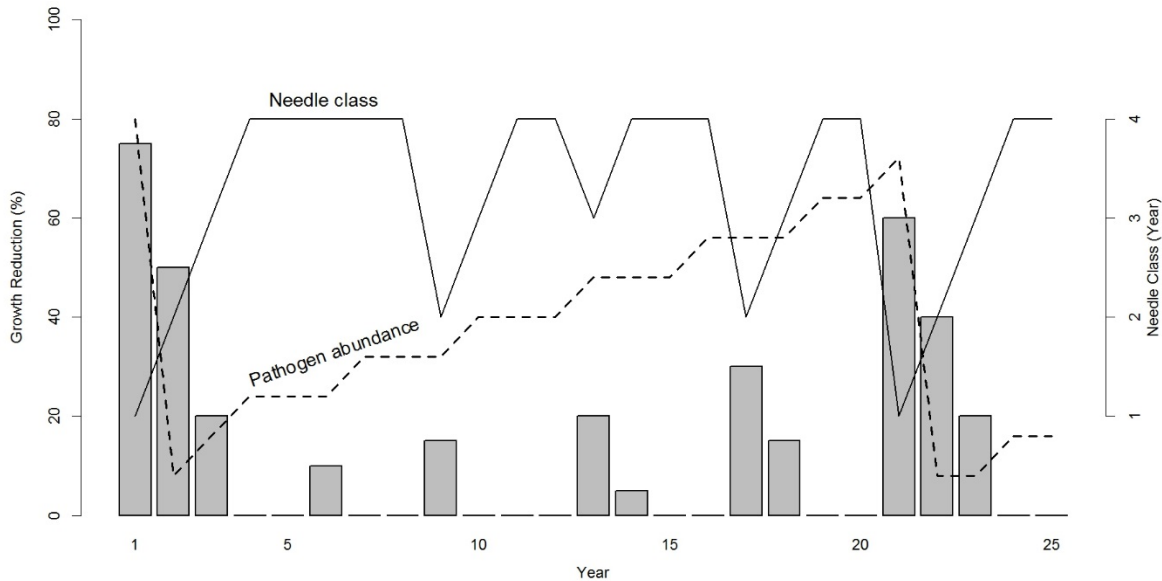


Figure 1. Conceptual model of Swiss needle cast (SNC) impact on tree growth in association with the abundance of *Phaeocryptopus gaeumannii* and number of needle classes retained (Lee *et al.*, 2013). The number of needle classes retained varies from one (when the tree is heavily infected) to four (least infected). Pathogen abundance increases from endemic (when two-year-old and older needles are abscised) to epidemic levels (when tree is heavily infected) over several decades. The disease cycle begins anew with a peak reduction in growth when pathogen abundance reaches epidemic levels and is then reset to endemic levels following the early abscission of two-year-old and older needles. Growth reductions display 4- and ~20-year periodicities because *P. gaeumannii* infects only the newly emerged needles at time of sporulation and has a four-year life cycle.

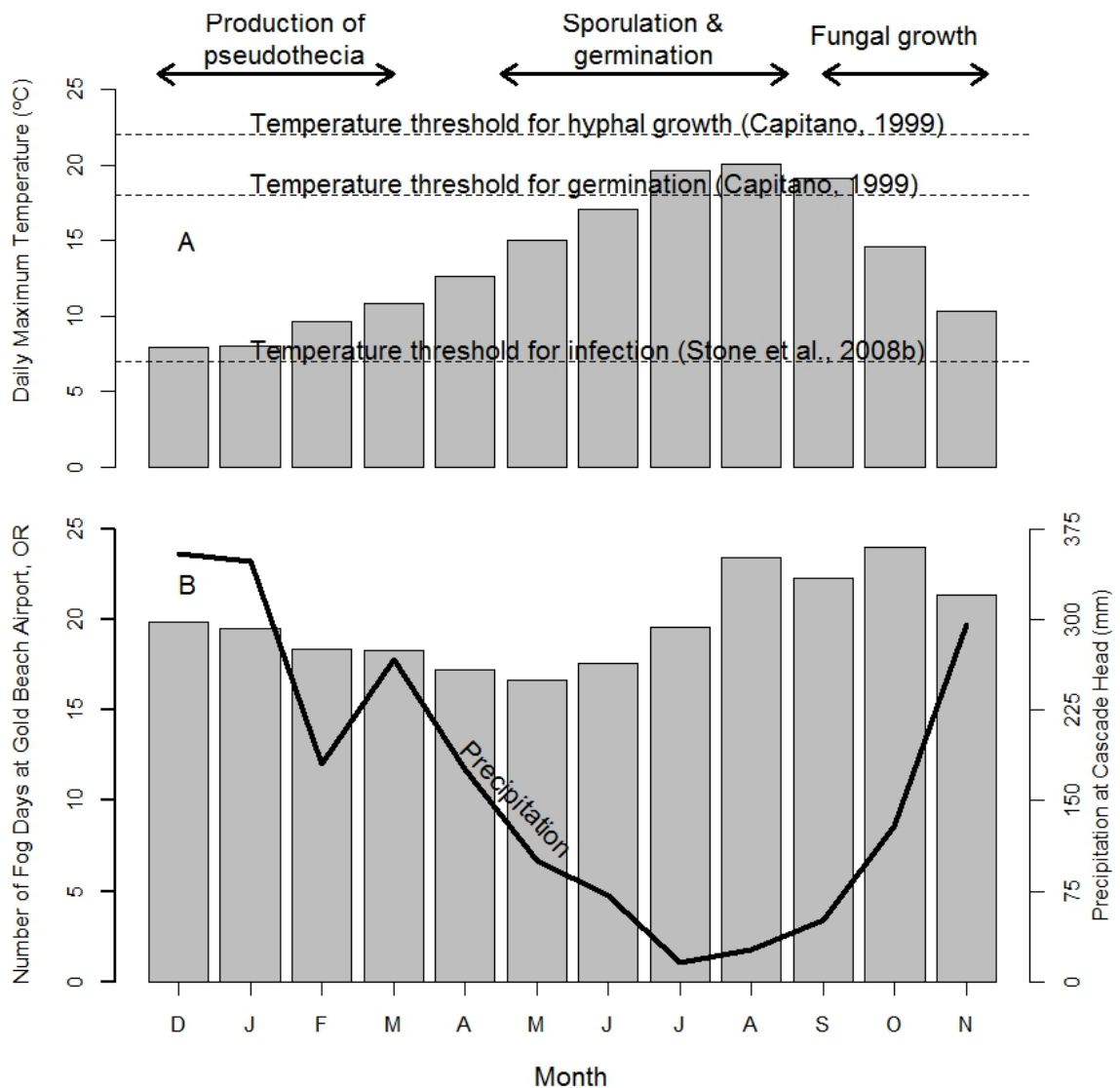


Figure 2. Seasonal pattern of (A) temperature, (B) precipitation and fog frequency at Cascade Head on west slopes of Coast Range of Oregon in relation to the three developmental stages of *Phaeocryptopus gaeumannii*. The climatic factors limiting pathogen dynamics are winter (November-February) and summer (June-July) temperatures and summer (June-July, primarily July) needle wetness.

Reconstruction of SNC Impacts on Douglas-Fir Growth in Coastal Oregon

We analyzed tree-ring chronologies from six late-successional Douglas-fir stands in the western Oregon Coast Range using Time Series Intervention Analysis (TSIA) to address how climate relates to the impact of SNC on tree growth

(Figure 4) (Lee *et al.*, 2013). Tree-ring chronologies of western hemlock (*Tsuga heterophylla*), a species not susceptible to the fungus *Phaeocryptopus gaeumannii*, and Douglas-fir at Soapgrass Mountain, a high Cascades site, were used as a climate proxy in the TSIA. We found that growth reductions associated with SNC dated back to the 1590s, the earliest record in our

dendrochronological data (Figure 5). Growth reductions were synchronous across the six sites indicating that the disease severity was influenced by regional climatic conditions. SNC impact peaked in 1984-1986 at all six study sites, followed by unprecedented disease impacts of 100% in 1996 and 2004 at one site, while decreasing to previous levels at the other five sites. SNC impacts displayed cyclical patterns having periodicities of 6, 12, and 25-30 years which were coherent across the region and represented the disease cycle unique to SNC (Figure 5). The synchronization of SNC impact on Douglas-fir across the landscape indicated that there were climate factors, which favored disease conditions at these sites in coastal Oregon.

SNC Impacts on Ancient Douglas-Fir

We analyzed tree-ring chronologies from two of 11 Douglas-fir logs that were unearthed in 2008-2010 by the Oregon Department of Transportation along the U.S. Highway 20 reconstruction site due east of Eddyville, Oregon (N44°39', W123°47'). The logs, needles, and seed cones were encased in ancient landslide deposits at 26 m below the surface and were remarkably preserved. Radiocarbon dating estimates ages ~53K Before Present (BP) in the Marine Isotope Stage 3 (MIS3, ca. 60 to 27 K BP) period which was generally cold but with intermittent Dansgaard-Oeschger warm phases (Panyushkina *et al.*, 2012). Stem diameters range from 64 cm for the 89 year-old log (351) to 128 cm for the 233 year-old log (355) and are comparable in size to contemporary coastal Douglas-fir trees of the same age. The similar growth rates indicate that the ancient Douglas-fir come from a temperate rainforest environment comparable to present day. Needles of the ancient Douglas-fir appear to have significant stomatal occlusion by structures resembling pseudothecia of *P. gaeumannii*, as seen under a scanning electron microscope (Figure 6). The two tree-ring series were successfully cross-dated but diverged and displayed periodicities of either 7.5 or 20 years (Figure 7), indicative of a non-climatic forest disturbance agent that affected

one tree (351) more than the other (355). The chronology of tree 351 displays several 3-year periods of growth reduction approximately every 7.5 years. The 7.5-year disturbance cycle is similar to that of Tillamook Lower which has a periodicity of 6 years caused by SNC (Figure 8). The 20-year disturbance cycle of tree 355 is similar to the 25-year SNC cycle at Horse Creek Trail Lower in the Siuslaw National Forest (not shown). We attribute the 7.5 and 20 year disturbance cycles of the ancient Douglas-fir to SNC because the periodicities of low growth are similar to those of the SNC disease cycles of contemporary Douglas-fir and the stomata are possibly occluded. Furthermore, the anomalously low growth years of tree 351 are not synchronous with those of tree 355, indicating a non-climatic stress on individual trees rather than a climatic stress on all trees.

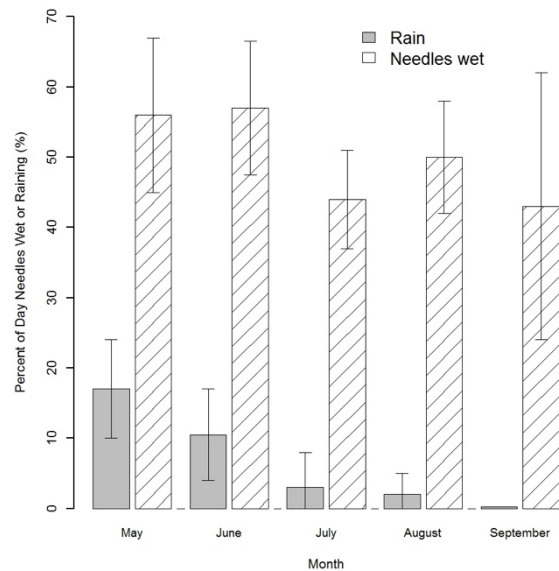


Figure 3. Percent of day when precipitation occurs versus when needles are wet in the summer of 2014 at Cascade Head. Needle wetness is maintained by coastal fog during the annual summer drought period.

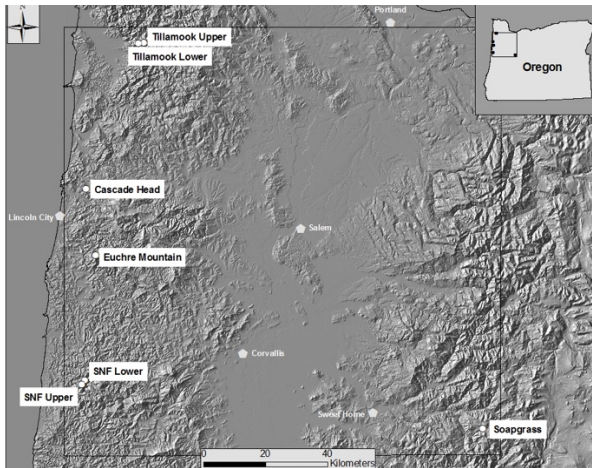


Figure 4. Locations of six study sites in Oregon Coast Range (Lee et al., 2013). One reference Douglas-fir site, Soapgrass Mountain, is located at about 1200 m elevation on the western slope of the Cascade Range of Oregon.

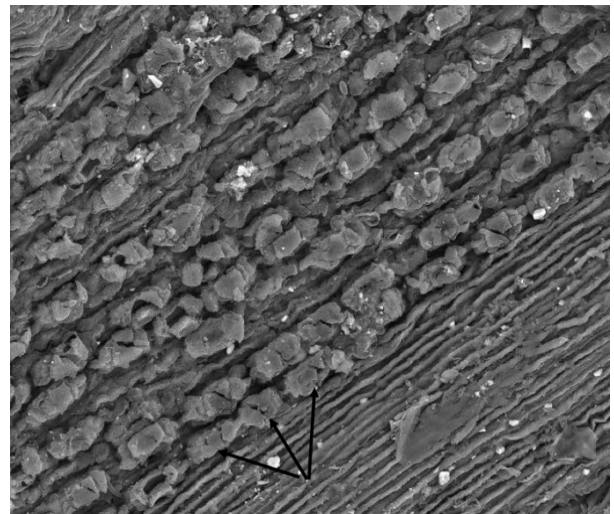


Figure 6. Scanning electron micrograph of pseudothecia primordia (see arrows) blocking the stomata of an ancient Douglas-fir needle found 29 m below the surface in landslide deposits near an Oregon Department of Transportation highway reconstruction project by Eddyville, Oregon. Image courtesy of William Rugh.

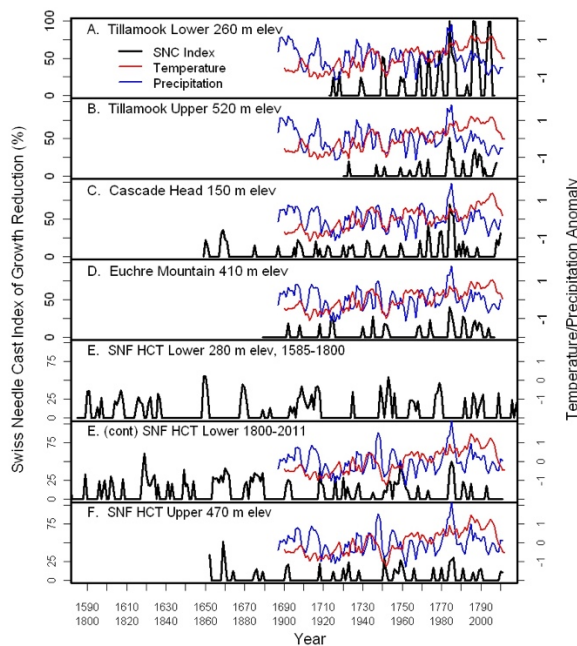


Figure 5. The percent reduction in annual tree-ring width increment of Douglas-fir adjusted for temperature and a climate proxy at six study sites in coastal Oregon (Lee et al., 2013). Temperature and precipitation were normalized to a mean of 0 and a variance of 1. The red line is the 5-year running average of mean daily maximum temperature for January and February. The blue line is the 3-year running average of total precipitation for June and July.

SNC Impacts Douglas-Fir Inland

We examined the spatial distribution of SNC impacts on mature Douglas-fir trees using TSIA of earlywood (EW) and latewood (LW) ring width chronologies from the west slope of the Coast Range to mid- and high-elevations on the west slope of the Cascade Mountains of Oregon (Figure 9). The EW and LW series represent a seasonal time series with a mean response function that contains components for climate and SNC outbreaks. The spatially-explicit predicted growth response to temperature and water was used as a climate proxy and was subtracted from the master chronology to isolate the disease signal. All sampled stands experienced significant radial growth reductions in Douglas-fir that could not be accounted for by current and previous-year seasonal climatic factors. The spatiotemporal patterns of growth reduction attributable to SNC were synchronous across the region, displayed periodicities of 25-30 years, and were strongly correlated with winter and summer temperatures and summer precipitation. Our findings indicate that detectable SNC impacts occur wherever Douglas-fir is found in western Oregon and is not limited to the coastal fog zone.

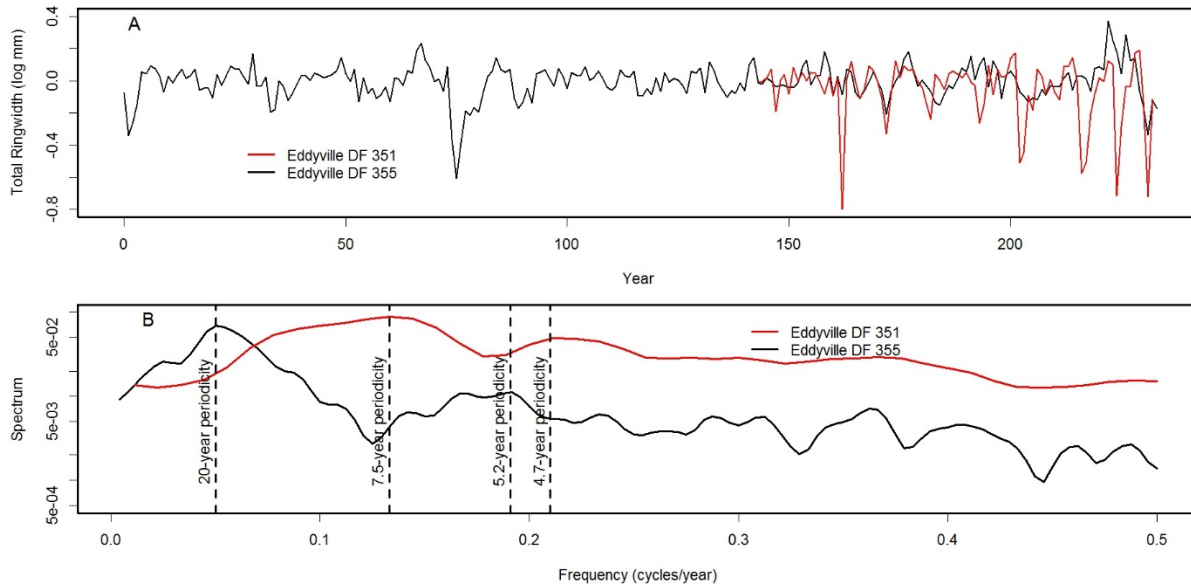


Figure 7. (A) Master chronologies of two ancient Douglas-fir trees from Eddyville, Oregon and (B) comparison of their spectrum. The chronology of tree 351 diverges from that of tree 355 and displays a cyclical pattern of anomalously low growth having a primary periodicity of 7.5 years and secondary periodicity of 4.7 years. Tree 355 displays a cyclical pattern having a primary periodicity of 20 years and a secondary periodicity of 5.2 years.

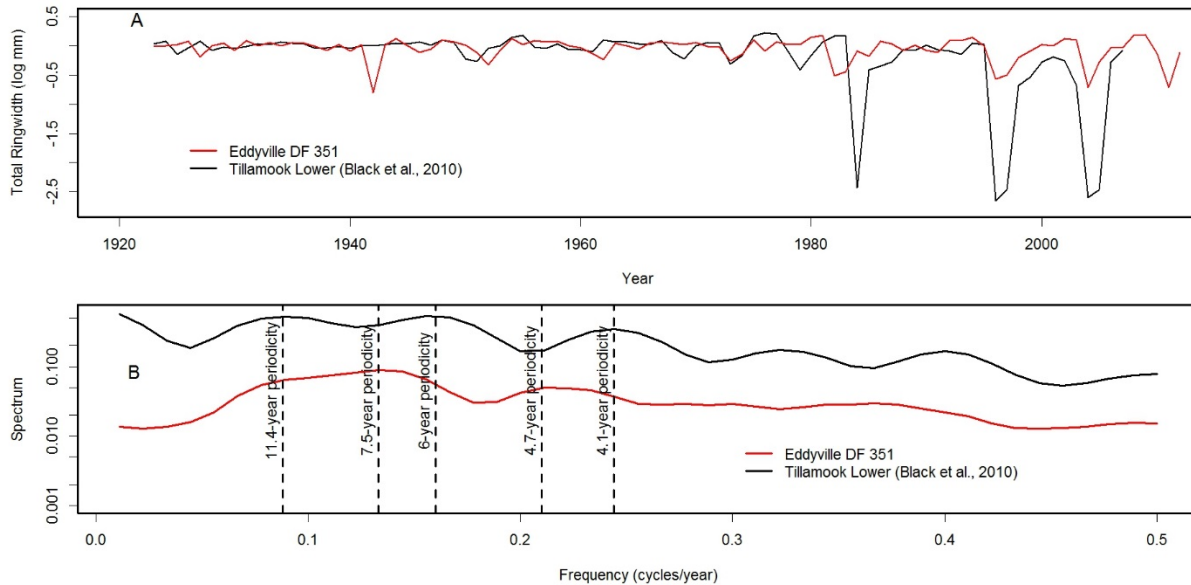


Figure 8. (A) The modern day analog of the ancient Douglas-fir tree 351 from Eddyville, Oregon is the master chronology of Douglas-fir at Tillamook Lower which displays cyclical patterns of anomalously low growth attributed to Swiss Needle Cast (Black et al., 2010). (B) The disease cycles at Tillamook and Eddyville have a similar primary periodicity of 6 and 7.5 years, respectively, and a secondary periodicity of 4.1 and 4.7 years, respectively. The disease cycle at Tillamook has another primary periodicity of 11.4 years which is less pronounced than at Eddyville.

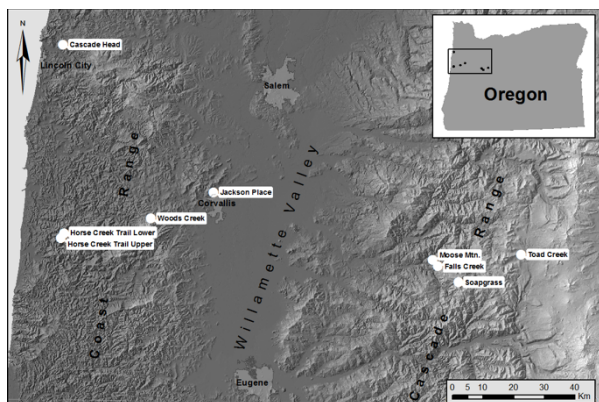


Figure 9. Tree core samples were collected from nine field sites located in mature Douglas-fir stands on the west and east sides of the Coast Range, in the Willamette Valley, and on the west side of the Cascade Mountains.

Climate Relations with SNC

To infer the climate relations with SNC, we used TSIA to classify each year into one of three disease states, SNC growth suppression, no suppression, and release. The dominant pattern of Douglas-fir growth at each site was a disease cycle with a primary periodicity of 25-30 years and secondary periodicity of ~4 years (Figures 5, 7, 8, and 10) that is the interaction of climatic and non-climatic factors (Figure 1). We hypothesize that changes in pathogen abundance, amount of inoculum, needle class retention, stomatal occlusion, and climate over one or more decades cause the cycling of disease states throughout the life of the tree.

To determine the climate relations with SNC, it was necessary to isolate the climate effects which were confounded with the biotic effects. The years classified as no SNC suppression or release have a SNC index value of 0% and do not correlate with climate. Consequently, the climate relations with SNC were determined using only the years classified as SNC suppression which have a negative pulse intervention resulting in a positive SNC index value. According to the conceptual disease cycle model, growth response to SNC was lagged and was the culmination of the interaction of climatic and biotic factors over multiple

decades. Rather than correlating the nonzero SNC index with seasonal temperature and precipitation in the current and each previous year, we calculated the canonical correlations of SNC index with temperature and precipitation for the current and previous 30 years by site (Lee *et al.*, 2013).

For Cascade Head, the SNC index of impact on latewood growth correlated best with the canonical variables for June-July temperature ($r_{can}=-0.98$) and June-July dewpoint deficit ($r_{can}=-0.98$) (Figure 11). The multiple regression equation of SNC impact on climate was

$$\text{SNC index} = 815 - 47.1 \text{ JJ_Temp} + 4.3 \text{ DJF_Temp} + 0.19 * \text{JJ_Prec}$$

and accounted for 96% of the variation where JJ_Temp= June-July mean daily maximum air temperature (°C), DJF_Temp= December-February mean daily maximum air temperature (°C), and JJ_Prec= June-July total precipitation (mm). The key explanatory variable was June-July temperature based on Kruskal's measure of relative importance, indicating that high summer temperatures reduce the SNC impacts on Douglas-fir growth at this coastal site. While summer temperature and precipitation were correlated, identification of the key climatic factors associated with SNC was possible because the long history of SNC impacts represented a century that had high climatic variability as well as high variability in the tree-ring records.

For the high Cascades site, Soapgrass Mountain, the SNC index correlated best with the canonical variables for June-July precipitation ($r_{can}=-0.91$) and February-April temperature ($r_{can}=0.88$) (Figure 12). The multiple regression equation of SNC impact on climate was

$$\text{SNC index} = -30 - 9.2 \text{ JJ_Temp} + 26 \text{ FMA_Temp} + 2.5 * \text{JJ_Prec}$$

and accounted for 95% of the variation where JJ_Temp= June-July mean daily maximum air

temperature (°C), FMA_Temp= February-April mean daily maximum air temperature (°C), and JJ_Prec= June-July total precipitation (mm). The key explanatory variable was February-April temperature based on Kruskal's measure of relative importance. The multiple regression results differed from the canonical correlation results because correlation considers one factor at a time whereas regression considers all factors simultaneously. Note also that the winter months of importance at Soapgrass were different than those at Cascade Head because temperatures in the two coldest months, December and January, were below the growth threshold in the high Cascades. Consequently, pseudothecia of *P. gaeumannii* likely formed one to two months later due to the colder environment.

Winter temperature, summer temperature and precipitation are the key limiting factors of pathogen abundance at all sites but, of these three climate factors, the primary limiting factor varies by site conditions and location (Figure 13). Summer precipitation is most limiting in warm, dry environments in the Willamette Valley and in some coastal sites in southern Oregon where summer needle wetness is less maintained by coastal fog. Winter temperature is most limiting in cool environments on the east slopes of the Coast Range and above the snowline on the west slopes of the Cascade Mountains. Summer temperature is most limiting at one coast site that lies more within the coastal fog zone.

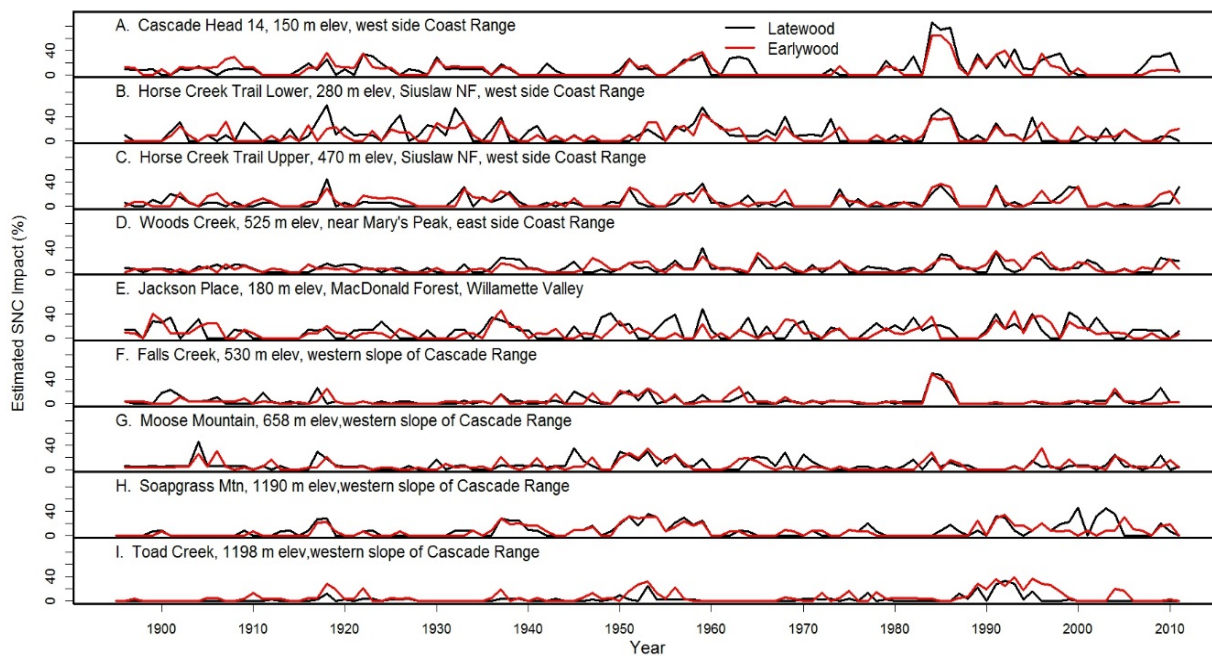


Figure 10. The percent reduction in earlywood and latewood ringwidth increment of Douglas-fir attributed to Swiss Needle Cast at nine study sites from the west slopes of the Coast Range to the west slopes of the Cascade Mountains of Oregon. The growth anomalies could not be explained by seasonal climate variables for temperature and water. Peak SNC impacts occurred in 1918, 1959, and 1984-1986 approximately 25-41 years apart and were synchronous across the region.

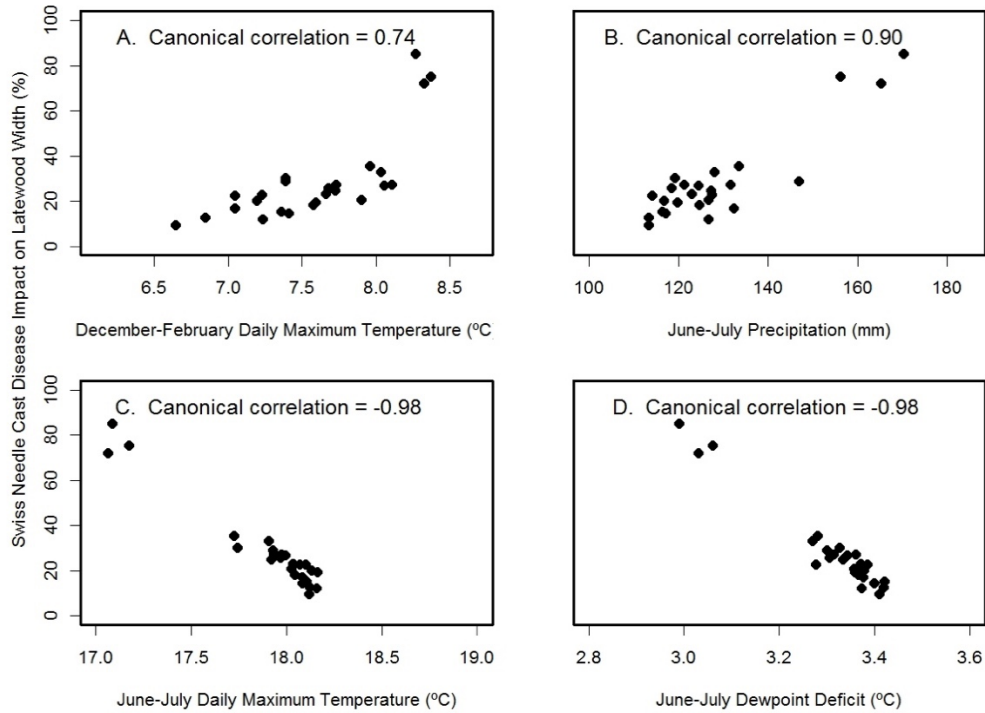


Figure 11. Canonical correlation of SNC index of impact on latewood growth with (A) winter temperature, (B) summer precipitation, (C) summer temperature, and (D) summer dewpoint deficit at Cascade Head. Temperature and precipitation were summarized on a seasonal basis so as to maximize the canonical correlations with the SNC index.

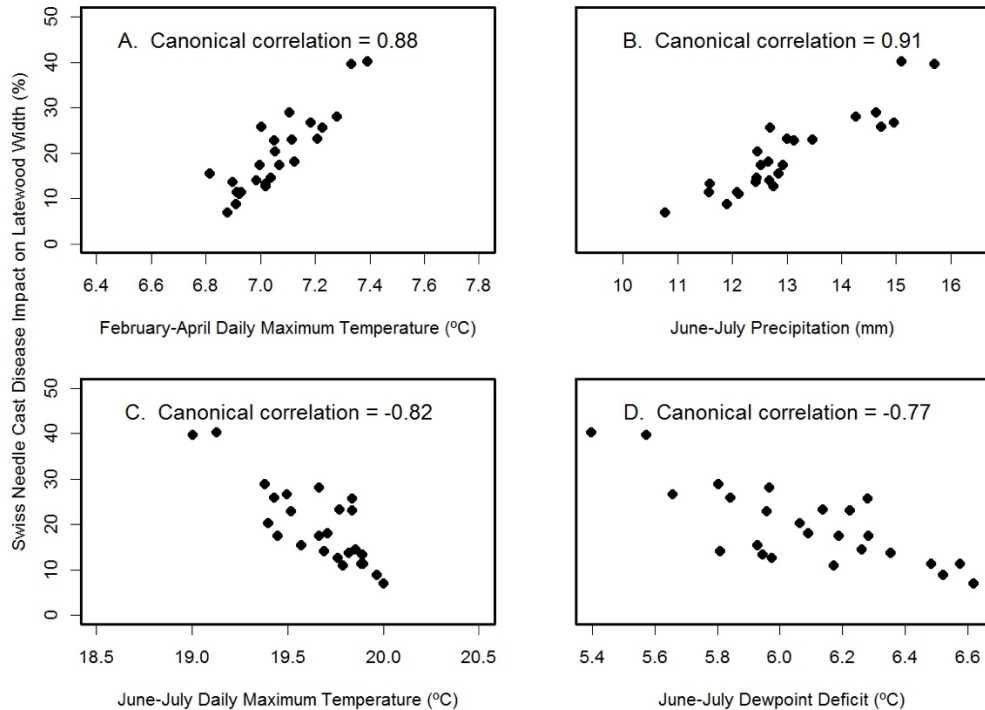


Figure 12. Canonical correlation of SNC index of impact on latewood growth with (A) winter temperature, (B) summer precipitation, (C) summer temperature, and (D) summer dewpoint deficit at Soapgrass Mountain. Temperature and precipitation were summarized on a seasonal basis so as to maximize the canonical correlations with the SNC index.

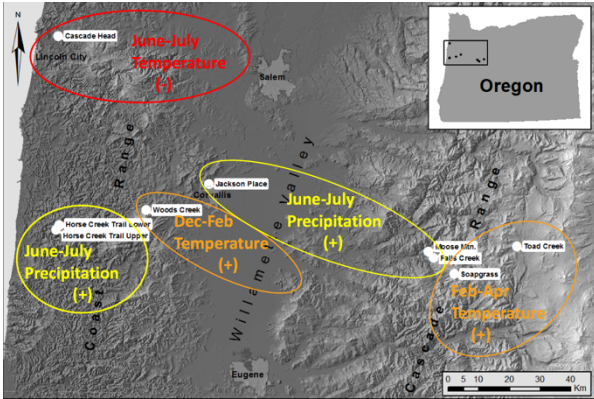


Figure 13. Map of study sites indicating the primary climatic factor that is most limiting to fungal development at each site based on time series intervention analysis of dendrochronological data.

SNC in Influences by the Pacific Decadal Oscillation (PDO)

In the 20th century, the PNW has experienced high climatic variability including a strong warm phase of the PDO (1925-1946), followed by a strong cool phase (1947-1978). We found an inverse relation between PDO and SNC effects on tree-ring width, i.e., reduced effects during the warm phase and increased effects during the cool phase (Figure 14).

The period from 1917 to 1940 was exceptionally warm and dry and the drought of the 1930s was the second most severe drought of the last 250 years (Gedalof *et al.*, 2004), likely resulting in less SNC impact on tree growth (Figure 14). This was followed by several wet periods from 1941 to 1955 and from 1968 to 1984, likely resulting in greater SNC impact and culminating with the 30-year peak impact in 1984-1986. The linkage between cool PDO phases and increased SNC impact continued after 1984 as evidenced by the intensification of SNC impacts on the east side of the Coast Range and in the high Cascades in recent decades during a mostly cool PDO phase (1998-2014) (Figure 14). However, the positive trend in SNC impacts at two sites (Woods Creek, Soapgrass Mountain) was more influenced by increasing winter temperatures due to climate warming than by summer conditions

which were more favorable to fungal development in part due to the cool PDO phase.

CONCLUSIONS

SNC impacts occur wherever Douglas-fir is found and are synchronous across western Oregon, indicating that SNC is influenced by regional climate. SNC impacts in the PNW date back to ~53K radioactive years BP as evidenced by the cyclical patterns of low growth in the master chronologies of ancient Douglas-fir that match the modern SNC disease cycles at coastal sites, and supported by the presence in the ancient needles of putative pseudothecia of *P. gaemannii*. This long history of SNC predates forest management practices and improves our understanding of the climate factors affecting the causal fungus SNC impacts on Douglas-fir growth as seen in tree-rings display 6 to 30 year periodicities throughout the life of the tree. The higher frequencies in the disease cycle represent a lagged growth response to SNC caused by the infection of only the newly emerged needles at time of sporulation, followed by colonization of the needle over several years which is unique to *P. gaemannii*. With warmer winters, SNC impacts are increasing in mature closed-canopy Douglas-fir stands on the east slopes of the Coast Range and in the high Cascades. Temperatures will likely continue to increase due to climate change, and consequently, SNC is expected to intensify in frequency and magnitude at higher elevations, north along the coast from northern Oregon to British Columbia, and inland where current winter temperatures limit fungal growth.

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Environmental Effects Research Laboratory's Western Ecology Division and approved for publication. Approval does not signify that the contents reflect the views of the Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

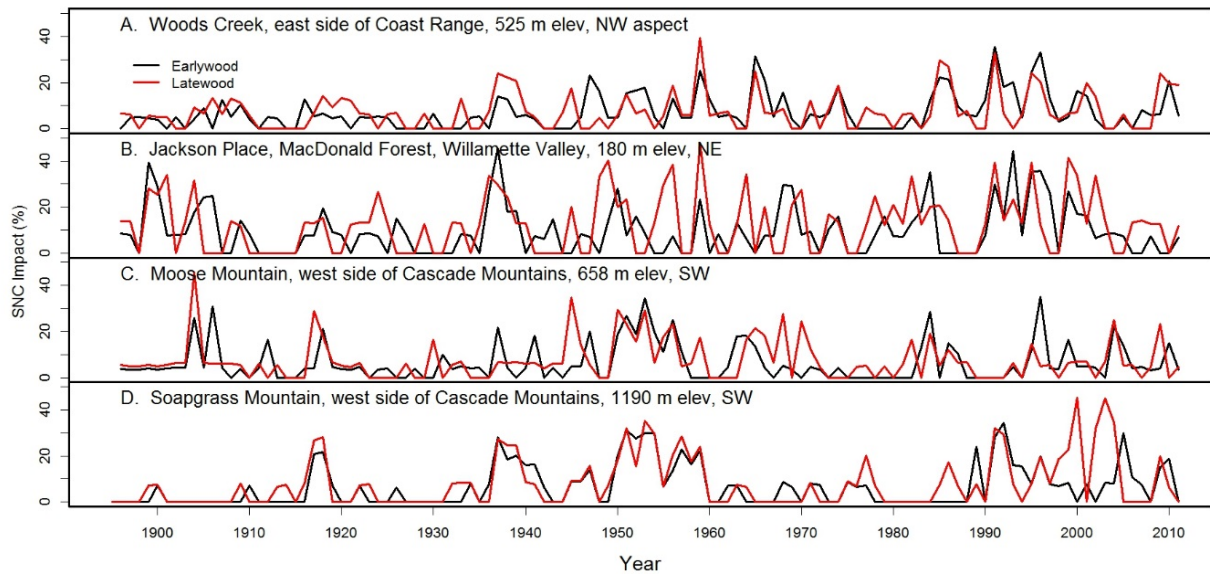


Figure 14. Swiss needle cast (SNC) index of impact at four inland sites display disease cycles having a 25-30-year periodicity, a relationship with the Pacific Decadal Oscillation (PDO), as well as an increasing trend at two sites, Woods Creek and Soapgrass Mountain. SNC impacts were less frequent and severe between 1925 and 1946 during a strong warm phase of the PDO than between 1947 and 1978 during a strong cold PDO phase.

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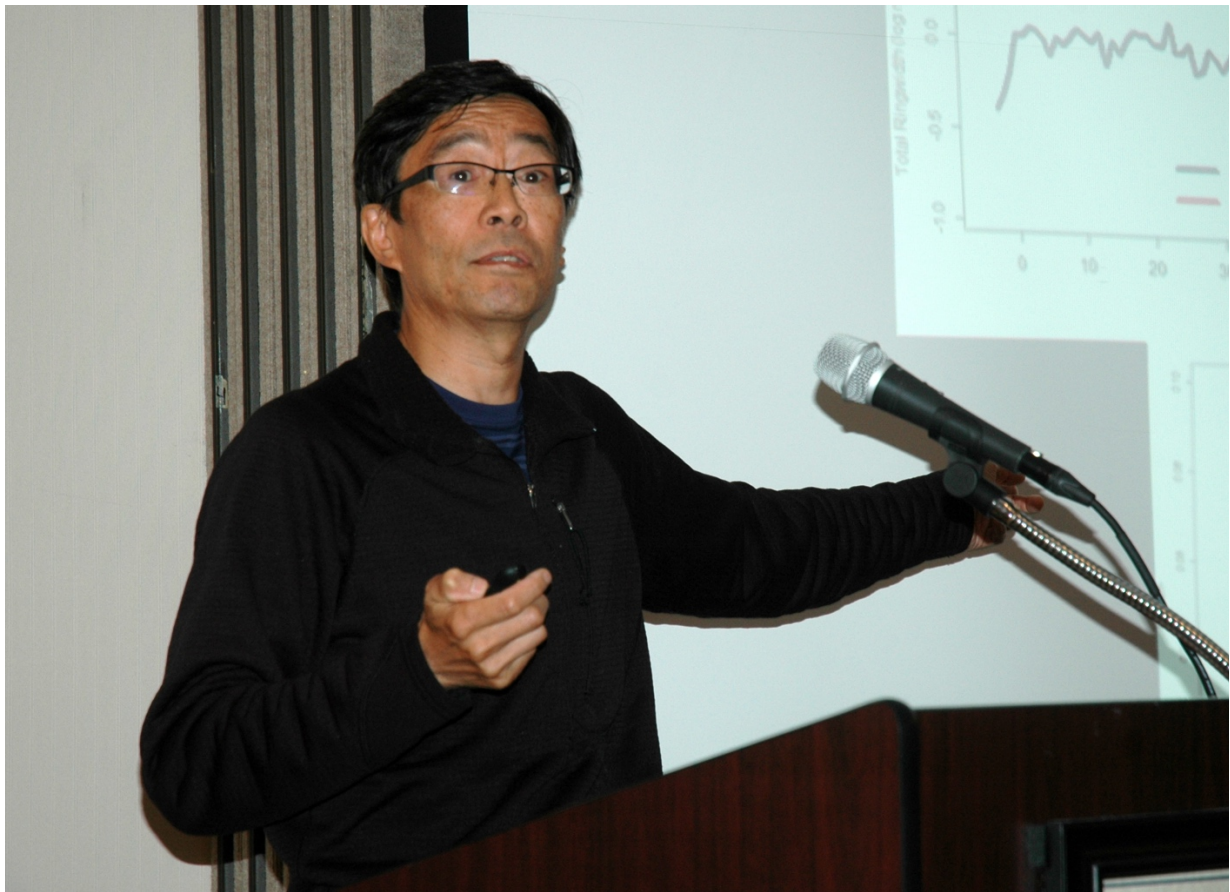
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Special Papers



TO THE VECTOR GO THE SPOILS: IDENTIFYING FUNGI ASSOCIATED WITH WALNUT TWIG BEETLE, A VECTOR OF THOUSAND CANKERS DISEASE COMPLEX

Dixie Daniels¹, Robert Lord², Phillip A. Wadl², Katheryne A. Nix², Lisa M. Vito², Paul R. Merten³, Mark T. Windham², Paris L. Lambdin², Jerome F. Grant², Gregory J. Wiggins², and Denita Hadziabdic²

ABSTRACT

The fungus, *Geosmithia morbida*, vectored by the walnut twig beetle (WTB), *Pityophthorus juglandis*, causes mortality in black walnut (*Juglans nigra*) known as Thousand Cankers Disease (TCD). Infected trees exhibit symptoms similar to drought, making disease identification difficult. Early detection and confirmation of the pathogen is feasible using molecular markers. In 2010, TCD was discovered in Tennessee and has since been detected in four more eastern U.S. states within the native range of black walnut. Our objective was to identify fungal pathogens associated with WTB infestation of black walnut in Tennessee. A total of 180 WTB were collected from two symptomatic trees in Knoxville, Tennessee. WTB were separated by sex, then divided into 3 treatment groups (decapitation, washing, or direct plating), and placed onto an antibiotic amended medium. After a week, fungal isolates were identified based on morphology and

confirmed by sequencing ITS1 and ITS4 regions. Additionally, microsatellite loci were used to detect the presence of *G. morbida*. Statistically significant associations between pathogen presence and direct plating method ($P=0.026$), and between male WTB and *G. morbida* ($P=0.044$) were determined. Although four species of *Fusarium*, including *F. solani*, were isolated from both female and male WTB, the findings were not significant with respect to sex. It is vitally important to fully understand TCD complex and the role of secondary pathogens in TCD pathogenicity.

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LIMBER PINE CONSERVATION STRATEGY: RECOMMENDATIONS FOR ROCKY MOUNTAIN NATIONAL PARK

Christy M. Cleaver^{1,2,3}, Anna W. Schoettle³, Kelly S. Burns⁴, and J. Jeff Connor⁵

ABSTRACT

Limber pine (*Pinus flexilis*), designated by Rocky Mountain National Park (RMNP) as a Species of Management Concern, is a keystone species that maintains ecosystem structure, function, and biodiversity in the park. In RMNP, limber pine is declining due to the interacting effects of recent severe droughts and the climate-exacerbated mountain pine beetle (*Dendroctonus ponderosae*) outbreak, and is imminently threatened by the invasion of the non-native pathogen (*Cronartium ribicola*) that causes the lethal disease white pine blister rust (WPBR) in five-needle white pines. Loss of limber pine will likely lead to cascading ecological impacts and loss of biodiversity in RMNP and surrounding area if impacts are not mitigated. This strategy is the outcome of collaboration between RMNP, Rocky Mountain Research Station (RMRS) and Forest Health Protection (FHP). It addresses the unique situation of limber pine in RMNP and the Southern Rockies. It recommends proactive conservation actions specific to the Southern Rockies based on knowledge from the RMNP/RMRS/FHP program and other research that provides site-based information before ecosystem impairment by WPBR. It focuses on timing specific monitoring efforts and interventions to sustain healthy limber pine populations and ecosystems during invasion and naturalization of WPBR and thereby putting limber pine on a trajectory that does not lead to ecosystem impairment in the future.

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The objectives of the Limber Pine Conservation Strategy are to: (1) conserve genetic diversity and populations of limber pine throughout the park, (2) provide science-based recommendations appropriate for the park and wilderness within the park to sustain healthy high-elevation ecosystems, and (3) develop the park's continued role in the Southern Rocky Mountains to preserve the genetic integrity of native flora and fauna (Schoettle *et al.* in press).

RMNP is in a unique and enviable position in western North America as it is proactively focusing on this threatened species while the ecosystems are still in "healthy" (pre-WPBR caused mortality of reproductive trees) condition, thus enabling informed management to avoid and mitigate ecological impacts before they are expressed. The Conservation Strategy recommends continued and expanded application of the current proactive management approach as it offers the best opportunity to prepare the landscape for greater resilience before WPBR-caused impacts develop and to sustain populations as WPBR spreads (Schoettle *et al.* in press). The major focal areas for management activities are: promote *ex situ* and *in situ* conservation; increase population size and sustain genetic diversity; maintain durability of qualitative WPBR resistance; discover, develop, and deploy local quantitative WPBR resistant sources; and monitor pines and rust.

In spring of 2015, five monitoring plots in riparian areas at high risk for WPBR were installed for early detection and mitigation of WPBR. As a result, the recommended monitoring networks in the park are complete and include the (1) Forest Health Monitoring Network comprised of the ten forest health plots in the limber pine research areas established in 2013 and the three forest health plots established by USFS Forest Health Protection in

2006, (2) Early Detection Monitoring Network with the five riparian monitoring areas for early detection and mitigation of WPBR, and (3) WPBR Virulence Monitoring Network comprised of the 28 individual trees determined to have *Cr4* complete resistance to WPBR for monitoring virulence in the rust. Assessment protocols for the monitoring networks will be compiled in a guide and submitted to the park. This guide and the Conservation Strategy can be applied to other lands in the Southern Rockies as well as elsewhere.

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***DOTHISTROMA SEPTOSPORUM* IN ALBERTA**

Tod Ramsfield¹, Colin Myrholm¹, and Brad Tomm¹

ABSTRACT

In 2012, *Dothistroma septosporum* (Dorog.) Morelet. (teleomorph = *Mycosphaerella pini* E. Rostrup) was isolated from lodgepole pine (*Pinus contorta* var. *latifolia* Dougl. ex Loud.) at the Alberta Tree Improvement and Seed Centre located near Smoky Lake, Alberta. The operators of the facility responded to this identification by introducing management activities to reduce the impact of the pathogen within the affected area (Romano 2013). This discovery also elevated the profile of this pathogen in Alberta and raised concern over its impact on jack pine (*Pinus banksiana* Lamb.) in the boreal forest Jack pine has been recorded as a host of *D. septosporum* in the Czech Republic (Jankovský *et al.* 2004) but records of this pathogen on jack pine in Canada are limited. Funk and Parker (1966) recorded the pathogen on hybrid *P. contorta* var. *latifolia* X *P. banksiana*, and the Forest Insect and Disease Survey records of the Canadian Forest Service contain two records of the pathogen infecting *P. banksiana* in Newfoundland and one record from *P. banksiana* in Ontario. Within the forest pathology herbarium of the Northern Forestry Centre (CFB) there are no accessions of *D. septosporum* on *P. banksiana*. The pathogen has been recorded as present in Alberta in the Canadian Plant Disease Survey reports of 1997 (Stobbs 1997), 1998 (Stobbs 1998), and 1999 (Morrall 1999), although the host information was recorded as “pine”, or lodgepole, or Scots pine (*Pinus sylvestris* L.). This search through our local resources indicated that *D. septosporum* has been recorded in Alberta in the past and that it can infect *P. banksiana*; however, the potential impact of this pathogen on *P. banksiana* in Alberta and the broader boreal forest is unknown.

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In order to investigate the impact of *D. septosporum* in Alberta, a targeted survey was conducted in the summer of 2015. We surveyed natural jack pine stands in close proximity to the Alberta Tree Improvement and Seed Centre and we did not detect *D. septosporum* in these stands. We also surveyed random jack pine stands in central Alberta and we did not detect the pathogen in those surveys. In addition to jack pine, we surveyed lodgepole pine stands in the Hinton and Rocky Mountain House regions of Alberta. We identified several foliar pathogens on lodgepole pine, including: *Elytroderma deformans* (Weir) Darker, *Lophodermella concolor* (Dearn.) Darker, and *Hendersonia pinicola* Wehm., that are known to be common in Alberta (Hiratsuka *et al.* 1995) but no *D. septosporum* was observed. To date, the greatest impact of *D. septosporum* in Alberta has been within the Alberta Tree Improvement and Seed Centre on lodgepole pine. We have not observed *D. septosporum* infection of *P. banksiana* in a natural stand. These results are preliminary and we will continue to monitor for the presence of *D. septosporum* in Alberta, especially in light of the impacts of this pathogen in British Columbia where its increased incidence has been linked to climate change (Woods *et al.* 2005).

ACKNOWLEDGEMENTS

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TEMPORAL EPIDEMIOLOGY OF SUDDEN OAK DEATH IN SW OREGON

Ebba Peterson¹, Everett Hansen¹, and Alan Kanaskie²

An effort to eradicate *Phytophthora ramorum*, causal agent of sudden oak death, has been underway since its discovery in Curry County in 2001. Despite the yearly removal of infected hosts, *P. ramorum* has continued to spread within Oregon forests. Considerable variation in measures of epidemic severity – distances between new sites and the nearest site of any year prior, or the total infestation size of a given year – is present between years. Most of this variation is likely attributable to variation in weather conditions between years, specifically those conditions influencing sporulation and spread. Using an information-theoretical approach we sought to model this yearly variation in an effort to better predict epidemic severity in subsequent years.

Per standard dispersal and disease curves, we expect to observe disease at further distances and greater amounts of disease at a given distance following environmentally conducive years resulting in larger sources of primary inoculum. However, due to the delay between site establishment and detection, the effect of these environmental conditions will be delayed by one or more years. Maximum dispersal distances were best modeled by spring and winter precipitation two years before detection, and infestation size the year prior. Infestation size was best modeled by infestation size and spring precipitation the year prior. In our interpretation, there is a two year delay between the introduction of inoculum and onset of mortality for a majority of sites. The full year prior to detection allows for the production of inoculum contributing to the spread of *P. ramorum* within a site (influencing infestation size) and to new areas (influencing maximum dispersal distances) (Peterson *et al.* 2015).

In application, years conducive to sporulation will have two impacts on epidemic development in subsequent years: conducive years will result larger infestation sizes the following year; however, greater dispersal distances will not be observed until after two years have passed. For example, the extra wet spring of 2010 was one factor contributing significantly to a sharp jump in new infestations observed in 2011, and the great number of dispersal events further than 4 km away from the nearest known inoculum source observed in 2012.

Of importance is the eradication of the EU1 lineage, first recovered from tanoak in Curry County in 2015. Given the weather conditions between 2013-2015 and the size of the 2015-EU1 detection, our model predicts new 2016-EU1 sites should be located within 1.7 km of the 2015 detection and encompass a total infested area of 4.89 ha. However, standard error estimates are very large due to the lack of predictive power in our model.

Post-eradication, we have observed an increase in the total area of new outbreaks and increased frequency in dispersal distances greater than 4 km, including dispersal distances greater than 8 km. We conclude that the eradication program was able to successfully eliminate local spread of *P. ramorum*, however the multi-year delay between infection and detection of new sites has prevented the full eradication of SOD from SW Oregon. SOD will likely spread at a faster rate now that full eradication is no longer being attempted, however weather conditions will continue to moderate the number and range of new SOD detections.

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DETERMINING THE EFFICACY OF STEAM AS A MITIGATION FOR PHYTOPHTHORA SPECIES IN SOIL

*Sarah Navarro*¹ and *Dr. Nancy Osterbauer*¹

Phytophthora ramorum, the causal organism of sudden oak death, has continued to persist in ornamental nurseries in the United States since its discovery in 2003 at a nursery in California. The United States Department of Agriculture (USDA), State Departments of Agriculture, and multiple research groups have worked cooperatively to develop pathogen mitigation options to prevent pathogen spread. Since 2004, the Oregon Department of Agriculture has been surveying ornamental nurseries shipping nursery stock interstate to meet the requirements of 7 CFR 301.92, which established a federal quarantine for *P. ramorum*. In 2014, the USDA revised the federal program with the issuance of Federal Order DA-2014-12. This federal order established new robust sampling protocols to survey all possible sources of *P. ramorum* in high-risk ornamental nurseries and high-risk nurseries comprised of interstate-shipping nurseries with known *P. ramorum* infestations on or after March 31st, 2011. Additionally, this federal order shifted the focus from pathogen eradication at these nurseries to the implementation of a systems approach for pathogen management to prevent future pathogen infestations.

In Oregon, fourteen interstate-shipping nurseries are participating in the revised federal program, of which eight nurseries had plants containing *P. ramorum* during the spring 2014 survey conducted by the Oregon Department of Agriculture (ODA), USDA Animal and Plant Health Inspection Service (APHIS), and Plant Protection and Quarantine (PPQ). Once *P. ramorum* is detected in a nursery, the Confirmed Nursery Protocol (CNP) must be enacted to eliminate the pathogen. This includes

destroying all plant material within a two-meter radius of the *P. ramorum*-positive plant, as well as testing the native soil beneath the plant for the presence of the pathogen. If the native soil is infested with *P. ramorum*, the nursery must decide on a mitigation option, which recently has included steam sterilization. Since 2014, the ODA has performed steam sterilization at four nurseries across the state following protocols developed by USDA APHIS, Center for Plant Health Science and Technology (CPHST), and the National Ornamentals Research Site at Dominican University (NORS-DUC).

During treatment at each of the nurseries, the soil infestation is delimited and a rectangular steam treatment area is determined. This area can range from a small area of 3 by 2 meters up to 8 by 14 meters. Composite soil samples are collected within the treatment area at each of the four corners and in the center at three different depths: 5cm, 15cm, and 30cm. The soil samples are processed according to USDA APHIS protocol and tested for the presence of *P. ramorum*. Temperature probes are placed into each of the sample holes at all three depths to monitor the heating of the soil throughout the steaming process. A Sioux Steam Flo Steam Generator owned by the ODA is used at the nurseries to generate steam. An 18 oz. PVC tarp and sand bags are used to keep the steam in contact with the soil in order to heat it to 50°C for 30 minutes at the 15cm depth. The steaming process took an average of 11 hours of steamer operation per treatment area. The steaming time depends on multiple factors including soil type, ambient temperature, treatment size, and steamer functionality. Following steam treatment, soil samples are again taken from the beginning sampling locations and tested for the presence of *P. ramorum*.

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If no *P. ramorum* is detected in the soil samples taken following steam sterilization, the area is determined to be mitigated for the pathogen and the nursery may use the area for production. The steam

treated areas are being continually monitored and *P. ramorum* has not been detected in subsequent soil samples six months after the steaming occurred.



Posters



DETERMINING THE PATHOGENICITY OF *PYTHIUM* SPP ON BIGLEAF MAPLES

Alex Abair¹, Everett Hansen¹, Wendy Sutton¹, and Paul Reeser¹

INTRODUCTION

Bigleaf maples in southwestern Washington state have recently been experiencing a decline due to an unknown cause. While on an observational expedition through the region of decline in the summer of 2014, we collected soil and root samples of maples exhibiting leaf scorching and major leaf loss. Of the known pathogens isolated from the field samples, several were determined by DNA sequencing to be species of *Pythium* most closely related to *Pythium attrantheridium* and *Pythium intermedium* (Figure 1). From these isolates, we decided to conduct two pathogenicity tests on bigleaf maple seedlings. One experiment was a soil inoculation under flooding conditions, and the other was a stem wound inoculation. The soil inoculation experiment consisted of just *Pythium* isolates, while the stem inoculation

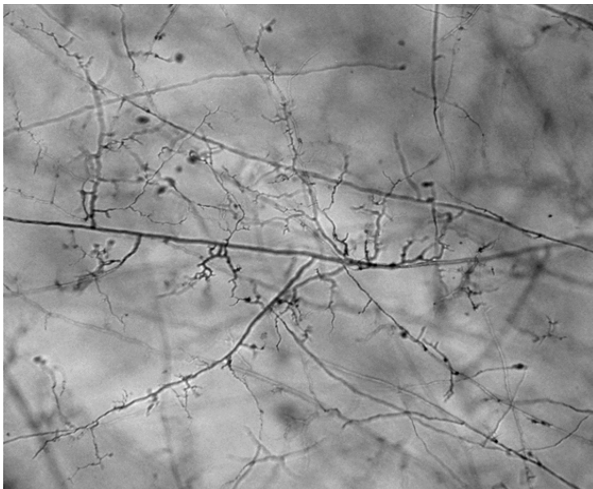


Figure 1. *Pythium intermedium*.

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consisted of both *Pythium* and *Phytophthora* isolated from field samples. The following table lists the isolates used in our experiments (Table 1).

Table 1. Isolates used for inoculations.

Isolate	Genus	Species	Source
Pyth. 1	<i>Pythium</i>	98% AB468806	Root Bait
Pyth. 2	<i>Pythium</i>	close to <i>intermedium</i> & <i>attrantheridium</i>	Root Lesion
Pyth. 3	<i>Pythium</i>	91% <i>attrantheridium</i>	Root Lesion
Pyth. 4	<i>Pythium</i>	<i>intermedium</i>	Root Lesion
Pyth. 5	<i>Pythium</i>	close to <i>intermedium</i> & <i>attrantheridium</i>	Root Lesion
Phyt. camb.	<i>Phytophthora</i>	<i>cambivora</i>	Soil
Phyt. plur.	<i>Phytophthora</i>	<i>plurivora</i>	Soil
Phyt. syring.	<i>Phytophthora</i>	<i>syringae</i>	Soil
Phyt. chlam.	<i>Phytophthora</i>	<i>chlamydospora</i>	Soil

Stem Inoculation

We investigated the pathogenicity of the selected *Pythium* isolates when they were directly applied to stem wounds. Four *Phytophthora* isolates collected from the field samples were also included in this pathogenicity test.

Stem Wound Inoculation

- *Pythium* and *Phytophthora* isolates were grown on corn meal agar with β -sitosterol.
- An incision was cut in the cambium so that a 3/4 cm triangular flap was created.
- Agar disks from the colonized plates were placed inside of the cambium flaps and then wrapped in gauze and foil.
- The stems were unwrapped after four weeks and the lengths of lesions (if present) were measured (Figure 2).

Results

All isolates used in this experiment produced larger lesions on average than the controls, but only five were statistically significant (Figure 3). A larger sample size might prove that all of the *Pythium* isolates would be pathogenic to bigleaf maples, but we can only say with a reasonable degree of confidence that two of them are. Three of the

Phytophthora isolates we used produced the largest lesions of all. All but *Phytophthora syringae* produced statistically significant results; *Phytophthora plurivora* produced the largest black lesions.

A.



B.



C.



D.



Figure 2. Lesions from the stem inoculations. **A)** Negative control, **B)** *Pythium*, **C)** *Phytophthora* and **D)** variation within treatment.

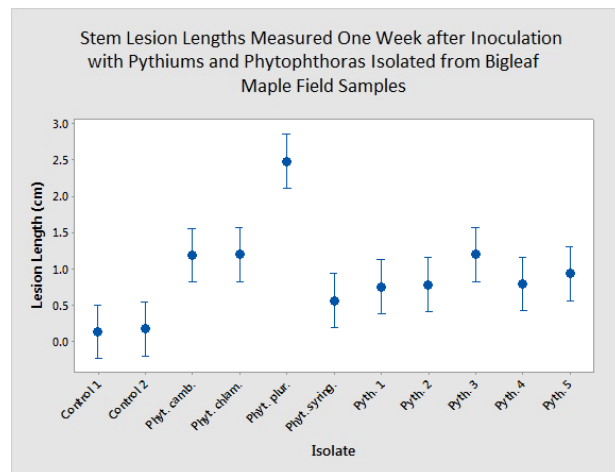


Figure 3. The vertical bars represent 95% confidence intervals. Stem lesions were measured as the length from the top to bottom of the dark discoloration at the site of infection.

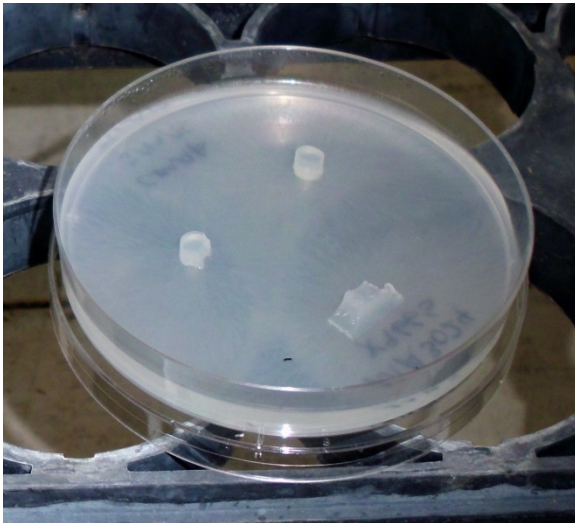
Soil Inoculation

We attempted to determine whether or not soil inoculated with the selected *Pythium* spp would cause symptoms of disease in bigleaf maple trees. Half of the trees were inoculated by inserting colonized agar plugs into the soil, and the other half were inoculated with colonized pea broth.

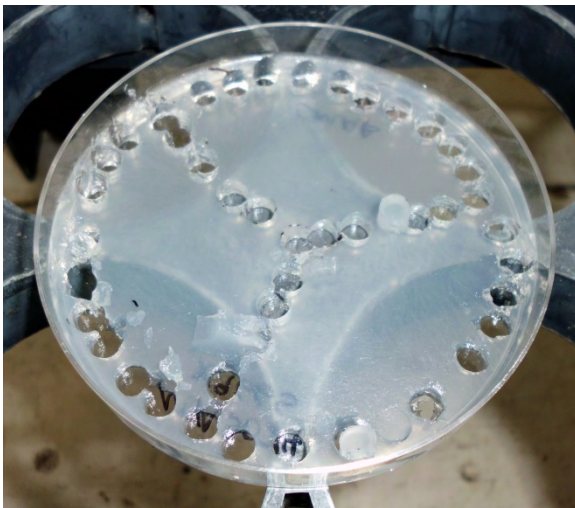
Agar Disk Inoculation

- *Pythium* isolates were plated onto corn meal agar with β -sitosterol.
- Transfer tubes were filled with 15 agar plugs from the periphery of the hyphal masses.
- The plugs were inserted 15cm into the soil at three equidistant holes. Soil was brushed on top and the trees were watered (Figure 4).
- Flooding was simulated three times over the next five weeks by placing the pots in buckets filled with enough water to submerge them up to the soil surface for 24 hours.

A.



B.



C.



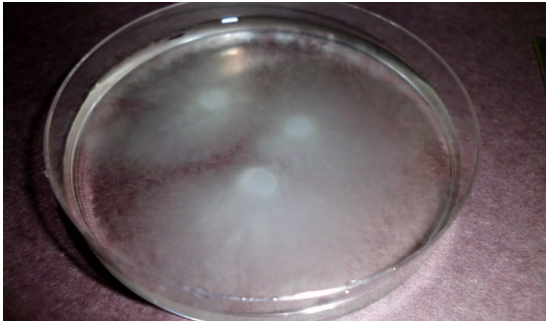
Figure 4. Agar disk inoculations. **A)** colonized agar plate, **B)** plugs taken from colony edge and **C)** BLM seedling inoculation.

Pea Broth Slurry Inoculations

- Three colonized agar plugs were placed into 15 mL of pea broth and allowed to grow into mycelial masses measuring 1-3 cm radially.
- Three equidistant, 13 cm deep holes were pressed into the soil, and one mycelial mass was inserted into each hole (Figure 5).

- The remaining pea broth from the slurry was poured equally into the holes, and the holes were then filled with soil.
- Trees were watered and flooding was simulated as in the agar plug inoculation.

A.



B.



C.



Figure 5. Pea broth slurry inoculations. **A)** Colonies grown in pea broth, **B)** making holes for mycelial masses and **C)** inoculating with mycelial masses.

Results

The oven-dried roots of each tree were compared by weight. There were no major root disease symptoms, and visual inspection showed no differences in the number or color of fine roots when inoculated trees were compared with the control group (Figure 6). Average weights of each of the isolate groups showed no significant differences (Figure 7). This may be due to the initial variation in tree size at the start of the experiment. A larger study may be required to obtain more conclusive results.

CONCLUSION

Direct stem inoculations with the selected *Pythium* spp resulted in the formation of lesions in every case, but only two isolates produced lesions of statistical significance when compared to the controls. *Phytophthora cambivora* and *Phytophthora clamydospora* produced lesions of a similar average size to the *Pythium* being tested, and *Phytophthora plurivora* produced lesions that were larger than all others. *Phytophthora syringae* had the smallest lesions of all of the isolates.

The same five *Pythium* isolates showed no statistically significant evidence of being pathogenic to bigleaf maple seedlings when they were introduced to the soil. The absence of symptoms and fatalities suggests that these *Pythium* isolates do not have an effect on the roots of maples simply by being present in the soil under the conditions of this experiment.

GENOTYPIC DIVERSITY, PHYLOGEOGRAPHY, AND POPULATION STRUCTURE OF TWO LINEAGES OF *PHAEOCRYPTOPUS GAEUMANNII* IN THE PACIFIC NORTHWEST

Patrick Bennett¹ and Jeffrey Stone¹

INTRODUCTION

The ascomycete fungus *Phaeocryptopus gaeumannii* is the causal organism of Swiss needle cast (SNC), a foliage disease of Douglas-fir (*Pseudotsuga menziesii*). The disease was first described in forest plantations in Europe in the 1920s (Boyce 1940). It has since been determined that *P. gaeumannii* is endemic to western North America and the native range of Douglas-fir in the northwestern United States. Initial assessments of genetic diversity and population structure performed using molecular markers known as single strand conformation polymorphisms (SSCPs) indicated the presence of two distinct sympatric populations (lineages) of the fungus in the Pacific Northwest (Winton *et al.* 2006). Subsequently a set of more variable molecular markers, microsatellites or short sequence repeats (SSRs), that permit more fine-scale analysis of *P. gaeumannii* populations were developed by Winton *et al.* (2007), but intensive sampling and analyses using these markers were not undertaken until now.

RESEARCH OBJECTIVES

The objectives of this study were to test whether *P. gaeumannii* comprises two distinct lineages in western Oregon and Washington, as inferred from SSCP data, and to compare the distributions of *P. gaeumannii* genotypes in the PNW with spatial variation in SNC severity as observed in ODF/USFS aerial surveys. The longer-term objectives of this research aim to assess *P. gaeumannii* population structure and diversity through the analysis of multi-locus microsatellite genotypes.

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METHODS

Field Sampling

The 14 sites included in this analysis (Figure 1) were selected from a long-term monitoring plot network established by the Oregon State University Swiss Needle Cast Cooperative (SNCC) (Ritokova *et al.* 2013). Secondary branches were collected from the upper canopies of 5 randomly selected trees at each site. Additional foliage samples were collected from the lower and middle canopies of one of these trees at each site.

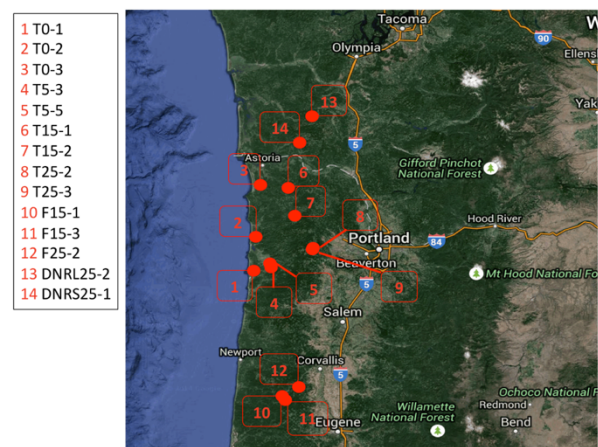


Figure 1. Map showing the distribution of the 14 sites included in this analysis.

Isolations and Culturing

Several needles bearing pseudothecia were attached to the covers of Petri dish with double-sided adhesive tape and suspended above the media to discharge ascospores onto the agar surface. After 24 hours the plates were examined under the stereomicroscope and single ascospores were isolated with the aid of flame-sterilized forceps onto 2% malt agar in 60 mm Petri dishes. These cultures were incubated at 20° C for 2–6 months to obtain adequate growth for DNA extraction.

Molecular Techniques

Genomic DNA was extracted by using the Qiagen DNeasy Plant kit following the manufacturer's protocol, with the addition of an initial maceration step. Ten microsatellite loci were amplified in 3 multiplexed PCR reactions. The primers were identical to those in Winton *et al.* (2007), but did not include the M13 universal tails resulting in shorter amplicons. A fluorescent dye label was included on each of the reverse primers to allow the amplified fragments to be distinguished from one another after genotyping. Fragment sizes were determined by capillary electrophoresis at the Oregon State University Center for Genome Research and Biocomputing (CGRB). Alleles were scored with the ABI GeneMapper 4.0 program. Lineage determination for each isolate was made according to the descriptions of multilocus

genotypes (MLGs) described by Winton *et al.* (2007).

Data Analysis

The microsatellite genotypes were analyzed initially by using GenAlEx 6.5 (Peakall and Smouse 2006, 2012). Population genetics parameters obtained with this analysis included locus statistics, allelic diversity, and PhiPT estimates from an analysis of molecular variance (AMOVA). Further analyses were performed with R version 3.2.1 via R Studio. *Poppr 2.0.2* (Kamvar *et al.* 2014) was used to calculate genotypic diversity and to construct UPGMA dendrograms with bootstrapping. Adegnet 2.0.0 (Jombart 2008) was used for the discriminant analysis of principal components (DAPC) and K-means clustering.

Table 1. a) Diversity statistics for each of the sampling sites. b) Diversity statistics for the two cryptic *P. gaeumannii* lineages. N= sample size, L1= abundance lineage 1, L2= abundance Lineage 2, MLG= number of multilocus genotypes, H= Shannon-Weiner diversity index.

a) Population	N	L1	L2	MLG	H
T-01	49	23	26	48	3.86
T-02	10	4	6	9	2.16
T-03	17	9	8	17	2.83
T5-3	29	22	7	29	3.37
T5-5	21	17	4	20	2.98
T15-1	46	23	23	45	3.80
T15-2	24	10	14	23	3.12
T25-2	99	99	0	90	4.43
T25-3	60	59	1	59	4.07
F15-1	29	27	2	27	3.27
F15-3	15	4	11	15	2.71
F25-2	37	25	12	32	3.41
DNRS25-1	21	13	8	25	3.22
DNRL25-2	25	19	6	16	2.71
14	482	358	124	454	6.09
b)					
Lineage 1	358			335	5.78
Lineage 2	124			119	4.76
	482			454	6.09

RESULTS

Of the 482 isolates from 14 sites in Oregon and Washington included in this analysis, 454 (94%) possessed unique multilocus genotypes (MLGs) (Table 1). A total of 358 of these isolates had MLGs corresponding to previously reported alleles characteristic of Lineage 1, while 124 corresponded with Lineage 2 (Winton *et al.* 2007). Sites along the western Coast Range in northern Oregon and southern Washington, where SNC is most severe, consisted of roughly equal proportions of the two lineages and had relatively low genotypic diversity compared to isolates from sites further east. Diversity increased gradually in sites to the east of Tillamook, and was highest in the eastern Coast Range where Lineage 1 is more abundant (Figure 2, Figure 3). Only a single isolate of Lineage 2 was found at a site over 25 miles inland from Tillamook, Oregon (Table 1, Figure 3).

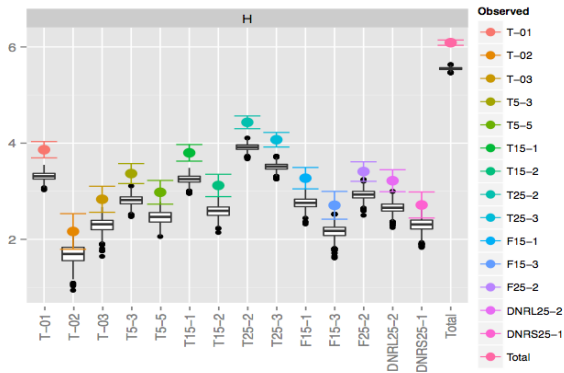


Figure 2. Comparison of genotypic diversity by sampling site. H = Shannon-Weiner diversity index. Diversity was calculated on a rarefied sample of 10 isolates per site. Error bars represent 95% confidence intervals; boxplots represent values estimated from bootstrap replicates.

A partitioning of molecular variance suggested that variation within subpopulations (sites or lineages) is greater than that between subpopulations. When sites were considered as subpopulations, within-site variation accounted for 90% of the total variation (Figure 4A), compared to 78% when lineages were considered subpopulations (Figure

4B). Estimates of subpopulation differentiation (PhiPT) were used to assess gene flow among populations. These analyses revealed that the populations across all sampled sites are only moderately differentiated from one another (PhiPT = 0.101, $P = 0.001$) (Figure 4A), whereas the lineages are strongly differentiated (PhiPT = 0.218, $P = 0.001$) (Figure 4B). The degree to which gene flow occurs between subpopulations was also assessed with a K-means clustering approach in which each isolate is assigned to a genetic group based on shared alleles, and the optimal number of genetic clusters in the data is determined based on estimates of the Bayesian Information Criterion (BIC). A total of 12 distinct genetic clusters were identified using this method. This technique was used in conjunction with the unweighted pair group method using arithmetic means (UPGMA) algorithm to analyze reproductive modes, population structure, and gene flow. The 12 clusters did not correspond to any distinct geographic group or lineage resulting in a mixing of genetic clusters across the branches of the UPGMA dendrogram (Figure 5A). This analysis was also performed with the isolates divided into 2 clusters without any *a priori* assignment to a lineage. This resulted in a clear assignment of the isolates to genetic clusters corresponding to the two lineages, as well as a correspondence of these clusters to the lineages on the UPGMA dendrogram (Figure 5B).

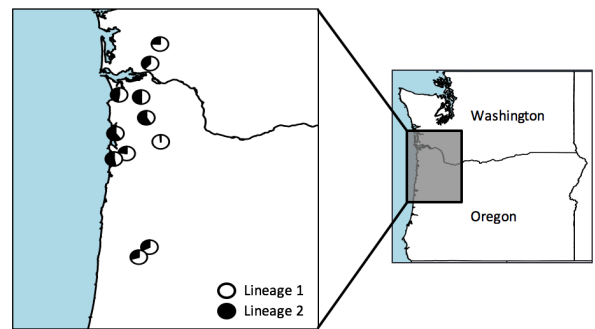


Figure 3. Geographic distributions of the two *P. gaumannii* lineages across the sampling sites. Isolates from sites T5-3 and T5-5, T25-2 and T25-3, F15-1 and F15-3 were pooled for this analysis to avoid overlap.

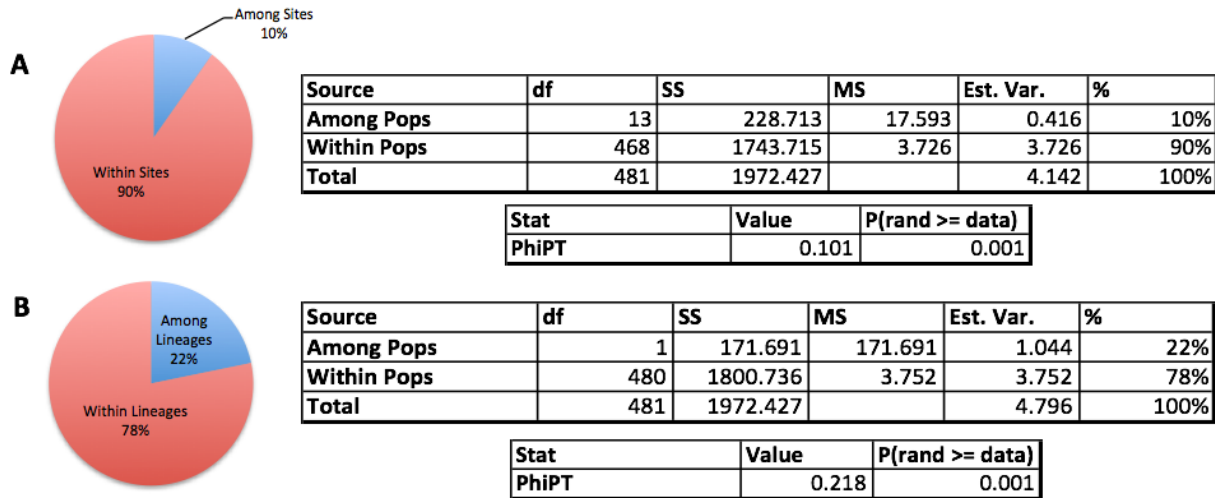


Figure 4. The results of an AMOVA comparing genetic variance within and among subpopulations. (A) Here, the subpopulations are the sampling sites. (B) Here, each lineage was considered a subpopulation. P-values from randomization test with 999 permutations.

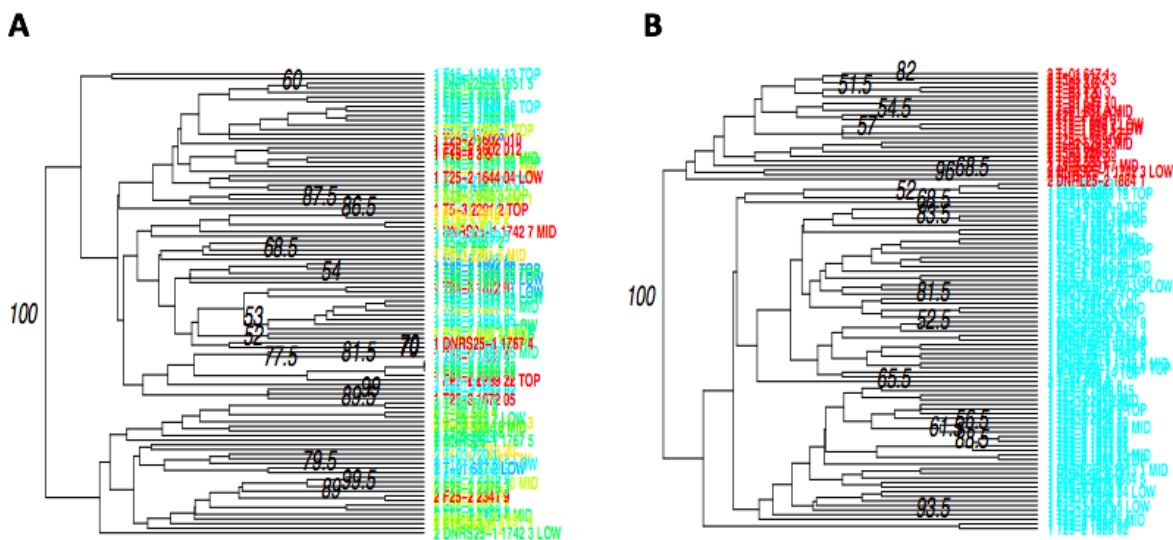


Figure 5. UPGMA dendrograms constructed with 100 randomly selected isolates. The colors represent genetic clusters identified using a K-means approach. Node labels represent bootstrap statistics for 200 replicate trees. **A**) The optimum number of clusters ($K=12$) was chosen based on Bayesian information criterion (BIC). **B**) $K=2$ was chosen, and the two clusters correspond to the two lineages. Here, blue= Lineage 1, red= Lineage 2.

The DAPC analysis was used to depict the relationship among subpopulations graphically. When sites were considered subpopulations, the isolates collected from T-02 and DNRS25-1 were significantly differentiated from those at all other sites, which formed a more compact cluster (Figure 6A). This differentiation was likely due to the

presence of unique alleles present at only these two sites. When lineages were treated as subpopulations for this analysis, there was a distinct separation with no overlap, further supporting their strong differentiation and the lack of sexual recombination between them (Figure 6B).

DISCUSSION

Geographic centers of origin of plants, fungi, and microorganisms are generally characterized by high genetic diversity (Grünwald and Flier 2005). Populations of *P. gaeumannii* exhibit very high genotypic diversity and richness in the Pacific Northwest compared to regions where the fungus is known to be introduced, such as New Zealand (Bennett and Stone *this proceedings*), supporting the conclusion that this fungus is native to western North America. While Lineage 1 was found at every site sampled, and exhibited high diversity at sites in the eastern Coast Range, Lineage 2 was restricted to the western Coast Range and showed relatively low genotypic diversity. Lineage phylogeography seems to be correlated with the geographic distribution of SNC disease severity. In northwestern Oregon, the area where the most severe disease has been consistently found in annual aerial surveys corresponds to the area where the two lineages co-occur. There is a strong geographic trend of decreasing disease severity to the east of the Coast Range, where Lineage 2 is virtually absent. However, the significance of this correlation, and the factors influencing the restriction of the range of Lineage 2 to the western Coast Range are not fully understood.

If subpopulations of *P. gaeumannii* (site or lineage) are reproducing primarily by selfing (homothallism), or if gene flow is restricted due to geographic or genetic barriers, genetic clusters (groupings based on shared alleles) should be strongly associated with the geographic origins of the isolates. This would result in a sorting of isolates into geographic groups on the terminal branches of a UPGMA dendrogram, and a clear sorting of the genetic clusters into these geographic groups. Instead, our analyses do not support the interpretation that the 12 K-means clusters (Figure 5A) are related to geography or lineage. The K-means clustering algorithm identified multiple distinct clusters within each lineage from across their geographic ranges. This resulted in a mixing of isolates from different sites into the same K-

means clusters. However when only two clusters were chosen for the K-means analysis, the resulting clusters corresponded exactly to the two lineages.

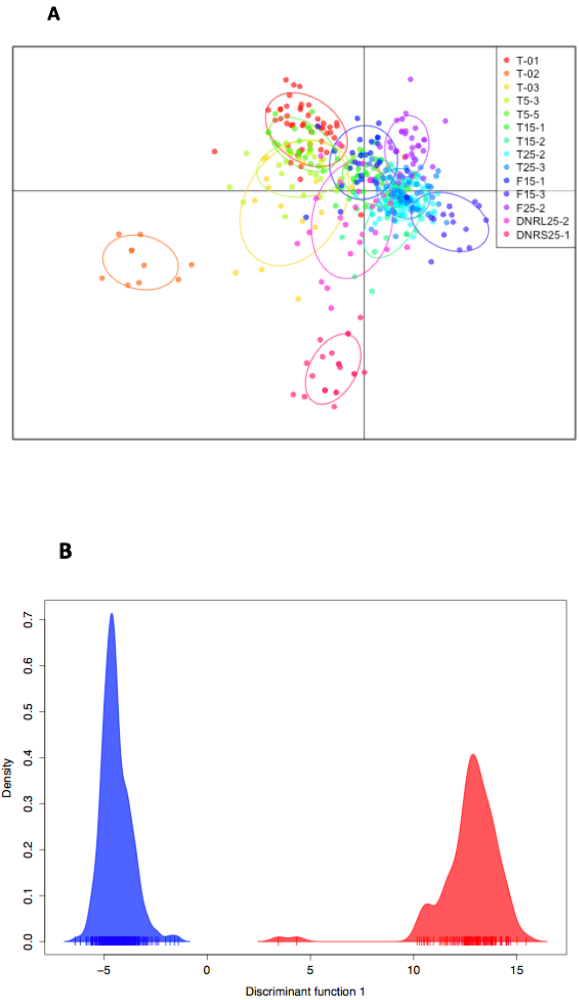


Figure 6. DAPC plots showing relationships among subpopulations. **A)** Colors correspond with sampling sites, and points correspond to individuals. **B)** Colors correspond with lineages. Blue= Lineage 1, Red= Lineage 2.

The separation of the two lineages at a basal node of the UPGMA dendrogram with high statistical support (Figure 5), and the sorting of isolates from the two lineages into distinct genetic clusters (Figure 5B) provides further evidence for the lack of recombination between lineages. These results are consistent with the assessments of differentiation determined with AMOVA.

The AMOVA showed that *P. gaeumannii* populations in the Pacific Northwest are heterogeneous in their genetic structure. Genetic differentiation is strong at the level of the lineage (PhiPT=0.218, P= 0.001), i.e.: the lineages are distinct subpopulations in our region, while the differentiation between sites is weaker (PhiPT= 0.101, P=0.001) suggesting the occurrence of dispersal and connectivity due to gene flow. The strong differentiation between the lineages is likely reflective of a form of reproductive isolation resulting from the predominance of homothallic reproduction and low rates of outcrossing, as suggested by Winton (2006). Although more variation occurred within lineages than between them, suggesting the existence of gene flow, the results of the UPGMA analyses, K-means clustering, and DAPC suggest otherwise. This AMOVA result is likely due to the existence of shared alleles at some of the loci that occurred through the process of convergent evolution rather than sexual recombination.

While the AMOVA allowed for the inference of gene flow and differentiation within and among subpopulations, the DAPC analysis allowed us to identify which, if any, subpopulations (sites or lineages) were significantly differentiated from the others. The results of this analysis, when sampling sites were considered subpopulations, suggested that two of the sites (T-02 and DNRS25-1) were differentiated from the other sites, and from one another (Figure 6A). While this may be due to the presence of rare or unique alleles at these sites, this may also be an artifact of homothallic reproduction resulting in an over-representation of genotypes from closely related individuals at these sites. This result could explain the moderate differentiation between sites revealed in the AMOVA analysis. The clear separation between isolates from the two lineages in the DAPC analysis (Figure 6B) provides further support for the lack of recombination between them, and also corroborates the results of the AMOVA analysis in which lineages were highly differentiated, suggesting a lack of gene flow between lineages.

The occurrence of homothallic reproduction, and the presence of significant subpopulation structure in populations of *P. gaeumannii* may have implications for the evolutionary adaptive potential of *P. gaeumannii*, and thus may have an impact on SNC management strategies and the mitigation of growth impacts in commercial timber production. In addition to the climate variables known to influence SNC disease distribution and severity, the relative proportions of the two lineages present at a site should be considered when making an assessment of future SNC disease risk.

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GENOTYPIC DIVERSITY AND POPULATION STRUCTURE OF *PHAEOCRYPTOPUS GAEUMANNII* IN NEW ZEALAND

Patrick Bennett¹ and Jeffrey Stone¹

BACKGROUND

Swiss needle cast (SNC) and its causal organism, *Phaeocryptopus gaeumannii*, were first detected in New Zealand in 1959 on the North Island near Taupo. Approximately 10 years later, the disease made its first appearance on the South Island (Kimberley *et al.* 2011). While the environmental factors influencing SNC severity in New Zealand have been well studied (Hood and Kimberley 2005, Stone *et al.* 2007, Watt *et al.* 2010, Watt *et al.* 2011), this study represents the first assessment of population genetic structure and diversity for introduced populations of *P. gaeumannii*.

RESEARCH OBJECTIVES

This study aims to compare the genetic variation and diversity present in introduced populations of *P. gaeumannii* from throughout New Zealand, and determine the relative abundances and distributions of two previously described lineages of *P. gaeumannii* present in the country. Population genetic parameters estimated for these populations will be compared to those measured in native populations from Northwestern North America (PNW) to contribute to a more thorough understanding of reproduction, dispersal, and population dynamics in this foliar pathogen of Douglas-fir.

MATERIALS AND METHODS

Field Sampling, Isolations, and Culturing

The isolates included in this analysis were collected from 17 Douglas-fir plantation sites in New Zealand (Figure 1, Table 1) in 2005 and 2007.

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Eight of the sites were on North Island, and the remaining 9 were on South Island (Figure 1). Foliage samples were collected from secondary branches in the upper crowns of 10 Douglas-fir (*Pseudotsuga menziesii* var. *menziesii*) trees at each site. Foliage bearing pseudothecia was affixed to the lids of Petri dishes with double-sided tape to allow ascospores to discharge onto the agar surface below for approximately 24 h. Individual ascospores were then isolated onto 2% malt agar and incubated for 2-6 months to allow adequate growth for DNA extraction.

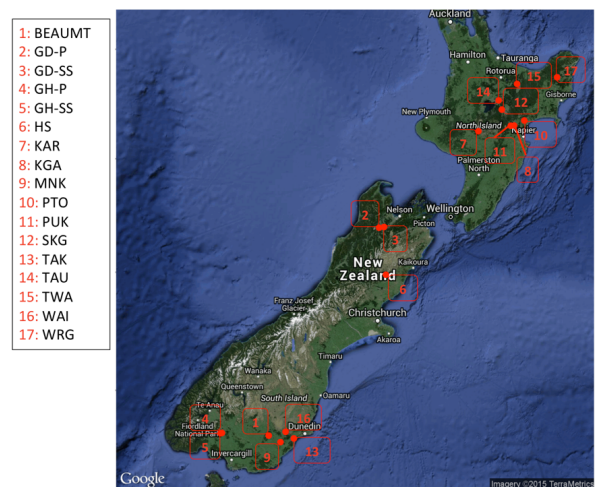


Figure 1. Map of New Zealand showing the distribution of sampling sites on the North and South Islands.

Molecular Techniques

Following thorough maceration of the *P. gaeumannii* mycelium, DNA extractions were performed with a CTAB extraction buffer followed by precipitation in 24:1 chloroform:isoamyl alcohol. The genomic DNA was then purified by passing through a QIAamp spin column. Ten microsatellite loci were amplified in 3 multiplexed PCR reactions using the primers described by Winton *et al.* (2007). Genotyping was performed via capillary electrophoresis at the Oregon State

University Center for Genome Research and Biocomputing (CGRB). For more information on molecular techniques and data analysis see the companion publication in this proceedings: Bennett and Stone- Genotypic Diversity, Phylogeography, and Population Structure of Two Lineages of *Phaeocryptopus gaeumannii* in the Pacific Northwest.

Table 1. Diversity statistics for each of the sampling sites.. *N*= sample size, *MLG*= number of multilocus genotypes, *H*= Shannon-Weiner diversity index. *L1*= Abundance of Lineage 1, *L2*= Abundance of Lineage 2.

Site	N	L1	L2	MLG	H
BEAUMT	144	144	0	42	3.15
GD-P	109	107	2	20	1.73
GD-SS	47	46	1	18	2.40
GH-SS	91	91	0	21	2.29
GH-P	112	112	0	29	2.56
HS	132	132	0	37	2.84
KAR	87	87	0	31	2.88
KGA	119	119	0	36	2.46
MNK	48	48	0	12	1.74
PTO	18	18	0	10	1.98
PUK	20	20	0	12	2.35
SKG	15	15	0	8	1.86
TAK	17	17	0	11	2.17
TAU	129	129	0	73	3.95
TWA	16	16	0	6	1.12
WAI	123	123	0	30	2.37
WRG	14	14	0	8	1.77
17	1241	1238	3	242	3.87

RESULTS AND DISCUSSION

Of the 1241 isolates analyzed for this study, 242 unique multilocus genotypes (MLGs) were detected (19.5%) (Table 1). A total of 1238 of the isolates were identified as Lineage 1, while only 3 isolates corresponded with Lineage 2. The Lineage 2 isolates were all isolated from foliage collected at two adjacent plantations in the northern South Island, GD-SS and GD-P (Table 1, Figure 1). Tauhara (TAU), a site in the central North Island,

had the highest diversity of genotypes among all sites ($H=3.95$) while the lowest genotypic diversity was found in the Hawkes Bay region of the North Island at TeWaka (TWA) ($H=1.12$) (Table 1, Figure 2). Total genotypic diversity was significantly lower for the collection of isolates from New Zealand ($H_{total}=3.87$) compared to those from the PNW ($H_{total}=6.09$) (Table 1, Figure 2).

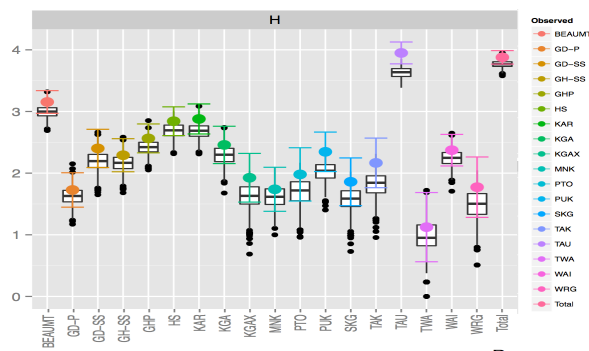


Figure 2. Shannon-Weiner diversity index calculated for a rarefied sample of 14 isolates from each site. Error bars represent 95% Confidence Intervals. Boxplots represent values obtained from bootstrap replicates.

A partitioning of molecular variance (AMOVA) within and among sampling sites indicated that most of the variation is found within sites, but the degree to which this estimate reflects the true underlying genotypic variance depended upon whether we chose to maintain the “clones”, or repeated MLGs, in the data. When these repeated MLGs were included in the analysis, approximately 78% of the variance occurred within sites, and the remaining 22% of the variance was due to among-site differentiation ($\Phi_{PT}=0.220$, $P=0.001$) (Figure 4A). However, when the data was “clone-corrected”, the within-site variance was estimated as 91.4%, and 8.6% of the variance was due to differentiation between sites ($\Phi_{PT}=0.086$, $P=0.001$) (Figure 4B). This result indicated that the apparent strong subpopulation structure was due to the presence of repeated MLGs that arose due to homothallic reproduction and did not reflect the true subpopulation differentiation, which was relatively weak.

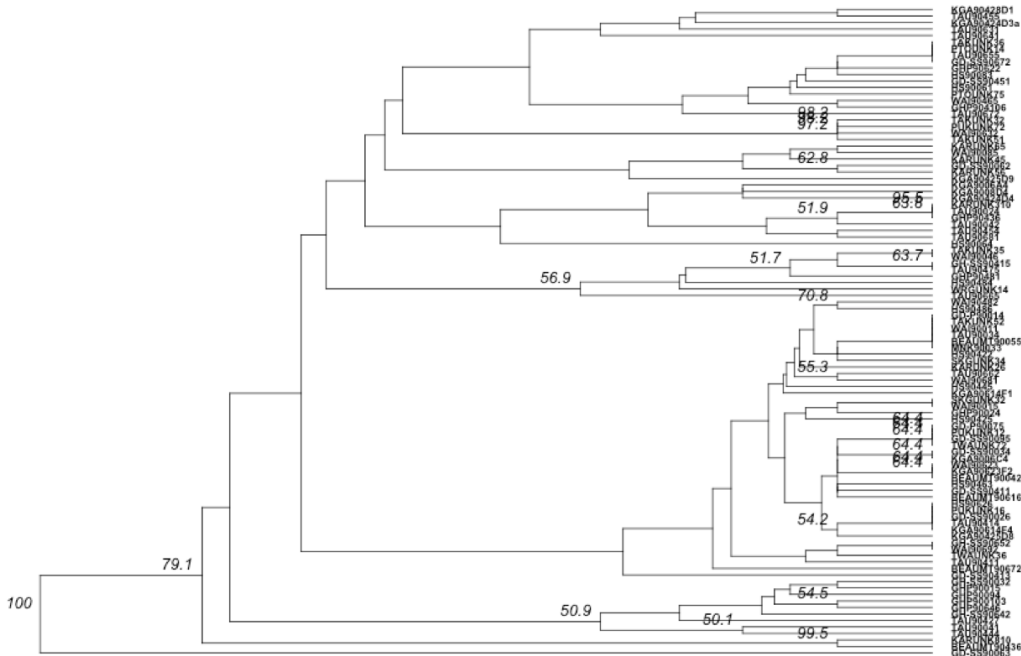
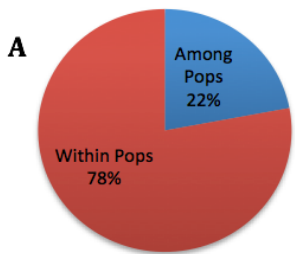
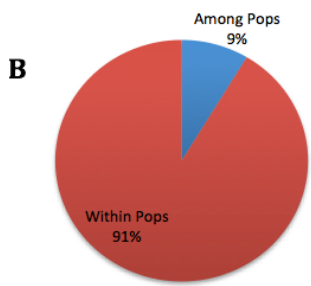


Figure 3. UPGMA dendrogram constructed with 100 randomly selected isolates from the clone-corrected data set. Node labels represent bootstrap values for 999 replicates. The basal node with bootstrap value of 100 represents the divergence of the two lineages.



Source	df	SS	MS	Est. Var.	%
Among Pops	16	1255616.082	78476.005	1052.232	22%
Within Pops	1225	4560463.170	3722.827	3722.827	78%
Total	1241	5816079.253		4775.059	100%

Stat	Value	P(rand >= data)
PhiPT	0.220	0.001



Source	df	SS	MS	Est. Var.	%
Among Pops	16	135.629	8.477	0.252	8.6%
Within Pops	387	1032.864	2.669	2.669	91.4%
Total	403	1168.493	2.889	2.921	100%

Stat	Value	P(rand >= data)
PhiPT	0.086	0.001

Figure 4. Analysis of Molecular Variance (AMOVA) comparing genetic variation within and among sampling sites. The P-value is from a randomization test with 999 permutations. **A.** Repeated multilocus genotypes were included in the AMOVA analysis. **B.** AMOVA with a “clone-corrected” dataset. (repeated multilocus genotypes were removed).

A UPGMA analysis resulted in the placement of isolates from diverse sampling sites into groups on the branch tips, indicating the dispersal and admixture of isolates from across the region. There was insufficient variation among genotypes to assign them to genetic clusters by using a K-means algorithm, further supporting the genotypic homogeneity of *P. gaeumannii* populations in New Zealand. Much like the UPGMA dendrograms constructed with isolates from the PNW (Bennett and Stone, this proceedings), the isolates representing two lineages were separated at a basal node with high statistical support (Figure 3).

The DAPC analysis performed using a clone-corrected data set supported the AMOVA results, and confirmed that there is not significant differentiation among the isolates from various sites in New Zealand. The populations of *P. gaeumannii* sampled for this study appear to form a relatively coherent grouping on the DAPC scatterplot (Figure 5) due to shared allelic states.

The abundance of shared multilocus genotypes in populations of *P. gaeumannii* in New Zealand resulted in a highly clonal structure. This likely reflects the predominance of homothallism (self-fertilization) in these populations, as this fungus is not known to reproduce asexually. The homothallic mode of reproduction indicates a diminished capacity for adaptation, as recombination does not occur between dissimilar individuals. Genotypes comprised of alleles indicative of Lineage 2 were recovered at a very low frequency (3 isolates, or approximately 0.2% of the total). These isolates occurred at two adjacent sites on the South Island (GD-P and GD-SS, Table 1). Whether this is a result of random sampling, or indicative of a separate introduction of *P. gaeumannii* from the Pacific Northwest is not clear. There did not seem to be a relationship between genotypic diversity and phylogeography as observed for the native PNW populations.

As a result of predominately homothallic reproduction in these populations, genotypic diversity was very low compared to native *P.*

gaeumannii populations in the Pacific Northwest of the United States where outcrossing likely occurs at higher frequencies. This could be the result of a founder event in which a small number of genotypes was initially introduced to a site on the North Island of New Zealand that subsequently spread to populate the North and South Islands. Low genetic diversity may also reduce the capacity for evolutionary adaptation in these populations.

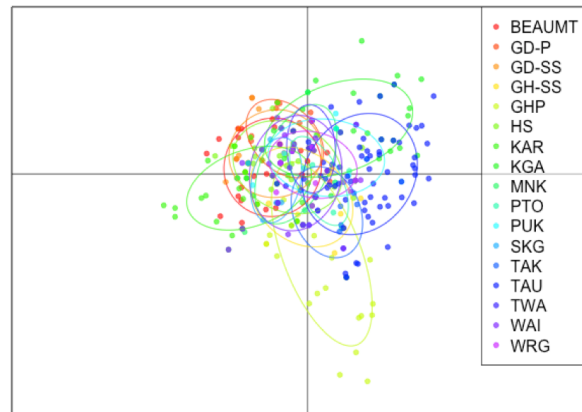


Figure 5. Discriminant Analysis of Principal Components (DAPC) showing relationships among sampling sites. Colors correspond to the 17 sampling sites, and points correspond to isolates.

One site, Tauhara (TAU), had much higher genotypic diversity than the others, and is near the site of initial introduction on the North Island, Taupo (Kimberley *et al.* 2011) (Figure 2). Centers of diversity such as this are generally indicative of sites of origin or regional introduction, and could serve as sources of genetic variation on which natural selection may act. There was apparently no correlation between disease severity and genotypic diversity in New Zealand, as the site with the highest genotypic diversity, Tauhara, and that with the least diversity, TeWaka (Figure 4), both grouped among sites which had the greatest abundance of *P. gaeumannii* in Douglas-fir foliage (Stone *et al.* 2007). Furthermore, the sites where isolates of Lineage 2 were recovered, GD-SS and GD-P, had only moderate amounts of *P. gaeumannii* in foliage and little reduction in foliage retention (Stone *et al.* 2007).

The AMOVA result suggests that the distribution of *P. gaeumannii* in New Zealand is not limited by dispersal, and spores move freely among the sites sampled for this study. The apparent differentiation between the sites is reflective of the mode of reproduction, as clone-correction resulted in the negation of this subpopulation structure. The close grouping of isolates from all of the 17 sites on the DAPC biplot further supports the conclusion that these populations, exhibit low diversity, share allelic states, and have a clonal structure.

The continued intensification of SNC in Douglas-fir plantations in this region of the world is likely a complex combination of factors including climate, fungal population dynamics, and host tree seed source genetics. Strategies for SNC disease management in New Zealand may be better informed with knowledge of population structure and genotypic diversity.

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STIGMINA FOLIAR BLIGHT ON PORT-ORFORD-CEDAR IN OREGON: GENETIC VARIATION RELATED TO PROVENANCE

Josh Bronson¹, Danielle Martin², Jeff Stone³, Jen Christie⁴, and Richard Sniezko⁴

INTRODUCTION

Port-Orford-cedar (*Chamaecyparis lawsoniana*) is endemic to southern Oregon and northern California, where it occurs in a variety of habitats from sea level to about 2,000 m. Port-Orford-cedar (POC) is an ecologically and economically significant tree species. POC is an important component for increasing structural complexity and multi-layered canopy that are key features for high quality habitat for the northern spotted owl. POC is one of the few conifers able to thrive in serpentine soils that support unique plant communities. Top quality logs may bring tens of thousands of dollars in the export market, making POC one of the most valuable softwoods harvested in Oregon today. POC wood is marketed for its strength, durability, and versatility, and is used for paneling, decking, fence posts and fence rails (Betlejewski *et al.* 2011).

Since around 1923, Port-Orford-cedar in the Pacific Northwest has been affected by Phytophthora root disease caused by the introduced pathogen *Phytophthora lateralis*. Because POC is also an important riparian species (where *P. lateralis* thrives); it is at high risk of infection throughout most of its natural range. In the past 50 years, scattered symptomless POC trees have been found growing in infested areas and had apparently escaped or survived infection, suggesting genetic resistance. A systematic resistance testing and breeding program, located at the Forest Service

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Dorena Genetic Resources Center (DGRC), was developed with the goal of producing resistant planting stock suitable for replacing wild native POC killed by the disease (Sniezko *et al.* 2012.)

Recent research has identified individuals and families with high levels of heritable resistance to POC root disease (Sniezko *et al.* 2004, 2012). In addition to *P. lateralis*, POC is also affected by various other native and nonnative pathogens, including a foliage blight caused by *Pseudocercospora thujina* (syn. *Stigmina thujina*), the cause of Stigmina foliage blight. Although this foliage blight is currently of only minor importance in the Pacific Northwest, a changing climate may alter this. Susceptibility to pathogens other than *P. lateralis* could compromise the success of deploying root disease resistant POC operationally. Determining the susceptibility of root disease resistant POC to other pathogens, such as *P. thujina*, is essential for successful management and restoration of POC in areas infested by *P. lateralis*.



Figure 1. *Stigmina* foliage blight on POC at the Tyrrell seed orchard. The clones on the right appear to be unaffected by the disease.

Pseudocercospora thujina was first described from living and dead leaves of western red-cedar (WRC, *Thuja plicata*) in Lane County, Oregon in 1924

(Doty 1982) Hodges (1982) confirmed that *P. thujina* is widely distributed throughout the range of WRC in Oregon, Washington, Idaho and British Columbia on the basis of numerous herbarium samples. The first report of Stigmina foliage blight affecting POC was from an off-site planting in Hawaii (Hodges 1982). *P. thujina* has also been reported as causing defoliation of non-native POC in New Zealand (Dick 1998), Austria and Croatia (Cech and Diminić 2007).

The pathogen has been reported on native POC from near Gasquet, California (Dale *et al.* 1991). In Oregon, *P. thujina* was recently identified on Port-Orford-cedar seedlings at a privately owned bough orchard near Port Orford (Martin 2008). The pathogen has since been found on POC seedlings in the root disease resistance breeding program clone bank at the Bureau of Land Management’s Tyrrell seed orchard near Eugene, Oregon, and at several other out-planting sites in California that were being used to test the long term survival of resistant selections.

MEASUREMENT

Personnel from the DGRC have been maintaining a reserve clone bank of tested POC at the Tyrrell seed orchard since 1999. Almost all of these clones were started from rooted cuttings and grown in a greenhouse before out-planting. In 2014, Richard Sniezko noticed that some of the out-planted trees had symptoms consistent with Stigmina foliage blight (Figure 1). Symptoms included chlorosis and defoliation, primarily in the lower half of affected trees. This was the first time that these types of symptoms were observed at this planting site. Plant pathologists from Oregon State University (Dr. Jeff Stone) and the USDA Forest Service (Josh Bronson) independently identified the causal pathogen as *P. thujina*. It soon became apparent that only some trees were affected, and others not at all. In several cases symptomless trees were located directly adjacent to trees that were heavily defoliated, suggesting genetic variation in susceptibility (Figure 1). All of the trees in the

study area were visually assessed for symptom development in August 2014. Severity was rated on a 0-9 scale, and percent crown dieback was estimated for each tree.

RESULTS

Data from the 2014 assessment are summarized in Table 1. Ninety-one percent of the trees that were assessed were not affected by the pathogen. However, many of those that were affected suffered from severe defoliation. Provenance was a substantial explanatory factor for variation in Stigmina foliage blight incidence. Trees from the California high-elevation, interior sources had the highest incidence. Low levels of incidence were found on trees from breeding zones 340 and 125.

Table 1. Summary of foliage blight incidence in 2014, organized by breeding zone. In many of the sources no foliage blight was detected.

Breeding Zone	# Dead or Missing	# Live	# Foliage Blight	% Foliage Blight	Breeding Zone Location
110	85	328	0	0	Coastal
125	104	508	4	0.79	Coastal
210	7	32	0	0	Coastal
225	20	58	0	0	Coastal
315	1	35	0	0	Interior
325	15	141	0	0	Interior
340	76	384	11	2.86	Interior
350	2	28	0	0	Interior
425	5	25	0	0	Interior
440	8	49	0	0	Interior
450	27	141	23	16.31	Interior
545	16	59	30	50.85	Interior
550	28	131	109	83.21	Interior

DISCUSSION

This disease was not observed on POC at Tyrrell prior to 2014. Since planting, the trees have grown well on the site with very few problems, although some did suffer from animal damage (voles) early on. The sudden occurrence and dramatic defoliation of some clones is perplexing. It is still unclear why this disease has not been seen here before or why it appeared when it did. However, it shows the importance of genetic sentinel plantings

(progeny tests, provenance trials, clone banks) in discerning changes in incidence of pathogen impacts over time, along with potential genetic variation in susceptibility. Permanent installations of this type can provide valuable guides for land managers contemplating reforestation and restoration.

Danielle Martin found similar results concerning the strong association with high-elevation, interior California sources (Martin 2008) on trees in a provenance trial in a coastal site in California (Humboldt Nursery). Her analysis was based on a range-wide planting of POC at the nursery, which was a blocked, replicated planting. The fact that trees from parent trees from particular regions, i.e. interior and higher elevation sites, are more susceptible to *Stigmina* blight suggests that evolution of resistance to *Stigmina* has been more strongly favored in the wetter, milder coastal seed zones. This also suggests that there may be differences in leaf morphology or chemical defenses related to provenance. Further research is required to understand these interactions.

Most foliar pathogens require moisture for infection, sporulation, and dissemination. Climate change will likely cause changes in precipitation and in general, will affect the likelihood of disease development of *Stigmina* foliage blight. The low level of disease noted on most clones could increase depending on changes in climate throughout the range of POC in the wild, leading to substantial economic and ecological losses. Similar to what has been observed in other places; this has implications for off-site plantings as some trees may not be adapted to local variations in climate and disease pressure. Plantings such as these could help guide assisted migration contemplated for some species.

Based on a walkthrough examination in September of 2015, it would appear that the disease has become more severe on the trees that were affected in 2014, and that it is likely present on POC trees throughout the planting site at a very low level. A more thorough measurement is planned for autumn 2015.

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ONGOING MOLECULAR STUDIES OF EUCALYPTUS POWDERY MILDEW IN BRAZIL

N.R. Fonseca¹; L.M.S. Guimarães¹; R.P. Pires¹; N.B. Klopfenstein²; M.-S. Kim³, and A.C. Alfenas¹

INTRODUCTION

Powdery mildew diseases are caused by biotrophic fungi in the Erysiphales. These fungal pathogens are easily observed by the whitish powdery appearance caused by their colonization of the aerial surfaces on living plants (Stadnik & Rivera, 2001) (Figure 1). In Brazil, powdery mildew of *Eucalyptus* spp is increasing under the current nursery production systems, and the causal agents are generally recognized as the anamorphic stage, such as *Oidium* spp or *Oidium eucalypti* Rostrup. (Mucci *et al.*, 1980). Unfortunately, pathogen identification is hampered by the absence of sexual reproductive structures in *Eucalyptus* powdery mildew pathogens of Brazil (Bedendo, 2011). Accurate identification of these powdery mildew fungi is essential for developing disease management practices, such as resistance-breeding and screening programs, which are dependent on the pathogen species or race. In addition, accurate identification of powdery mildew pathogens will enhance chemical control methods, because different species may respond differently to various fungicides that have specific modes of action.

OBJECTIVES

The main objectives of this project are to: i) Identify the powdery mildew pathogens that infect *Eucalyptus* in different regions of Brazil by molecular and morphological characterization; ii) Generate transcriptome sequencing data of *Eucalyptus* powdery mildew; and iii) Identify

genetic markers from transcriptome sequences that can be used to analyze the genetic diversity and population structure among isolates of *Eucalyptus* powdery mildew pathogens.

MATERIAL AND METHODS

Eucalyptus powdery mildew specimens were collected from mini-clonal hedges in greenhouses from several clonal *Eucalyptus* nurseries in five states of Brazil (Figure 2). For DNA-based identification, total cellular DNA was extracted from mycelia and conidia using a Chelex[®] method (Walsh *et al.* 1991). ITS and 28S rDNA (LSU) regions were amplified by PCR and sequenced using the primers ITS5/ITS4 and PM3/TW14, respectively. Resulting sequences were aligned with homologous sequences available on GenBank. Phylogenetic trees were obtained using Bayesian analysis.

For morphology-based identification, mycelia and conidia from infected leaf surfaces were scraped into a drop of lactic acid on a glass slide for light microscopy. Morphological characters, such as size and shape of conidia; presence or absence of fibrosin bodies; nature of conidiogenesis; characteristics of the conidiophore (e.g., size and shape of the foot cell); and hyphal morphology were observed and recorded. Transcriptome studies are in progress to develop genetic markers for use in population genetic analyses or identifying genes involved in host-plant infection and environmental interactions.

PRELIMINARY RESULTS

Of 82 samples of powdery mildew pathogens collected from *Eucalyptus* plants, 48 samples yielded sufficient DNA for PCR, which resulted in 40 sequences of ITS rDNA and 48 sequences of 28S rDNA. Searches on Blastn

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(<http://blast.ncbi.nlm.nih.gov>) revealed that all isolate sequences were very similar to species within the genus *Podosphaera* for both genetic regions. For phylogenetic analysis, the sequences were aligned with 33 ITS rDNA sequences of *Podosphaera* spp available on GenBank.



Figure 1. A) *Eucalyptus* nursery in State of Minas Gerais, Brazil, with plants showings powdery mildew signs and symptoms; B) Plants highly infected with powdery mildew pathogen; and C) *Eucalyptus* leaf with mycelia and conidia of powdery mildew pathogen.

Results of phylogenetic analyses for both DNA regions showed that all sequences generated in this study were comprised within a single clade of *Podosphaera pannosa*, supported by high posterior probability (97%) (28S rDNA results not shown) (Figure 3). Phylogenetic and sequence analysis revealed that all pathogen isolates of *Eucalyptus* powdery mildew possessed identical (or nearly identical) sequences for the ITS and 28S rDNA regions. Identical rDNA sequences could be

indicative of a clonal population structure, perhaps attributable to lacking sexual reproduction in tropical regions or perhaps reflecting a recent introduction of this pathogen to Brazil. Thus, continued studies using other genetic markers are needed to confirm the population structure of *Eucalyptus* powdery mildew pathogens in Brazil



Figure 2. Collection locations in Brazil (Font: Google Maps).

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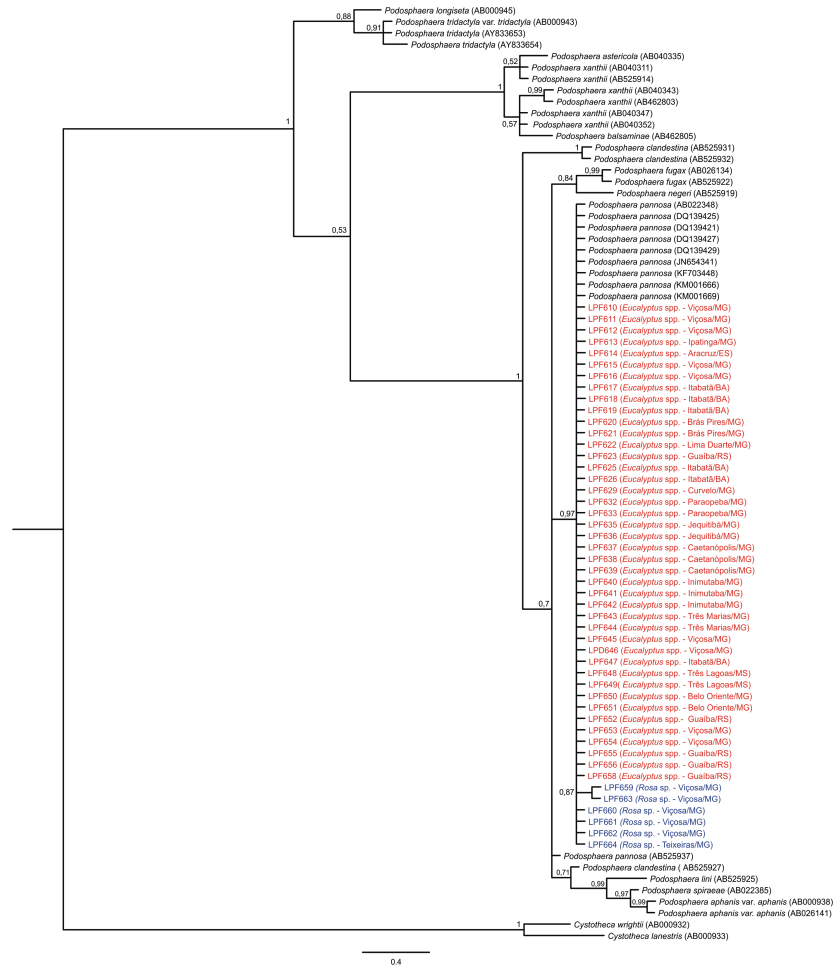


Figure 3. ITS sequence-based Bayesian phylogenetic analysis of powdery mildew pathogens of *Eucalyptus* spp (depicted in red). Posterior probability support percentages are indicated at the branch nodes.

BIOCLIMATIC MODELING PREDICTS POTENTIAL DISTRIBUTION OF *ARMILLARIA SOLIDIPIPES* AND *PSEUDOTSUGA MENZIESII* (DOUGLAS-FIR) UNDER CONTEMPORARY AND CHANGING CLIMATES IN THE INTERIOR WESTERN USA

J.W. Hanna¹, M.V. Warwell¹, H. Maffei², M.L. Fairweather³, J.T. Blodgett⁴, P.J. Zambino⁵, J. Worrall⁶, K.S. Burns⁷, J.J. Jacobs⁸, S.M. Ashiglar¹, J.E. Lundquist⁹, M.-S. Kim¹⁰, A.L. Ross-Davis¹, C. Hoffman¹¹, R. Mathiasen¹¹, R. Hofstetter¹¹, J.D. Shaw¹², E.W.I. Pitman¹, E.V. Nelson¹¹, G.I. McDonald¹, M.R. Cleary¹³, S. Brar¹⁴, B.A. Richardson¹⁵, and N.B. Klopfenstein¹

INTRODUCTION

Pseudotsuga menziesii (Douglas-fir) is a dominant component of forest stands in much of western North America. It is an important tree to the timber industry, yielding more timber than any other species in North America. It is also extremely important for wildlife as habitat and food. Many small birds and mammals feed on its seeds. *Armillaria solidipes* [pending vote to conserve *A. ostoyae* (Redhead *et al.* 2011)] is also a key component of forest stands throughout much of western North America (Hanna 2005). It is an aggressive pathogen of conifers causing tree mortality and growth loss (Cruickshank 2000). In particular, *P. menziesii* (and specifically the inland subspecies) is one species along with *Abies grandis* (grand fir) and *A. lasiocarpa* (subalpine fir) that can have the highest rates of susceptibility to *A.*

solidipes (McDonald *et al.* 1987a). These species have a long history of co-evolution and co-distribution over millions of years. Under the host/stress/saprogen concept, disease develops when these secondary pathogens already on-site invade host tissue after environmental stress (Houston 1992). These stressors include climate, human disturbance, arthropod pests, and other pathogens. Such diseases are believed to increase in severity and prevalence under climate change as trees become progressively maladapted to their environments (Kliejunas *et al.* 2009). In this study, we use DNA-based methods to confirm species identification and utilize location-specific climate data for bioclimatic modeling to predict where *A. solidipes* is likely to occur and cause increased disease pressure on *P. menziesii* under changing climatic conditions.

OBJECTIVES

The objectives of this study are to 1) determine suitable climate space (potential distribution/realized climate niche) for *Armillaria solidipes* across inland North America for contemporary climate and projected climate for the average of years 2061-2080; 2) determine suitable climate space (potential distribution/realized climate niche) for *P. menziesii* across inland North America for contemporary climate and projected climate for the average of years 2061-2080; and 3) make comparisons between the prediction models of the pathogen and host to examine potential maladaptation pressures caused by climate change.

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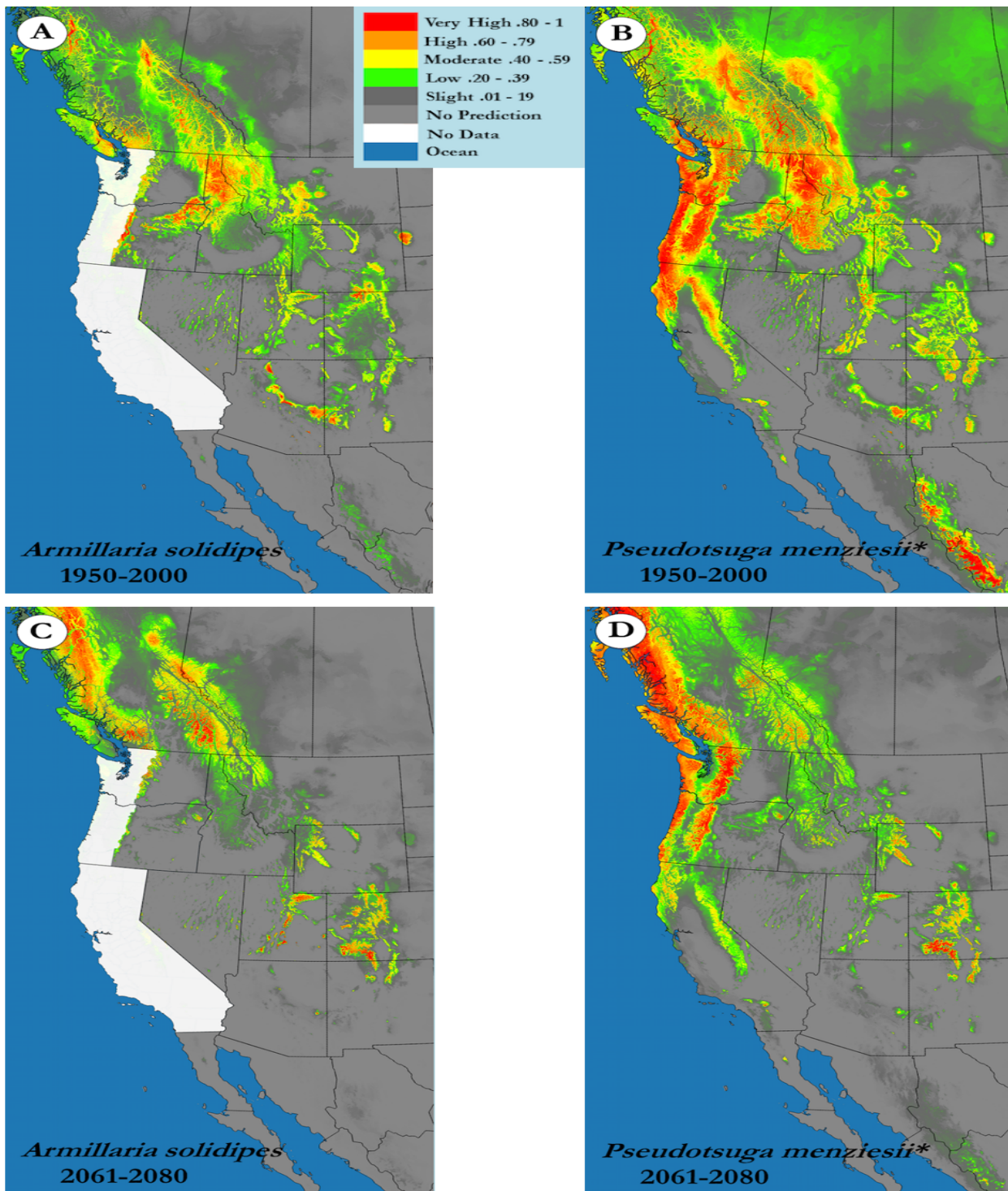


Figure 1. Maximum Entropy bioclimate model of suitable climate space (potential distribution) for **A:** predicted contemporary (1950-2000) *Armillaria solidipes*, **B:** predicted contemporary (1950-2000) *Pseudotsuga menziesii*, **C:** predicted for the years 2061-2080 *Armillaria solidipes*, **D:** predicted for the years 2061-2080 *Pseudotsuga menziesii*. Areas of lowest to highest suitability of climate space are indicated by dark grey, light green, yellow, orange, and red. *Predicted climate suitability of *Pseudotsuga menziesii* based solely on U.S.A. locations. Therefore, Canadian and Mexican predictions are less accurate.

METHODS

Climate-based, species-distribution models using Maximum Entropy (MaxEnt) were created using the techniques described in Phillips *et al.* 2006 and Klopfenstein *et al.* 2009; Figure 1, A - *A. solidipes* contemporary (1950-2000) predicted suitable climate; B - *P. menziesii* contemporary (1950-2000) predicted suitable climate, C - *A. solidipes* year 2061-2080 predicted suitable climate; and D - *P. menziesii* year 2061-2080 predicted suitable climate. The models used 19 bioclimatic variables (e.g., annual mean temperature, maximum temperature of warmest month, annual precipitation, precipitation of wettest month, precipitation of coldest quarter, etc.) in two sets of interpolation grids (ca. 1-km² resolution) from worldclim.org (Hijmans 2005). One set contained environmental data for contemporary climate (based on data from 1950-2000) and the other had climate projection grids for the years 2061-2080 based on data from the Intergovernmental Panel on Climate Change/Coupled Model Intercomparison Project Phase 5 (IPCC/CMIP5). For the 2061-2080 predictions we used the representative concentration pathway 8.5 (RCP8.5), which represents a “business-as-usual” continued rise in CO₂ greenhouse-gas scenario and the global circulation model (GCM) HadGEM2-ES (Collins *et al.* 2008; Riahi *et al.* 2011).

To allow calculations in suitability models, input data for MaxEnt consisted of SWD “samples with data” files for each species that linked climate variable values for each of the 19 bioclimatic variables with geographic coordinates (presence point localities). *Armillaria solidipes* point locations were collected from previous studies of distribution and ecology from the states/provinces of Washington, Oregon, Idaho, Montana, Utah, Wyoming, Arizona, Colorado, New Mexico, British Columbia, and Chihuahua (McDonald *et al.* 1987b; Shaw 1989; Omdal *et al.* 1995; McDonald *et al.* 1998; Kim 1999; Kim *et al.* 2000; Ferguson *et al.* 2003; Worrall *et al.* 2004; Hanna 2005; Hanna *et al.* 2007; Hanna *et al.* 2008a; Hanna *et al.* 2008b; Blodgett and Lundquist 2011; McDonald *et*

al. 2011; Klopfenstein *et al.* 2012; Hanna *et al.* 2014; Hoffman *et al.* 2014; Blodgett *et al.* 2015; and Hanna *et al.* unpublished data). From these studies, *A. solidipes* isolates were recorded from 378 distinct locations throughout inland western North America. The isolates were confirmed as *A. solidipes* using DNA-based species identification at the Forestry Science Laboratory, Rocky Mountain Research Station in Moscow, Idaho using similar techniques as described by Kim *et al.* (2006), Ross-Davis *et al.* (2012), and/or Elías-Román *et al.* (2013). Many isolates were also identified by somatic incompatibly testing and/or basidiocarp morphology. For *P. menziesii*, a total of 12,152 locations were used from selected ‘fuzzy’ coordinates obtained from Forest Inventory Analyses data (please refer to Rehfeldt *et al.* 2014 for information about how these points were selected) within the continental USA. Locations from Canada and Mexico were not included. MaxEnt also uses SWD files of background locations or “pseudo-absences” to “train” the models. For the *A. solidipes* model, background points were created from 10,000 randomly selected locations within the geographic range of the collected isolates. For the *P. menziesii* model, 292,639 actual absence point locations were used as background data. MaxEnt’s logistic output (an index of probability from 0 to 1) was chosen for easier conceptualization compared to MaxEnt’s raw exponential model.

Armillaria solidipes predictions for coastal areas of Oregon and Washington are not shown (white areas) due to lacking occurrence records for this area, which indicates that additional distribution data for *A. solidipes* are needed for these areas (see below). California is also not shown (white) because *A. solidipes* has not been recorded in California (Baumgartner and Rizzo 2001).

RESULTS, DISCUSSION, AND FUTURE WORK

Two results are obvious from the models (Figure 1). Predicted suitable climate for *A. solidipes* and *P. menziesii* are highly correlated and the predicted change in climate suitability moves dramatically

northward (and toward higher elevations) for year 2070 using the RCP8.5 “business as usual” scenario. MaxEnt is better suited for presence only data and over predicts *P. menziesii* compared to a presence/absence model (see Rehfeldt *et al.* 2014 for a climate niche model of *P. menziesii* based on a Random Forests classification algorithm). Nevertheless, absence data for *Armillaria* are difficult to obtain and if available difficult to confirm with certainty. Thus, we used MaxEnt to compare both pathogen and host using the same modeling parameters. These models do not take adaptation into account, but it is presumed unlikely that either of these relatively long-lived species can adapt dramatically within such a short time. A plausible hypothesis, shared by others (Kliejunas *et al.* 2009; Sturrock *et al.* 2011) is as hosts become maladapted (stressed) due to changing climates they will have a higher likelihood of susceptibility to many pathogens. While suitable climate is predicted to decrease in many areas of the inland west for *A. solidipes*, the pathogen will likely persist in many areas of the interior west, where will likely contribute significantly to tree mortality and growth loss it where maladapted hosts remain.

A number of projects could be implemented to improve the predictive capabilities of these *A. solidipes* prediction models, including: 1) removal of bias by using bias grids and/or collection of additional isolates from under-represented areas, 2) adding additional predictive variables (i.e. soil types, solar radiation, and/or predictions of other *Armillaria* spp), and 3) obtaining population-level data to run independent predictions based on separate populations.

While *A. solidipes* is well known to exist west of the Cascade Range, our models are unreliable sources of prediction for this area because we lack knowledge of specific geographic occurrences west of the Cascade Range. Furthermore, it is well established that climate and ecological behavior of *A. solidipes* is different in regions west of the Cascades compared with the interior western North America. However, Douglas-fir is one of the most susceptible tree species east of the Cascade Range (Morrison 1981; Robinson and Morrison 2001), but the coastal variety of Douglas-fir seldom

succumbs to infection (Johnson *et al.* 1972; Robinson and Morrison 2001). To address these differences, population-level data are needed for both host and pathogen to refine climate-based models. Genetic variation within *A. solidipes* has already been demonstrated, but only on the basis of a few genes (Hanna 2005; Hanna *et al.* 2007; Hanna *et al.* 2012). It is now feasible to incorporate whole-genome variation data to better determine population structure.

The climate-based modeling methods developed from this project can also be used to model other important forest pathogens and examine the potential for invasive species to occupy new geographic areas under contemporary and future climates.

Predicting the distribution of fungal species relies strongly on DNA-based identification methods to verify fungal species and/or populations. Furthermore, fungal taxonomy continue to change over time. Thus, living specimens are important to obtain the quantity and quality of DNA needed for fungal identification and population analyses. The availability of living cultures allow us to update/verify species identification of cultures collected in the past. For example, many of the isolates and data used to complete this study are from the USDA Forest Service-RMRS forest fungi collection located in Moscow, Idaho. This collection houses over 15,000 living specimens and associated collection data including many *Armillaria* specimens collected over the past 30 years. Future support is needed to maintain such invaluable collections, in which fungal isolates are associated with a specific time, place, and climate. These collections are critical to understanding species distribution changes under climate change (Ashiglar *et al.* 2014).

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AN INTEGRATED TAXONOMIC APPROACH TO SURVEY ARMILLARIA IN IRAN

Saeideh Jafarpour¹, Khalil-Berdi Fotouhifar¹, Mohammad Javan-Nikkhah¹, Mohhamad Reza Asef², Anthony S Davis³, Amy L Ross-Davis⁴, John W Hanna⁴, Simona Margaritescu⁵, Jean-Marc Moncalvo⁶, and Ned B Klopfenstein⁴

INTRODUCTION

Iran's most valuable forests are located on the coast of the Caspian Sea and cover 1.85 million ha in the northern region of the Alborz mountain range, which is the highest mountain range in the Middle East. Dense forests cover two major provinces, Gilan and Mazandaran; however, less than 10% of Iran is forested. These forests comprise temperate, deciduous, broad-leaved tree species. Conifers are usually absent in Iranian forests, with only a few relics of coniferous species remaining.

Armillaria root disease can cause significant damage in Iranian forests and it is widely distributed throughout these forests. *Armillaria mellea* is a well-known cause of root disease of Caucasian or Persian oak (*Quercus macranthera*) within the Hatam Baigh forest in northwestern Iran (Davari *et al.* 2005). Based on the biological species concept (Korhonen 1978), a previous study of *Armillaria* spp in Iran showed the existence of four intersterility groups, which represented *A. mellea*, *A. cepistipes*, *A. gallica* and *A. borealis* (Asef *et al.* 2003). However, the distribution of *Armillaria* spp in Iran remains largely undocumented a better understanding of

Armillaria distribution is needed for disease management and comparisons with other regions. In recent years, the utility of DNA sequence-based identification has been demonstrated for *Armillaria* spp, and translation elongation factor 1 α (*tef-1 α*) gene sequences have been especially useful for phylogenetic analysis to differentiate closely related *Armillaria* spp (e.g., Maphosa *et al.* 2006; Hasegawa *et al.* 2010; Ross-Davis *et al.* 2012). Previously, no DNA sequence data were available for validating *Armillaria* spp in Iran. The objective of this study is to identify *Armillaria* spp from Iran using integrated taxonomic methods based on basidiocarp morphology, interfertility, and DNA sequences.

MATERIALS AND METHODS

We are basing species identification of *Armillaria* in Iran on basidiocarp morphology, interfertility (biological species), and phylogenetic analyses of DNA sequences from the *tef-1 α* gene. Fresh and dried basidiocarps were used to determine classical morphological characteristics DNA sequences (e.g., *tef-1 α*) from a set of European biological tester strain cultures of annulate *Armillaria* spp, obtained from Kari Korhonen (Finland) and Nenad Keca (Serbia), were also included in the analyses. Additional *tef-1 α* sequences of North American *Armillaria* spp were obtained from GenBank. Sequences were aligned using the MAFFT software (Kato & Standley 2013) and a phylogenetic tree was constructed using maximum-likelihood in Garli 2.2 (Bazin *et al.* 2014). Interfertility tests from basidiospore-derived cultures between Iranian collections and European biological species testers are ongoing.

In: Ramsey, A. & P. Palacios (Comps). Proceedings of the 63rd Annual Western International Forest Disease Work Conference; 2015 Sept. 21-15; Newport, OR. ¹Department of Plant Protection, Faculty of Agricultural Science and Engineering, College of Agriculture and Natural Resources, University of Tehran, Karaj, Iran. ²Department of Botany, Research Institute of Plant Protection, Tehran, Iran. ³Department of Forest, Rangeland, and Fire Sciences, College of Natural Resources, University of Idaho, Moscow, Idaho. ⁴USDA Forest Service, Rocky Mountain Research Station, Moscow, Idaho. ⁵Department of Natural History, Royal Ontario Museum, Toronto, Canada. ⁶Department of Natural History, Royal Ontario Museum and Department of Ecology and Evolutionary Biology, University of Toronto, Toronto, Canada.



Figure 1A. Sampling Armillaria in Iranian forests.

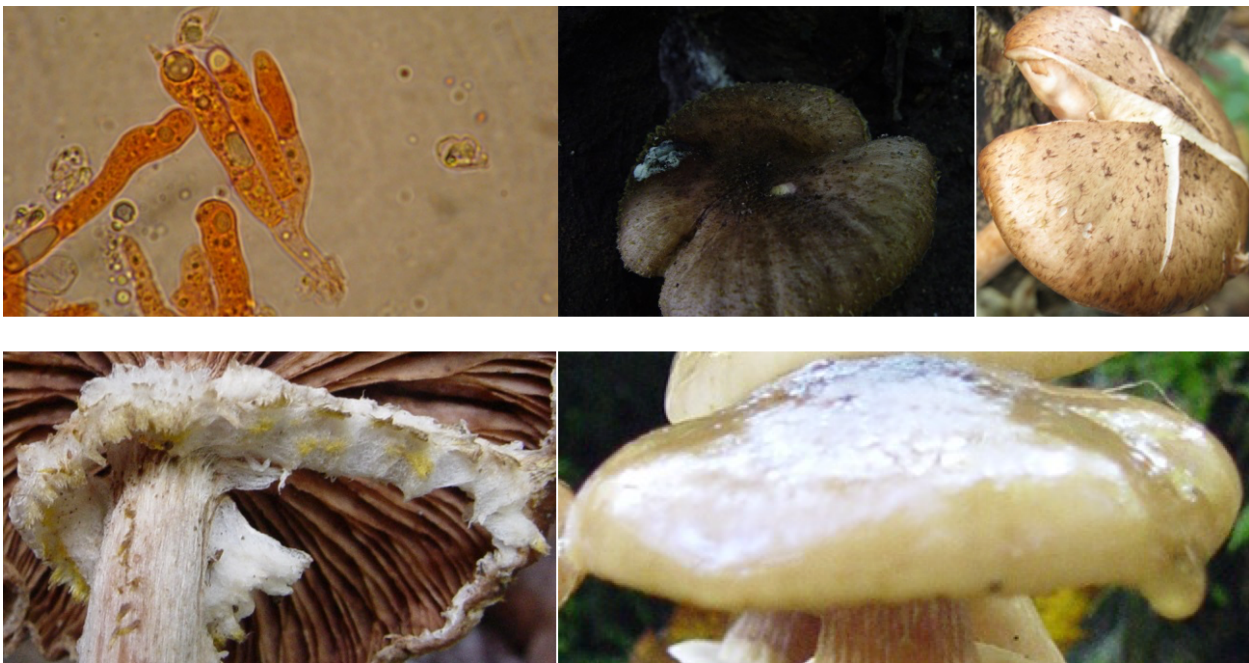


Figure 1B. Morphological study of Armillaria.

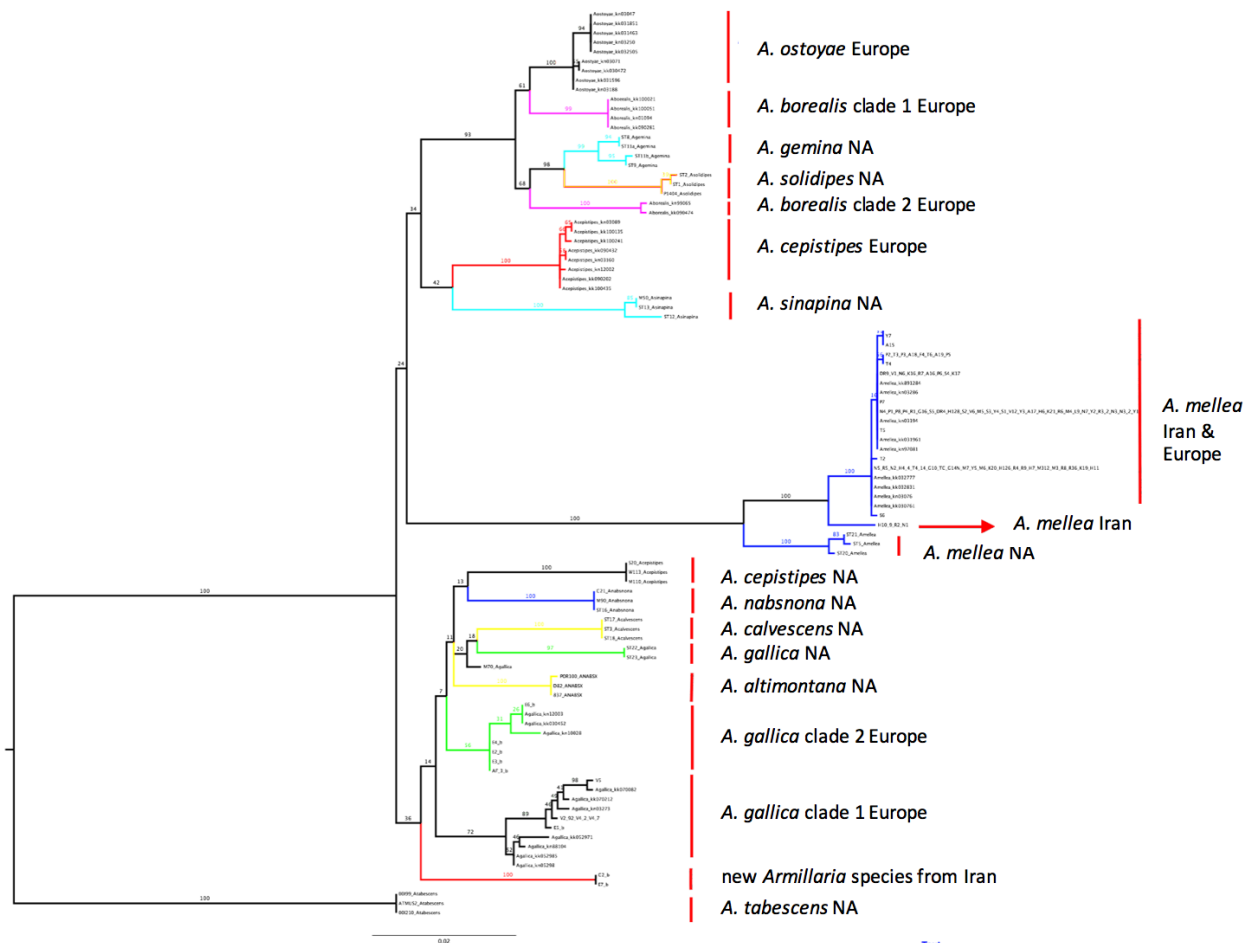


Figure 2. Preliminary phylogenetic tree of global *Armillaria* spp constructed in Garli 2.2. (NA = North America).

RESULTS AND DISCUSSION

In this study, approximately 100 *Armillaria* basidiocarps were collected from 11 different forest sites in northern Iran (Figure 1A and 1B). Based on morphology and preliminary phylogenetic analyses, most samples are associated with *A. mellea* and *A. gallica* complexes (Figure 2). In Iran, *A. mellea* is associated with wide-spread tree mortality and windthrow, which suggests that *A. mellea* is a primary pathogen in Iranian forests. Interestingly, three *A. mellea* isolates generated *tef-1a* sequences containing 15 different variable sites that separated these isolates into a distinct subclade

within the *A. mellea* complex. Although the *A. mellea* complex is widely distributed around the northern hemisphere, phylogenetic analysis shows that North American *A. mellea* and Iranian *A. mellea* reside in distinct clades. Further studies are needed to resolve the taxonomy of the polyphyletic *A. mellea* and *A. gallica* complexes.

A better understanding of the taxonomy and the global distribution of *Armillaria* spp will provide information on the potential invasiveness and potential impacts of climate change on *Armillaria* root disease.

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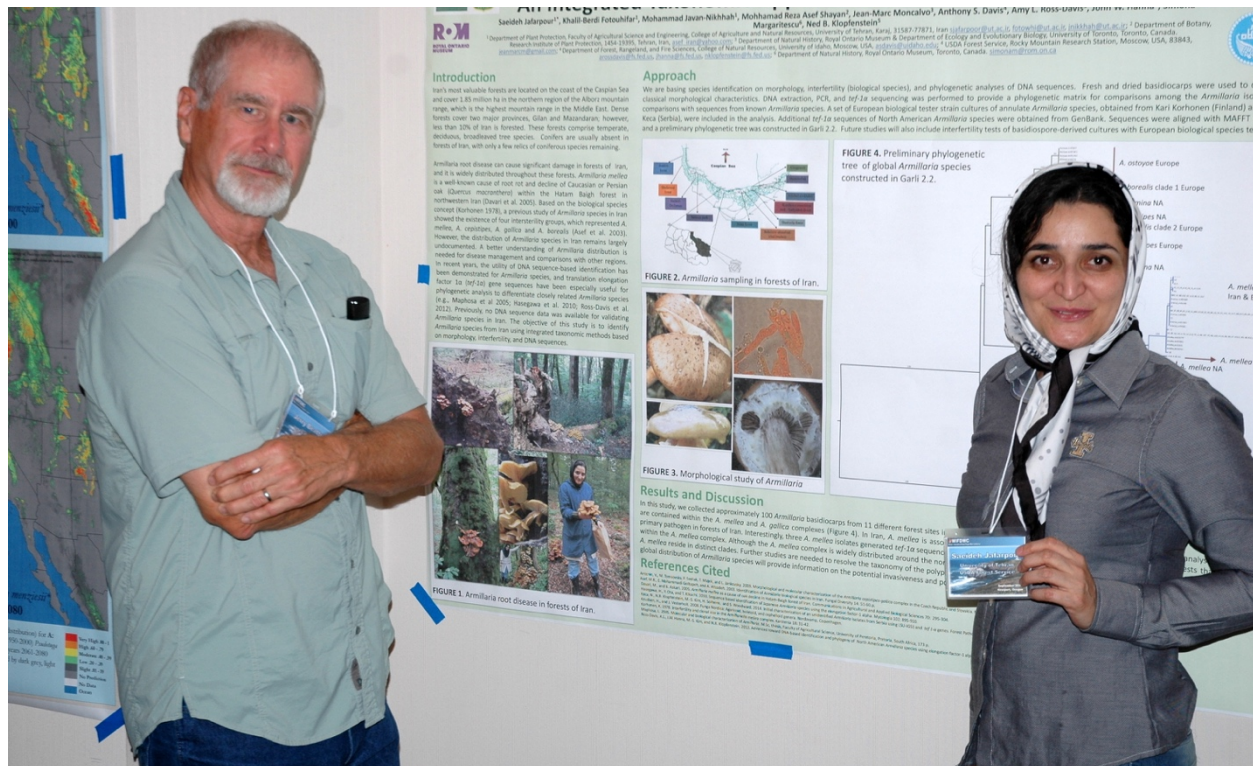
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CAN METAGENETIC STUDIES OF SOIL MICROBIAL COMMUNITIES PROVIDE INSIGHTS TOWARD DEVELOPING NOVEL MANAGEMENT APPROACHES FOR ARMILLARIA ROOT DISEASE?

Mee-Sook Kim¹, Amy L. Ross-Davis², Jane E. Stewart³, John W. Hanna², Marcus V. Warwell², Paul J. Zambino⁴, Christy Cleaver⁴, Geral I. McDonald², Debbie S. Page-Dumroese², Bruce Moltzan⁵, and Ned B. Klopfenstein²

Armillaria Root Disease

Armillaria root diseases are among the most damaging and broadly distributed group of forest diseases in the world (Lockman *et al. in press*). Armillaria root disease is typically more severe in highly susceptible tree species and in trees that are maladapted due to rapidly changing climatic conditions (Ayres and Lombardero 2000, Kliejunas *et al.* 2009, Sturrock *et al.* 2011). Unfortunately, Armillaria root disease is notoriously difficult to manage in forests. Because Armillaria root disease impacts are dependent on complex ecological interactions among the host, soil microbial community, and environment, we propose the development of metagenetic tools to examine soil interactions that suppress (or enhance) Armillaria root disease. Tools to identify these interactions are paramount to develop effective management approaches for Armillaria root disease.

Potential Application of Metagenetics in Disease Management

Several studies have previously demonstrated the potential biological control of Armillaria root disease (e.g., Reaves *et al.* 1990, Raziq 2000, DeLong *et al.* 2002, Chapman *et al.* 2004, Raziq

and Fox 2005). Previous evaluations of biological control have typically focused on myco-parasitic fungi (e.g., *Trichoderma* spp) or saprophytic wood-decay fungi (e.g., *Ganoderma* spp, *Hypholoma* spp). However, they focus on only one or a few microbes, and applications are restricted by the need for massive inoculum. More importantly, inoculations with biocontrol agents must consider the risks of introducing biological organisms in nature, and navigate a lengthy and expensive process to obtain federal licensing and permits for applications within even small areas. Metagenomics and metagenetics have illuminated our understanding of how microbial communities relate to health and disease. An alternative approach to developing biological control methods for Armillaria root disease is based on the natural microbial community of the soil, which can be assessed using recently developed metagenetic technology. With this approach, 1,000s to 10,000s of microbial taxa are identified or assigned to Operational Taxonomic Units from a 1-g sample of soil (or other substrate) with accompanying environmental and soil properties. Through replication, key microbial taxa associated with suppressed disease activity are identified, along with the environmental factors that favor those microbial taxa. Subsequent tests can use management methods that favor natural microbial communities for biological control of Armillaria root disease.

Approaches

Metagenetic tools can be used to characterize soil microbial communities that may suppress Armillaria root disease. Identifying environmental factors, such as soil temperature, moisture, pH, and

In: Ramsey, A. & P. Palacios (Comps). Proceedings of the 63rd Annual Western International Forest Disease Work Conference; 2015 Sept. 21-15; Newport, OR. ¹Department of Forestry, Environment and Systems, Kookmin University, Seoul, South Korea. ²USDA Forest Service-Rocky Mountain Research Station, Moscow, Idaho. ³Department of Bioagricultural Sciences and Pest Management, Colorado State University, Ft. Collins, Colorado. ⁴USDA Forest Service, Forest Health Protection (FHP), Region 1, Coeur d'Alene, Idaho. ⁵USDA Forest Service, FHP, Washington, D.C.

organic matter, associated with these communities can inform effective management of *Armillaria* root disease. Similar tools have been developed to characterize root disease-suppressive soils in agricultural systems, and identify appropriate disease management practices (e.g., Mendes *et al.* 2011, Damon *et al.* 2012, Bonito *et al.* 2014, Penton *et al.* 2014, Qiu *et al.* 2014, Štursová *et al.* 2014).

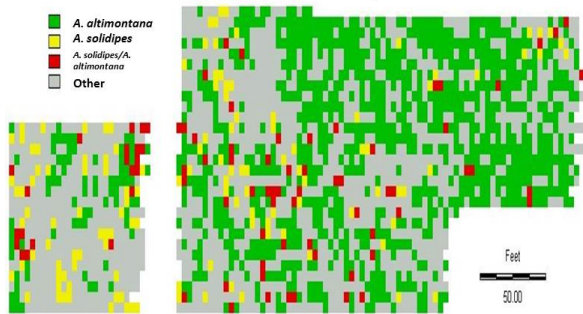


Figure 1. Does *Armillaria altimontana* help protect trees from *A. solidipes* (formerly known as *A. ostoyae*), or does *A. altimontana* reflect environmental conditions that are unfavorable for *A. solidipes*? The map depicts locations of *A. altimontana*, *A. solidipes*, and mixed *A. solidipes* and *A. altimontana* associated with a western white pine (*Pinus monticola*) provenance plantation at the Ida Creek site (Priest River Experimental Forest, northern Idaho). For this proposed study, sampling will focus on individual genets of *A. altimontana* (green areas) and *A. solidipes* (yellow areas).

Proposed Methods

The proposed tool will be assessed on six plots of each *A. altimontana* and *A. solidipes* for a total of 12 plots established within the Priest River Experimental Forest in northern Idaho (Figure 1). Total DNA will be extracted from each soil sample. Double-barcoded and pooled PCR products will be sequenced for multiple amplicons to assess the eukaryotic and prokaryotic microbial communities. Forest soil microbial communities will be analyzed to 1) determine differences among subplots, 2)

assess how differences relate to the soil properties (chemical and physical), and 3) examine how communities compare between plots associated with saprophytic *A. altimontana* and pathogenic *A. solidipes*.

Anticipated Outcomes

This project will develop metagenetic tools to characterize soil microbial communities and soil properties/environmental factors related to *Armillaria* root disease (i.e., trees colonized by saprophytic *A. altimontana* and pathogenic *A. solidipes*). This study will contribute tools for developing management practices that suppress *Armillaria* root disease and enhance forest resilience.

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MOLECULAR GENETIC CHARACTERIZATION OF THE KOA-WILT PATHOGEN (*FUSARIUM OXYSPORUM*): APPLICATION OF MOLECULAR GENETIC TOOLS TOWARD IMPROVING KOA RESTORATION IN HAWAI'I

Mee-Sook Kim¹, Jane E. Stewart², Nicklos Dudley³, John Dobbs³, Tyler Jones³, Phil G. Cannon⁴, Robert L. James⁵, R. Kasten Dumroese⁶, and Ned B. Klopfenstein⁶

INTRODUCTION

Several forest diseases are causing serious threats to the native Hawaiian forest. Among them, koa-wilt disease (caused by *Fusarium oxysporum*) is damaging to native populations of koa (*Acacia koa*), and it also hinders koa restoration/reforestation. Because *F. oxysporum* likely represents a complex of species with distinct pathogenic activities, more detailed characterization is needed to better understand the *F. oxysporum* that is associated with koa wilt. Such data would allow assessments of genetic diversity and population structure, while also providing inferences as to whether the pathogen is indigenous and/or potential modes of spread. DNA-based characterization of the koa-wilt pathogen will allow assessments of pathogen populations to inform koa restoration efforts, and assist resistance-breeding and screening programs for koa.

Importance of Koa (*Acacia Koa*) In Hawai'i

(A) Ecological importance

- Provides habitat for many native bird and insect species.

In: Ramsey, A. & P. Palacios (Comps). Proceedings of the 63rd Annual Western International Forest Disease Work Conference; 2015 Sept. 21-15; Newport, OR. ¹Kookmin University, Department of Forestry, Environment and Systems, Seoul, South Korea. ²Colorado State University, Department of Bioagricultural Sciences and Pest Management, Fort Collins, Colorado ³Hawaii Agriculture Research Center, Maunawili Research Station, Kailua, HI ⁴USDA Forest Service, Forest Health Protection, Region 5, Vallejo, CALIFORNIA ⁵Plant Disease Consulting, Vancouver, Washington ⁶USDA Forest Service, Rocky Mountain Research Station, Moscow, Idaho.

- Predominate nitrogen-fixing, native tree species.
- Critical role in watershed health and rehabilitation.
- Dominant tree species in many native ecosystems.

(B) Cultural importance

- Native Hawaiian's primary wood for canoes.
- Tannins from bark used as red dye for kapa cloth.
- Integral part of many Hawaiian ceremonies.

(C) Economic importance

- Koa represents 90% of the Hawaiian Forest Products Industry.
- Valued at \$30 million annually (2001).
- Harvested primarily from native forests.
- Koa is the preferred wood of the Hawaiian Specialty Hardwood Market.
- Koa is one of the most valuable woods in the world, with prices exceeding \$125 per board foot.

The Problem – Koa Wilt Disease

- Koa wilt disease causes high rates of mortality in koa plantings.
- Capable of killing trees of all ages in native forests.
- Causal agent of koa wilt disease - *Fusarium oxysporum* f. species *koa*.
 - ✓ First described in koa by Gardner in 1980.
 - ✓ Species infects wide range of plants, but non-pathogenic and pathogenic isolates cannot be distinguished morphologically.
 - ✓ Little information is available on genetic characterization of koa wilt pathogen.

A)



B)



Figure 1 (A, B) Koa wilt disease caused by *Fusarium oxysporum*.

Molecular Methods to Characterize Koa Wilt Pathogen

The Hawaii Agriculture Research Center collected many isolates of koa wilt pathogen and we will visit more sites to collect additional samples for

genetic characterization. Because *F. oxysporum* likely represents a complex of species with distinct pathogenic activities, more detailed characterization is needed to better understand the *F. oxysporum* “subspecies” (phylogenetic clade) that is associated with koa wilt. Initial characterization will be accomplished with phylogenetic analysis of multiple gene regions and Restriction Associated DNA sequencing (RAD-seq), which will be used to further characterize the koa wilt pathogen populations in Hawai‘i. Such data would allow assessments of genetic diversity and population structure, while also providing inferences as to whether the pathogen is indigenous and/or potential modes of spread.

PRELIMINARY RESULTS

- A total of 24 *Fusarium* spp isolates (indicated in red; previously identified as *F. oxysporum* based on morphology) collected from diverse substrate associated with diseased and healthy koa in Hawai‘i. All the isolates were sequenced for the translation elongation factor 1 α (*tef-1 α*) gene.
- All the isolates clustered together except three isolates (Foxy85 – *F. commune* and Foxy1 and Foxy80 – *F. proliferatum*). Some *Fusarium* isolates went through pathogenicity tests (red dots - highly virulent isolates vs. green dots non-pathogenic isolates) (Figure 2).
- Three *Fusarium* spp identified by *tef-1 α* were associated with koa wilt. We plan to 1) collect more *Fusarium* spp isolates associated with koa wilt across Hawai‘i; 2) identify isolate at the species and/or subspecies level; 3) conduct pathogenicity tests of selected isolates from each species/subspecies; 4) develop diagnostic primers for detecting pathogenic *F. oxysporum* f. species *koae*; 5) assess genetic diversity and population structure of *F. oxysporum* f. species *koae* using RAD-seq; and 5) provide a guidance to koa wilt breeding resistance program based on results from this project.

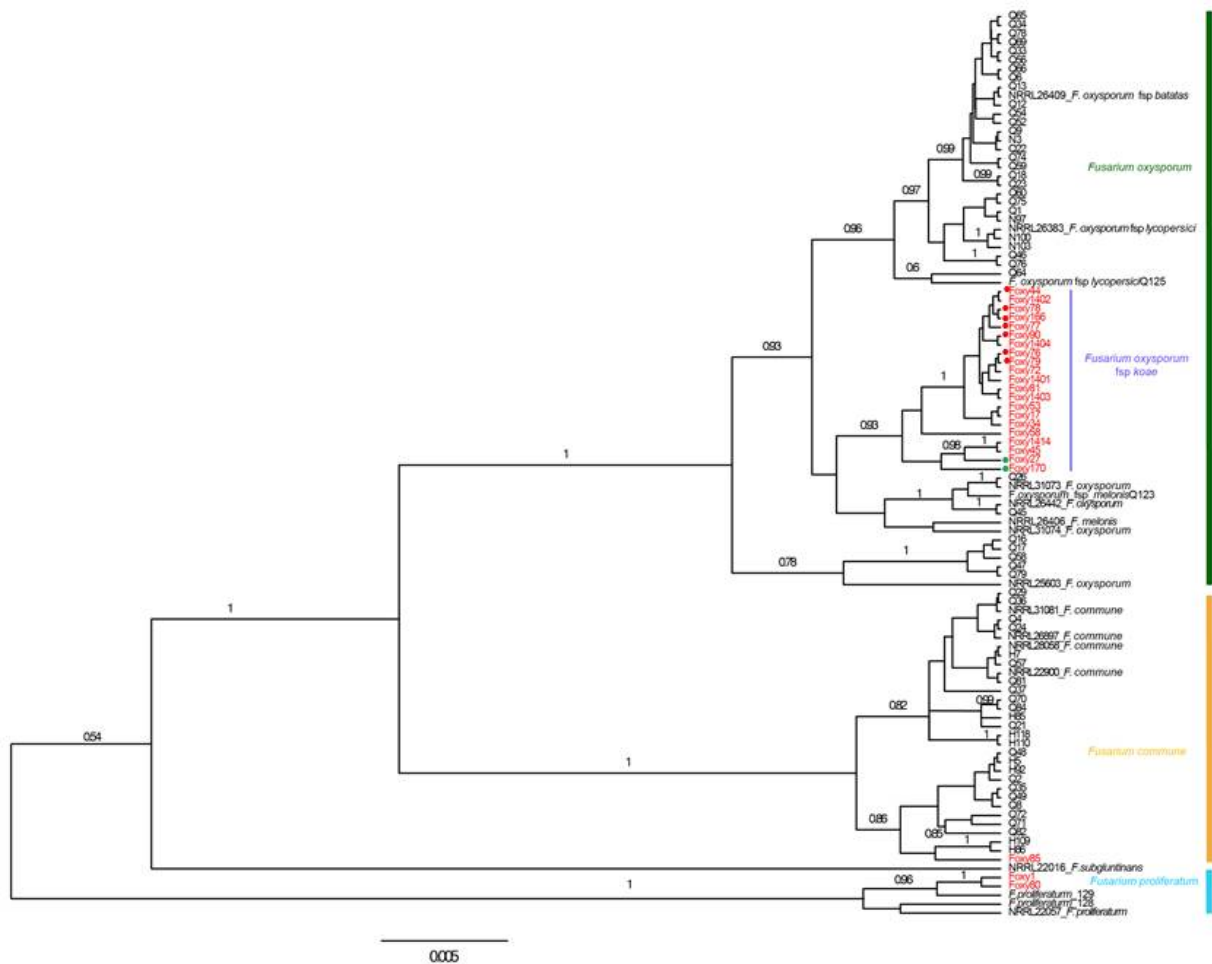


Figure 2. Consensus phylogeny of coalescence-based Bayesian analyses estimated in BEAST under the strict clock with a GTR model of substitution on the translation elongation factor 1a gene consensus (50% strict) sequences of 24 *Fusarium* spp isolates (colored in red). Posterior support values >0.5 are indicated at the nodes.

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A PRELIMINARY BIOCLIMATIC APPROACH TO PREDICTING POTENTIAL DISTRIBUTION OF *Phellinus noxius* AND GEOGRAPHICAL AREAS AT RISK FROM INVASION

Ned B. Klopfenstein¹, Eric W. I. Pitman¹, John W. Hanna¹, Phil G. Cannon², Jane E. Stewart³, Norio Sashashi⁴, Yuko Ota⁴, Tsutomu Hattori⁴, Mitsuteru Akiba⁴, Louise Shuey⁵, Robert L. Schlub⁶, Fred Brooks⁷, Ndeme Atibalentia⁸, Alvin M.C. Tang^{9,10}, Regent Y. C. Lam^{9,10}, Mike W. K. Leung⁹, L. M. Chu¹⁰, H. S. Kwan¹⁰, Mohd Farid bin Ahmad¹¹, Su See Lee¹¹, Hsin-Han Lee¹², Jyh-Nong Tsai¹³, Yu-Ching Huang¹², Chia-Lin Chung¹², Ruey-Fen Liou¹², and Mee-Sook Kim¹⁴

INTRODUCTION

Phellinus noxius, the cause of brown root-rot disease, is an invasive pathogen that was first described by Corner in Singapore (Corner 1932). It has a wide host range of primarily woody plants representing over 200 species from diverse families (Ann *et al.* 2002). This pathogen is also widespread, and has been reported to occur in many tropical/subtropical areas of Asia, Australia, Central America, Africa, and Oceania, where it can be quite destructive. *Phellinus noxius* appears to attack hosts regardless of health condition, and it can survive in organic matter long after host death. Early symptoms of brown root-rot disease are similar to other root diseases, including leaf chlorosis, wilt, and branch dieback; however,

mortality can occur relatively quickly after infection (Sashashi *et al.* 2012). A prominent sign of brown root-rot disease is a dark brown or blackish mycelial crust covering the stem base and root collar (Figure 1A). The associated wood decay typically displays honeycomb-shaped zone lines of reddish-brown to black (Figure 1B). The pathogen is often spread via root-to-root contact, but dispersal via basidiospores is also possible.

The general objective of this study is to predict suitable climate space for *P. noxius*, based on documented occurrences of DNA-sequence verified samples. By comparing bioclimatic variables associated with the precise locations of verified *P. noxius*, bioclimatic variables that influence pathogen occurrence can be identified and geographic locations that have suitable climate space for the pathogen can be predicted. Such information can predict potential distribution of *P. noxius*, and predict areas at risk from this invasive pathogen.

MATERIALS AND METHODS

A Maxent (Maximum Entropy) Species Distribution Model (version 3.3.3k) was used, because of its applications designed for presence-only data (Phillips *et al.* 2005). For this model, 19 bioclimatic variables (e.g., annual mean temperature, annual precipitation, mean temperature of coldest quarter, precipitation in warmest quarter, etc.) were used from WorldClim (worldclim.org). Sequencing of the ITS rDNA and translation elongation factor 1 α gene confirmed the identity of ca. 100 isolates (Figure 2) used for

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pathogen locations (latitude and longitude) to create SWD (Samples With Data) files that served as a basis for the Maxent bioclimatic model (Auto Features) using WorldClim climate surfaces. The regularization multiplier was kept at default, response curves and jackknifes were added to measure variable importance, and default settings were used for all experimental options. The final output, which is an average of all replicates under set parameters, displays current areas with suitable climate space for *P. noxius*. Minimal evidence of outliers was found, and cross-validation was used to verify data results. A total to 10 replicates were run with data split into training and test groups to assure accuracy and provide a statistical analysis. Quantum GIS (QGIS) was used to create the final outputs of the maps (Figure 3).

RESULTS AND DISCUSSION

The preliminary prediction maps show potential distribution (areas with suitable climate space) of *P. noxius* and areas that are at risk from this invasive pathogen, based on known locations of ca. 100 confirmed isolates from southeastern Asia, Australia, and Pacific islands (Figure 3). Please note that *P. noxius* isolates from Central America, Caribbean, South America, and Africa were not included in this study. Continued DNA-based characterization will help determine if isolates

belong to different species or genetic groups. Until more is known, caution is warranted for geographic areas that have suitable climate space for *P. noxius*, especially where this invasive pathogen does not currently occur (e.g., movement of plant material, infected wood, and infested soil should be restricted from areas where *P. noxius* occurs).

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Figure 1. *Phellinus noxius* infection on a dying flame tree (*Delonix regia*) during the dry season (A) and associated wood decay (B).



Figure 2. DNA-confirmed occurrences of *Phellinus noxius* that were used for bioclimatic modelling.

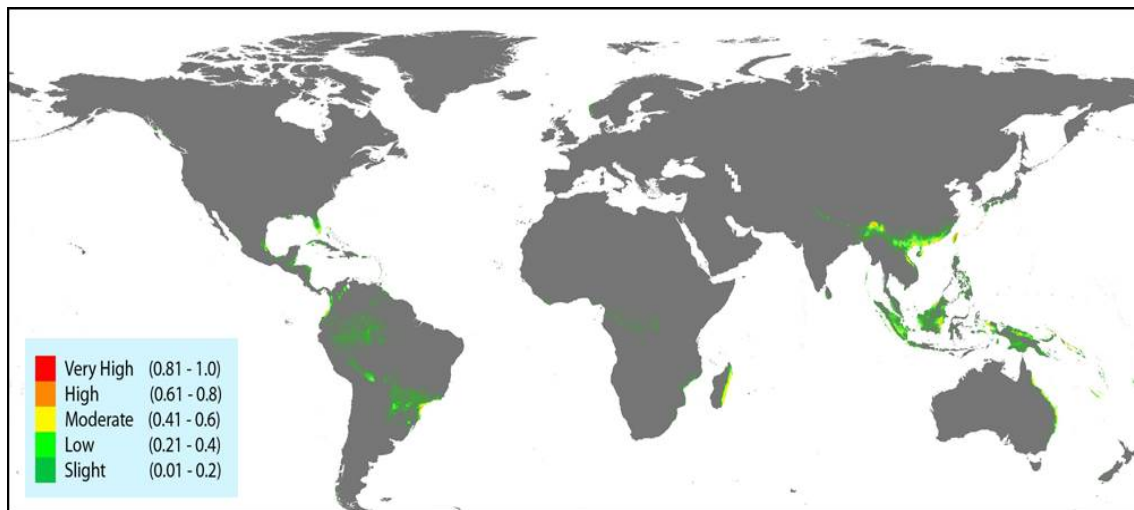


Figure 3. Preliminary predicted suitable climate space for *Phellinus noxius*. The colors indicate increasing climatic suitability from dark green (marginally suitable) to red (highly suitable). These predictions are based on 19 bioclimatic variables and the default settings in MaxEnt.

DIPLODIA SHOOT BLIGHT ON PONDEROSA PINE FOLLOWING HAIL IN NORTHEAST OREGON

Brent W. Oblinger¹

ABSTRACT

Diplodia shoot blight epidemics in ponderosa pine have been reported previously in parts of western North America but not in northeast Oregon (NE OR). Shoot blight caused by *Diplodia* spp was recently observed at multiple locations in NE OR. Symptoms were noted on ponderosa pine growing near portions of the Grande Ronde, Wenaha, Wallowa and Minam Rivers. Severe shoot blight was detected following a hailstorm surrounding Troy, OR. Very little dieback was associated with western gall rust and Diplodia shoot blight appeared to be widespread at this location based on samples collected. To monitor health of pines with shoot blight and severity of shoot blight in Troy, pole- to sawtimber-sized trees with varying severities were evaluated after the 2012 growing season and revisited after two growing seasons. Each third of tree crowns was visually rated for

shoot blight severity (0-2). After two years 31% of trees had died and those dead had higher initial severity ($p < 0.0001$ based on a Mann-Whitney test) than those still living. Trees killed were often attacked by western pine beetle (*Dendroctonus brevicomis*), *Ips* spp or had Armillaria root disease as well. Severity on trees still living did not change ($p = 0.21$) after two year (increased on 38% and did not change on all others). Based on PCR assays, *D. pinea* was detected on cones and shoots (unknown whether other *Diplodia* spp also are present). Results document Diplodia shoot blight damage in NE OR and the potential for this disease to predispose trees to mortality. Future climatic conditions influencing the frequency of hailstorms may affect incidence and severity of this disease.

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WEB BLIGHT AND PHYTOPHTHORA NEEDLE CAST DISEASES OF DOUGLAS-FIR

Paul W. Reeser¹, Wendy Sutton¹, Everett Hansen¹, and Alan Kanaskie²

INTRODUCTION

Web blight of Douglas-fir was first recognized damaging Christmas trees in plantations in central Willamette Valley around 1996. The disease also affected noble fir and grand fir. The disease is caused by an undescribed species of *Rhizoctonia* (aka *Ceratobasidium*) and pathogenicity on Douglas-fir was published in 1999. At this time symptoms were also seen in forest Douglas-fir in central Willamette Valley, but damage was not appreciable. Spotty occurrence of the disease was noted in Christmas trees and in forest settings over the next 15 years. Phytophthora needle cast of Douglas-fir, caused by *Phytophthora pluvialis*, was first recognized in New Zealand in 2012, although the pathogen was found in Oregon as early as 2002, being present in soil, stream water, and rain water in mixed tanoak and Douglas-fir forests in Curry County. The pathogenicity of *P. pluvialis* on Douglas-fir was confirmed at Oregon State University in 2014. During 2013 and 2014 limited rain trap surveys confirmed the presence of *P. pluvialis* in 30+ year old Douglas-fir plantations in the central and southern Oregon Coast Range, although appreciable defoliation was not observed at the sampling sites.

In late winter 2015 abnormal needle casting with distinctive needle symptoms was seen in Douglas-fir forest settings in the central Coast Range in Oregon (Figure 1). Either *P. pluvialis* or the *Rhizoctonia* pathogen was isolated from affected foliage. An ad hoc survey was conducted by personnel from Oregon State University Extension, Oregon Department of Forestry, and USDA Forest

Service to try to determine the extent of the web blight and Phytophthora needle cast occurrence.

METHODS

Cast and intact needles were collected from trees showing significant defoliation and characteristic needle clumping (Figure 2), and were diagnosed at the Forest Pathology lab at Oregon State University. For web blight symptoms, needles were surface disinfested in 5 % bleach, rinsed 3 times in deionized water, and plated in ¼ strength PDA amended with 100 ppm Streptomycin sulfate. Characteristic hyphae were sub-cultured for purity, DNA extraction, and storage. Identity of isolates was confirmed by sequencing of the nuclear rDNA-ITS region, and by staining hyphae with trypan blue to count the number of nuclei per cell. Reference isolates collected during the original outbreak were recovered from long-term storage and used for comparison.

For Phytophthora needle cast symptoms (Figure 3), needles were surface disinfested in 5 % bleach, rinsed 3 times in deionized water, and plated in corn meal agar amended with 20 ppm Delvocid, 200 ppm Na-Ampicillin, and 10 ppm Rifamycin SV. Characteristic hyphae were sub-cultured for purity, DNA extraction, and storage. Identity of isolates was confirmed by sequencing of the mitochondrial COX spacer region, and comparing these sequences with those of reference isolates in our collection.

RESULTS AND DISCUSSION

Rhizoctonia isolates collected during the 2015 survey were similar in appearance, number of nuclei per cell, and DNA sequence to our reference isolates collected in 1998. The pathogen was confirmed in Douglas-fir needles collected at locations shown in Figure 4. At six locations the

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pathogen was also isolated from diseased western hemlock needles. Sequence of the nuclear rDNA-ITS region showed a degree of variation that would suggest that the population of *Rhizoctonia* is not clonal and that the pathogen may be indigenous. This variation also encompasses the recently described *R. butinii*, which causes a web blight of spruce (*Picea abies*) in Germany and Austria. It is not possible to establish identity of the Douglas-fir *Rhizoctonia* with *R. butinii* as we have not observed basidia on needles or in culture. Severe infection may cause twigs and buds to be killed.

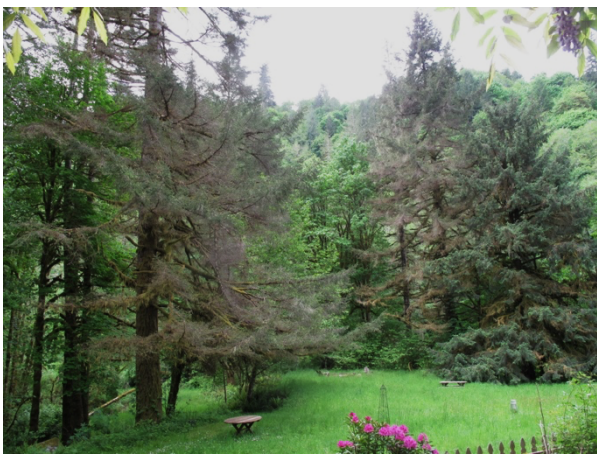


Figure 1. Douglas-fir trees affected by web blight or *Phytophthora* needle cast appear very similar when viewed from a distance. *Rhizoctonia* was isolated from foliage from the tree to the left, *P. pluvialis* was isolated from foliage from the tree to the right. Photo by E. Hansen.

P. pluvialis was confirmed in Douglas-fir needle samples collected during the 2015 survey. Locations are shown in Figure 4. Symptomatology is problematic. We have associated infection of needles with a non-descript chlorosis, but infected needles are shed rapidly, especially if the tree is disturbed by wind or handling (as when attempting to collect samples). Sometimes we can recover the pathogen from pre-symptomatic needles on the twig, or from recently cast needles. *P. pluvialis* can cause twig cankers in heavily inoculated seedlings in conditions of abundant moisture and dense shade. Severe infection may cause twigs and buds to be killed.

ACKNOWLEDGEMENTS

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Figure 2. Detail view of Douglas-fir needles killed by the web-blight fungus. The manner in which needles cling together and hang from the branch are distinctive characters for the disease. Close inspection reveals a fine fungal webbing over the needles. Photo by P. Reeser.



Figure 3. Artificially inoculated Douglas-fir needles showing chlorosis. *P. pluvialis* was isolated from circled areas. Photo by P. Reeser.

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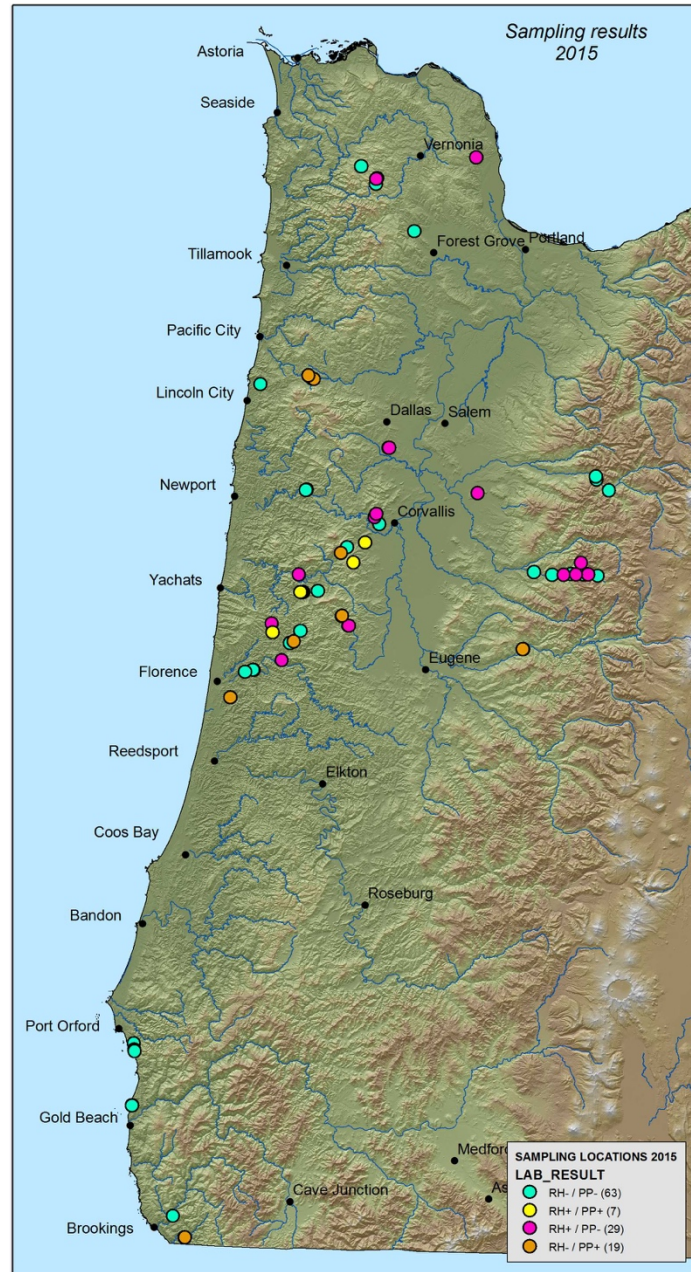


Figure 4. Locations where *Rhizoctonia* (RH) or *Phytophthora pluvialis* (PP) were recovered from symptomatic Douglas-fir and western hemlock. Map by Alan Kanaskie.

INTENSIFICATION OF SWISS NEEDLE CAST FOLIAGE EPIDEMIC ALONG THE COASTS OF OREGON AND WASHINGTON

Gabriela Ritóková¹, Alan Kanaskie², and David Shaw¹

ABSTRACT

Swiss needle cast, an endemic foliar disease of Douglas-fir (*Pseudotsuga menziesii*), is caused by an ascomycete fungus (*Phaeocryptopus gaeumannii*), and has reached epidemic proportions in the northwest coast of United States. Annual surveys since 1996 have demonstrated that the number of hectares displaying SNC symptoms continues to increase with 2015 showing all times high of nearly 590,000 acres. Because of sustained growth losses suffered by Douglas-fir, and the subsequent economic impact, a major research effort aimed at combating this disease has been initiated. Areas of research have included fungal biology and epidemiology, silvicultural treatments, genetic tolerance, predictive disease models. Numerous studies and predictive models have confirmed the association of spring leaf wetness and warm winter temperatures with increased fungal germination and development. Although conventional silvicultural treatments have been applied within SNC-infected stands (thinning,

fertilization, vegetation management, genetic tree improvement), disease symptoms have generally remained unchanged. Nevertheless, use of SNC-tolerant stock or thinning can improve the likelihood of infected stands reaching merchantable size in an acceptable amount of time. Foliage retention, the index of SNC infection intensity most highly correlated with growth loss, has been successfully predicted using seasonal climate variables. Application of future climate scenarios to this model provide an uncertain prognosis for the future. A new network of 120 plots is being installed throughout Oregon and southwest Washington coastal ranges as a means of monitoring future disease impact and providing framework for additional studies aimed at understanding this most dastardly disease.

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FINE-SCALE VARIABILITY OF FOREST SOIL FUNGAL COMMUNITIES IN TWO CONTRASTING HABITAT TYPE SERIES IN NORTHERN IDAHO, USA IDENTIFIED WITH MICROBIAL METAGENOMICS

Amy Ross-Davis¹, Jane E. Stewart², Matt Settles³, John W. Hanna¹, John D. Shaw⁴, Andrew T. Hudak¹, Deborah S. Page-Dumroese¹, and Ned B. Klopfenstein¹

INTRODUCTION

Forests are home to some of the most complex microbial communities (Fierer *et al.* 2012) which drive biogeochemical cycles (Clemmensen *et al.* 2013; van der Heijden *et al.* 2008) and account for substantial terrestrial biomass (Nielsen *et al.* 2011). Fungi, through their ecological roles as decomposers, mutualists, or pathogens, are particularly important in breaking down organic matter and mediating plant nutrition. Despite this, little is known about the variability, composition, and structure of forest soil fungal communities (Tedersoo *et al.* 2014). In fact, much of global fungal diversity remains undocumented (Blackwell 2011; Hawksworth 2012).

Our objective with this work is to apply next-generation sequencing technology to characterize forest soil fungal communities within a small subset of permanent plots established in two contrasting habitat type series within the Priest River Experimental Forest (PREF), Idaho, USA. Specifically, we are interested in exploring how the forest soil fungal community relates to environmental gradients and determine if and how it differs with depth below the forest floor and between habitat type series and seasons.

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MATERIALS AND METHODS

Soil cores were sampled from the center of each of 12 Forest Inventory and Analysis (FIA) subplots established within PREF at three depths below the forest floor (i.e., 0, 7.5, and 15 cm) at the midpoint between the bole and drip line of the nearest grand fir (*Abies grandis*) or Douglas-fir (*Pseudotsuga menziesii*) at two time points: 16-19 September 2013 and 3-6 June 2014. Six plots were established in the western redcedar (*Thuja plicata*)/western hemlock (*Tsuga heterophylla*) habitat type series (THPL/TSHE; Cooper *et al.* 1991) and six in the Douglas-fir/grand fir habitat type series (PSME/ABGR) providing a comparison of varying soil moisture levels. Trees were surveyed for the presence of *Armillaria* species which were identified using a combination of somatic incompatibility tests and *tef1a* sequence variation (Ross-Davis *et al.* 2012).

DNA was isolated from each soil subsample using the PowerLyzer PowerSoil DNA extraction kits (MoBio, Carlsbad, CALIFORNIA). Double-barcoded amplicons of the internal transcribed spacer 1 (ITS1) ribosomal DNA (located between the 18S and 5.8S rRNA genes) generated from replicated PCR were sequenced at the I-BEST Core Facility (University of Idaho, Moscow, Idaho) on a paired-end 300bp Illumina MiSeq run in a four file format: Read 1, Index Read 1, Index Read 2, and Read 2.

Raw fastq files were screened for presence of template-specific primer sequences and separated by primer sequence (maximum allowed mismatches 4) and index sequence (maximum allowed mismatches 1). Sequences

were overlapped using Flash (maximum proportion of mismatch 0.25 = number of mismatches/length of overlap) and then classified with the fungal database. Bootstrap cutoff was 0.5. The resulting fixrank file for the amplicon target was parsed to generate abundance of taxa by proportion and number of reads per sample as well as summaries of taxonomic classifications.

A UPGMA cluster dendrogram was created using the plot function in R to visualize patterns of soil fungi composition among plots, depths, and seasons. Environmental data and fungal richness were compared among samples to determine if and how they differ between habitat type series and seasons and across small spatial scales using SAS version 9.2 Software (SAS, Inc., Cary, North Carolina) via PROC GLIMMIX with type III tests of fixed effects used to examine interactions and main effects.

RESULTS AND DISCUSSION

Compared to THPL/TSHE plots, PSME/ABGR plots had thinner forest floors ($p = 0.0030$) with more abundant ($p = 0.0208$) and diverse ground vegetation ($p = 0.0067$). PSME/ABGR plots were also more variable with regard to soil moisture and temperature profiles (Figure 1): soil temperatures in PSME/ABGR plots warmed up to $> 0^{\circ}\text{C}$ in early-March compared to mid- to late-April in the THPL/TSHE plots. PSME/ABGR plots also had greater mean soil temperatures ($p = 0.0004$), with lower minima ($p = 0.0471$) and higher maxima ($p = 0.0033$), compared to THPL/TSHE plots, and volumetric water content reached significantly lower minima among PSME/ABGR plots ($p = 0.0009$). No significant differences were detected between habitat type series for most of the measured physical and chemical variables of either the forest floor or mineral soil.

Table 1. Richness and diversity (Shannon's index) of soil fungi by season, soil depth and habitat type series based on ITS1.

Treatment	Level	Richness	Diversity
Season	Spring	126.17 A	2.3387 A
	Fall	109.97 B	2.2768 A
	SE	5.4566	0.06238
Depth	0	125.75 A	2.3861 A
	7.5	112.96 A	2.2204 A
	15	115.50 A	2.3167 A
	SE	6.6830	0.07640
Habitat type series	PSME/ABGR	128.81 A	2.3667 A
	THPL/TSHE	107.33 B	2.2487 A
	SE	5.4566	0.06238
<hr/>			
Type III Test of Fixed Effects	df	F (Pr > F)	F (Pr > F)
season	1/60	4.40 (0.0401)	0.49 (0.4853)
depth	2/60	1.03 (0.3644)	1.19 (0.3125)
season*depth	2/60	1.75 (0.1822)	6.11 (0.0038)
habitat type series	1/60	7.74 (0.0072)	1.79 (0.1863)
season*habitat type series	1/60	1.76 (0.1891)	0.55 (0.4594)
depth*habitat type series	2/60	0.13 (0.8809)	1.10 (0.3406)
season*depth*habitat type series	2/60	0.40 (0.6726)	0.50 (0.6067)

A total of 605 fungal taxa was recovered from the forest soil samples, most of which were identified to genus (77%). The most ubiquitous genera were the common woodland agaric *Hygrocybe* and the ectomycorrhizal genera *Russula* and *Wilcoxina*. Fungal richness (number of taxa) differed significantly between habitat type series and seasons but not among soil depths, with no significant interactions among main effects (Table 1). A greater number of fungal taxa were recovered from PSME/ABGR plots compared to THPL/TSHE plots and from spring samples compared to fall. Community diversity was significantly higher just below the forest floor (0 cm) compared to greater sampling depths (7.5 and 15 cm) in the fall only.

The UPGMA cluster dendrogram did not reveal any clear patterns associated with depth, season, or habitat type series (Figure 2). However, when

specific taxa were examined individually, patterns emerged. For example, three genera were much more common in the spring relative to the fall (*Hohenbuehelia*, *Diversispora*, and *Conocybe*) while some were more common in the fall (*Cordyceps*, *Hebeloma*, *Brevicellicium*, and *Boletellus*). Several taxa were much more abundant among PSME/ABGR plots (*Cordyceps*, *Amanita*, *Calycina*, *Tuber*, *Gilkeya*, and *Conocybe*), while others were more abundant among the THPL/TSHE plots (*Hebeloma*, *Ramaria*, *Tylospora*, *Sarcosphaera*, *Leucogaster*, and *Boletellus*). For some taxa, abundance increased with soil depth (*Cordyceps*, *Hohenbuehelia*, *Leucogaster*, *Ramaria*, and *Brevicellicium*) while for others, abundance decreased (*Tylospora*, *Sarcosphaera*, *Conocybe*, *Amanita*, and *Boletellus*). Some patterns clearly relate to timing of fruiting (e.g., for *Hebeloma* and *Boletellus*). In-depth analysis is currently underway.

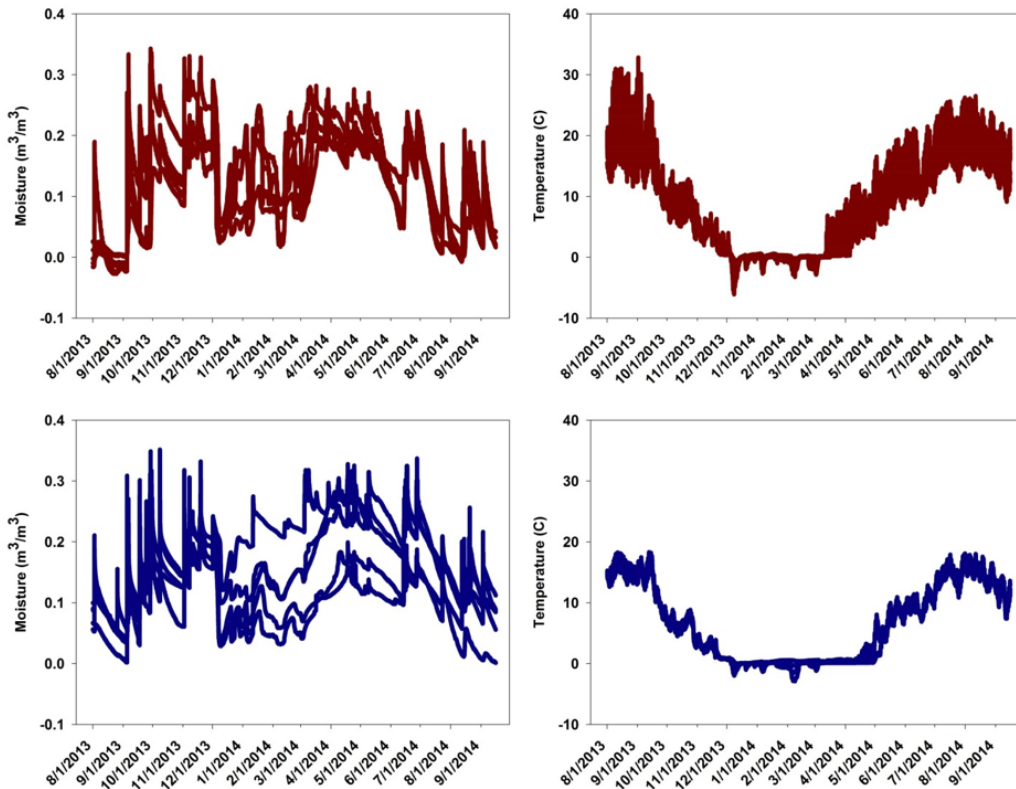


Figure 1. Profiles of soil moisture (left) and temperature (right) measured at 5 cm below the forest floor in each habitat type series (PSME/ABGR plots in red and THPL/TSHE plots in blue) every hour from 8/1/2013 through 9/15/2014.

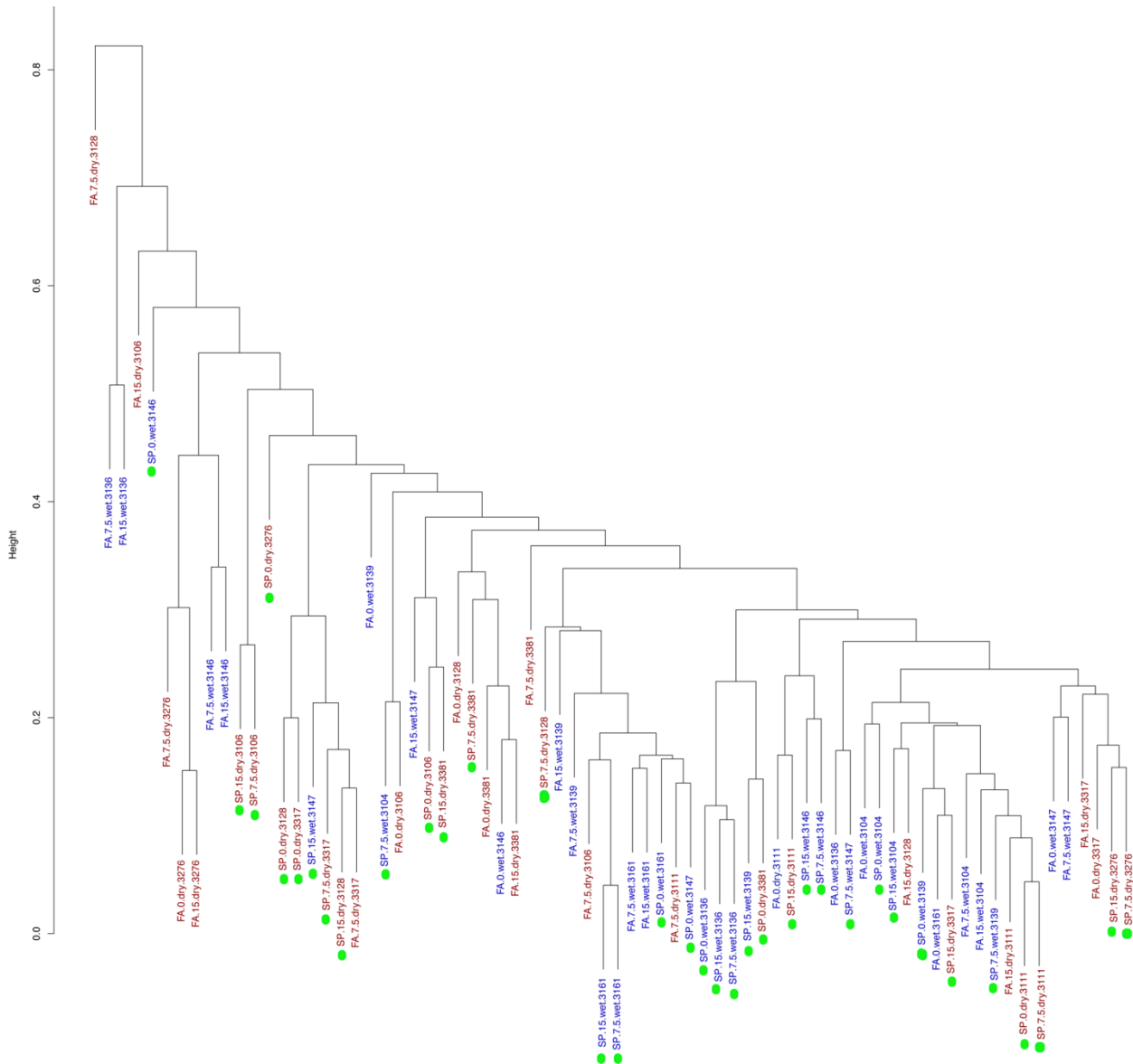


Figure 2. UPGMA cluster dendrogram of fungal abundance among subsamples in different habitat type series, seasons, and depths based on the ITS1. PSME/ABGR plots in red, THPL/TSHE in blue, and spring samples indicated by green dot.

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POPULATION GENOMIC ANALYSES OF THE BROWN ROOT-ROT PATHOGEN (*Phellinus noxius*): EXAMINING POTENTIAL INVASIVE SPREAD AMONG PACIFIC ISLANDS

Jane E. Stewart¹, Mee-Sook Kim², Louise Shuey³, Norio Sahashi⁴, Yuko Ota⁴, Robert L. Schlub⁵, Phil G. Cannon⁶, and Ned B. Klopfenstein⁷

INTRODUCTION

Phellinus noxius (Corner) G. H. Cunn is a vastly destructive, fast-growing fungal pathogen that affects a wide range of woody hosts in pan-tropical areas, including Asia, Australia, Africa, and Oceania (Ann *et al.* 2002; Figure 1). This pathogen causes brown root-rot disease on cacao, coffee, and rubber, as well as diverse fruit, nut, ornamental, and other native/exotic trees, with little indication of host specificity (Sahashi *et al.* 2010). Pathogenic symptoms of *P. noxius* infection can include reduced tree growth, defoliation, and branch dieback; however, *P. noxius* can survive as a saprophyte by colonizing heartwood and/or other organic matter. Brown root-rot disease can develop over several years, or in some cases, *P. noxius* infection can cause tree mortality within a year. Understanding the genetic diversity and evolutionary history of *P. noxius* populations worldwide will help assess the evolutionary origins, worldwide movement, and potential ecological differences within *P. noxius*.

The objectives of this study are to:

- 1) estimate the worldwide genetic diversity and evolutionary history of *P. noxius* movement;

- 2) determine if populations of *P. noxius* from Pacific islands show genetic signatures of introduced populations; and

- 3) characterize *P. noxius* for climatic modeling efforts to determine geographic areas at risk from *P. noxius* introductions.



Figure 1. Characteristic symptoms/signs of brown root rot caused by *Phellinus noxius*: mycelial crust (A) and mycelial mats and reddish-brown hyphal zone lines between the infected bark and sapwood (B).

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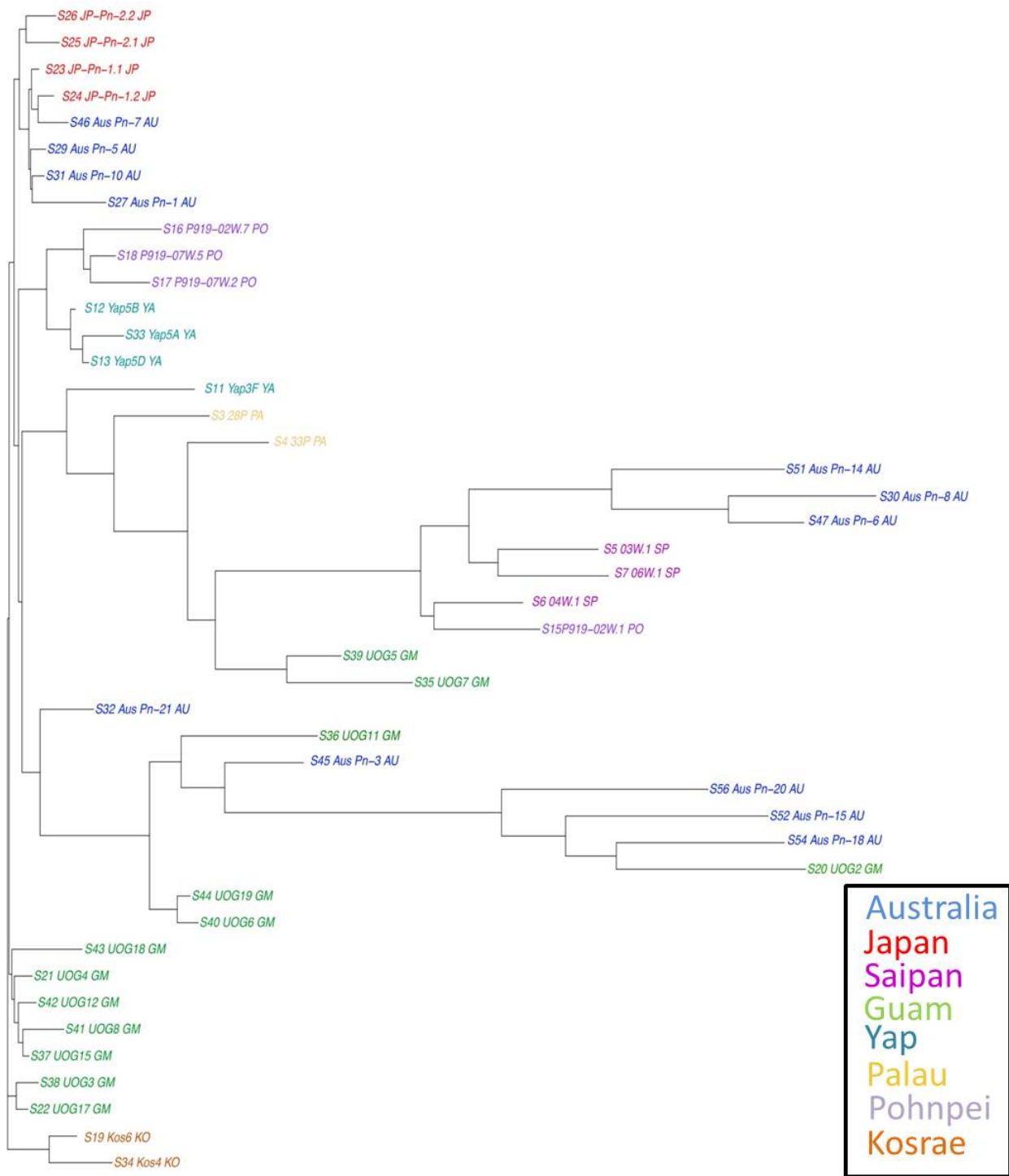


Figure 2. Preliminary results: Neighbor-joining of 1,449 SNPs within 396 loci. Isolates are color-coded by location.

METHODS

Isolates

A total of 56 isolates were included from Japan (4 isolates), Australia (19), and the Pacific islands including Saipan (5), Guam (10), Palau (4), Pohnpei (5), Kosrae (2).

Molecular characterization and analyses of molecular data

Sequence data were generated by Illumina sequencing of double-digest, reduced representation libraries (ddRAD). Restriction site associated DNA markers (RADseq), 3RAD design - Enzymes: *Bam*HI, *Cla*I, *Msp*I. RADseq loci were *de-novo* assembled, cataloged and analyzed in STACKS (Catchen *et al.* 2013). Pegas (Paradis 2010) (implemented in R) was used for neighbor-joining analyses (Figure 2).

RESULTS

Reads were assembled *de-novo* and grouped using STACKS (Catchen *et al.* 2013). A total of 24,142 total RAD loci were catalogued with 12,000 RAD loci per individual. The average depth per locus was 14x. Of the total RAD loci, 396 loci were used for analyses. These loci were found in 80% of the total 44 samples at a depth greater than 5x. We had a total of 50% missing data across the 396 loci for the 44 individuals. We recovered a total of 1,449 SNPs within the sampled population.

DISCUSSION AND FUTURE WORK

Preliminary results of the ddRAD single nucleotide polymorphism data show multiple genotypes of *P. noxius* that are structured geographically (Figure 2). Isolates from Pacific islands showed reduced levels of genetic diversity, which supports the hypothesis of potential introductions to some Pacific islands. Future research will include more populations from diverse geographic areas. Continued analyses will examine levels of gene

flow among populations, examine potential pathways of spread, and predict the potential spread of specific genotypes related to the current and changing climates. This study is aimed toward performing a wide-scale, population-genetic study of *P. noxius* isolates from eastern Asia, Pacific islands, and Australia to determine the regional population structure, estimate diversity in recently observed populations, and assess the potential suitable climate space for specific genotypes.

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THE EFFECTS OF SEED SOURCE AND PLANTING ENVIRONMENT ON DOUGLAS-FIR FOLIAGE DISEASES

Nicholas Wilhelmi¹, Dave Shaw¹, Connie Harrington², Brad St.Clair³, and Lisa Ganio⁴

ABSTRACT

Douglas-fir (*Pseudotsuga menziesii*) is an important commercial and ecological tree species in western North America. The current rates of climate change are predicted to have serious impacts on the successful regeneration of Douglas-fir forests and may render many of the current Douglas-fir seed zones obsolete. This will result in trees being maladapted to their current geographic locations. Strategies such as assisted migration and the revision of current seed zones may become very important management strategies in the mitigation of these negative impacts on Douglas-fir forests. However, the mechanisms that influence the successful movement of Douglas-fir seed to novel locations are not well understood.

Severe impacts of pathogens and insects are commonly associated with maladapted Douglas-fir populations. The foliar pathogens *Phaeocryptopus gaeumannii* (*P.gaeumannii*), the causal agent of Swiss Needle Cast, and *Rhabdocline* spp, the causal agent of Rhabdocline needle cast, are two very important Douglas-fir pathogens. These pathogens have been shown to disproportionately affect genetically maladapted seed sources, causing serious growth impacts and sometimes mortality. The relationship between the levels of susceptibility/tolerance to these foliar pathogens and the climate of the seed source is a key component in the identification of proper seed sources for reforestation. Understanding the variation in susceptibility/tolerance to Swiss needle

cast and *Rhabdocline* spp will be influential in the modification of current seed zones and the successful movement of seeds to novel locations.

This study is part of the Douglas-fir Seed Source Movement Trials, a large scale common garden, reciprocal transplant study. The study is comprised of 12 diverse west side Douglas-fir (*Pseudotsuga menziesii* var. *menziesii*) seed sources, ranging from northern California to southern Washington. These seed sources are planted in nine diverse planting environments ranging from southern Oregon to southern Washington, from the high elevation to the coast. These sites and seed sources were chosen to represent the wide variation in temperature and precipitation of western Oregon and Washington and offer an incredible opportunity to assess the influence *P. gaeumannii*, and *Rhabdocline* spp will have on these seed sources under various climate scenarios. Similar studies have compared inland Douglas-fir (var. *glauca*) to west side Douglas-fir (var. *menziesii*), whereas this study is composed strictly of west side Douglas-fir (var. *menziesii*). Every tree in this study was assessed for the presence and impacts of *P. gaeumannii* and *Rhabdocline* spp. We rated infection levels, needle retention, crown color and crown density. Our objectives are to: 1) Identify variation in the incidence and impact of these pathogens among these different seed sources. 2) Compare climatic variables between locations of seed sources and test sites to identify patterns in susceptibility/tolerance related to climate. We hypothesize that: 1) Susceptibility/tolerance to *Rhabdocline* spp and *P.gaeumannii* is a function of climatic differences between the seed source climate and the planting environment. 2) Seed sources from regions of high spring/early summer precipitation, low continentality index, and high mean winter temperatures are least affected by *P.gaeumannii*. and *Rhabdocline* spp.

In: Ramsey, A. & P. Palacios (Comps). Proceedings of the 63rd Annual Western International Forest Disease Work Conference; 2015 Sept. 21-15; Newport, OR. ¹Department of Forest Engineering, Resources and Management, Oregon State University, Corvallis, Oregon. ²USDA Forest Service, Pacific Northwest Research Station, Olympia, Washington. ³USDA Forest Service, Pacific Northwest Research Station, Corvallis, Oregon. ⁴Department of Forest Ecosystems and Society, Oregon State University, Corvallis, Oregon.

Preliminary results indicate that seed sources from southern Oregon and northern California are, on average, most susceptible to *Rhabdocline* needle cast and consistently exhibited lower crown density and needle retention. Little variation was observed in Swiss Needle Cast infection levels among the regions.

Through this project we will provide a better understanding of the impact *P. gaeumannii*, and *Rhabdocline* spp will have on west side Douglas-fir in different climate change scenarios. An increased understanding of the levels of susceptibility/tolerance to these foliar pathogens will provide current land managers valuable information to assist in the identification of proper seed sources.



Figure 1. Variation in susceptibility. Resistant Washington coast seed source (left) and susceptible California Sierra seed source (right) at Oregon central Cascades low elevation site.

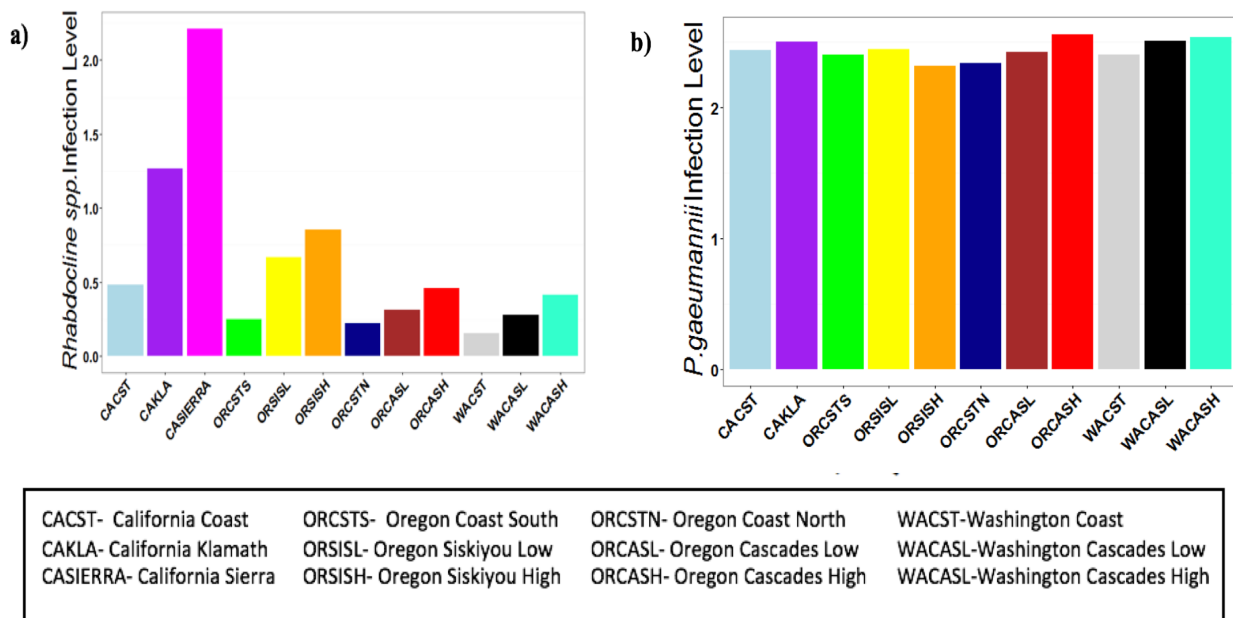


Figure 2. Infection levels of seed source regions by a) *Rhabdocline* spp and b) *P. gaeumannii*.

POSTER TITLES AND AUTHORS, WITHOUT INCLUDED ABSTRACTS

Author(s)	Title
Brandon Alveshere	Distribution and ecology of <i>Armillaria</i> spp in riparian areas of the Northern Great Plains. [Page 21 these proceedings]
Zolton Bair	Using transcriptomics to identify candidate genes associated with blister rust resistance in whitebark pine. [Page 23 these proceedings]
Jennifer S. H. Beck, Richard A. Sniezko	Blister rust resistance in whitebark pine at Crater Lake National Park.
Richard Cobb	The potential of tree resistance to <i>Phytophthora ramorum</i> as an alternative to scorched earth eradication measures on tribal lands.
John Dobbs, Mee-Sook Kim	Operational Disease Screening Program for fusarium wilt resistance in acacia koa.
Kelsey Dunnell	Characterizing resistance in <i>Populus nigra</i> to a population of <i>Sphaerulina musiva</i> .
Marianne Elliot, Gary Chastagner, Alan Kanaskie, Richard A. Sniezko, Doug Savin, Jim Hamlin, Connie Harrington, Valerie Hipkins, Jennifer DeWoody	The Pacific Madrone (<i>Arbutus menziesii</i>) common garden study: sentinel trials for monitoring genetic resistance to pathogens, insects, and potential impacts of a changing climate.
Betsy Goodrich	<i>Pinus strobiformis</i> in southwestern mixed conifer forests threatened by a non-native forest pathogen: Geographic and environmental variations in regeneration success and adaptive traits.
Jim Hamlin, Richard A. Sniezko, Fikret Isik, Douglas P. Savin, Angelia Kegley, Sally Long, Robert Danchok	Genetic variation in white pine blister rust resistance in sugar pine.
Rick G. Kelsey, Gladwin Joseph, Doug Westlind and Walter G. Thies	Ethanol in Douglas-fir roots stressed by <i>Phellinus sulphurascens</i> infection: Implications for detecting diseased trees and beetle host selection.
Harv Koester, Matt Buss, Richard A. Sniezko	White pine blister rust hazard rating for 265 sites in southern Oregon.
Luoma, D.L. and Eberhart, J.L.	Relationships between Swiss needle cast and ectomycorrhizal fungus diversity.
Jay Pscheidt, Everett Hansen	Yoda speaks to us.
Douglas P. Savin, Robert Danchok, Angelia Kegley, Richard A. Sniezko	White pine blister rust infection in whitebark pine seedlings over a wide range of inoculum density levels in an artificial inoculation trial.

Authors(s)	Title
Richard A. Sniezko, Jeff Stone, Douglas P. Savin	The World turned upside down: White pine blister rust pathogen goes down to go up.
Richard A. Sniezko, Robert Danchok, Douglas P. Savin, Angelia Kegley, Sally Long	Blister rust infection dynamics over 25 years in sugar pine progeny tests on six sites rated as low, mid, and high rust hazard in southern Oregon.
Richard A. Sniezko, Angelia Kegley, Douglas P. Savin, Robert Danchok	Dissecting blister rust resistance in whitebark pine – early results following artificial inoculation.
Richard A. Sniezko, Angelia Kegley, James Jacobs, Robert Danchok, Brian Luis, Sally Long, Douglas P. Savin	Variation in blister rust resistance and early height growth in three populations of southwestern white pine and implications for management and conservation.
Wendy Sutton, Paul Reeser, Everett Hansen	Bigleaf maple decline – history, survey and results.
C. Villari, R.A. Sniezko, A. O Conrad, D.P. Savin, L.E. Rodriguez-Saona, P. Bonello	Fourier-Transform Infrared (FT-IR) spectroscopy discriminates <i>Chamaecyparis lawsoniana</i> (Port-Orford-cedar) individuals that are resistant and susceptible to the invasive pathogen <i>Phytophthora lateralis</i> .



Committee Reports

2015 CLIMATE CHANGE COMMITTEE REPORT

Committee Co-Chairs:

Alex J. Woods, British Columbia Ministry of Forests, Lands, and Natural Resource Operations, Skeena Region, Smithers, BC. **Susan J. Frankel**, USDA Forest Service, Pacific Southwest Research Station, Albany, CALIFORNIA. **Charles G. “Terry” Shaw**, Emeritus, USDA Forest Service, Western Wildland Environmental Threat Assessment Center, Prineville, Oregon.

For WIFDWC 2015, the Climate Change Committee organized, “Drought and other factors as contributors to tree mortality”. A few key points from the presentations are provided below.

FUNCTIONAL TRADE-OFFS ALONG A CONTINUUM OF TREE DROUGHT RESISTANCE

Rick Meinzer, USDA Forest Service, Pacific Northwest Research Station, Corvallis, Oregon. Decisive answers regarding mechanisms that cause mortality due to drought are elusive, there are multiple stressors and there are trade-offs between survival mechanisms. Intense drought stress causes two types of physiological failure: hydraulic connectivity failure (embolisms), and carbon starvation due to stomata closure. Biological agents can exacerbate both mechanisms.

Different plant species have different drought coping mechanisms. Isohydric species maintain internal water potential at a steady state regardless of how much drought stress occurs. Anisohydric species alter their internal water potential in response to drought conditions. Juniper is an anisohydric species, pinyon pine is isohydric. The wide-scale pinyon pine die-off in the Southwest US, from 2002 to 2003, was the result of a severe drought. The pinyon pines died while the junipers survived so the area is now dominated by juniper. Pinyon pines lost their ability to open their stomata because of a loss of turgor pressure, eventually the trees starved to death. The hydraulic risk for a given tree is influenced by xylem efficiency, xylem vulnerability to embolisms, hydraulic capacity, stomatal control of xylem tension, and xylem recovery from embolisms.

STRUCTURAL MECHANISMS USED BY WOODY PLANTS TO DEAL WITH WATER STRESS

Barbara Lachenbruch, Oregon State University, Corvallis, Oregon.

Water is vital to many essential plant processes: cooling, mechanical support, nutrient and waste transport, and photosynthesis. Lack of water can cause plants to overheat, starve for carbon, wilt, and cease growth. Water moves through plants in a continuous column from roots to leaves driven by strong internal cohesion along a tension gradient. The gradient can be impeded by embolisms (air bubbles), or blocked by tyloses or gums (substances produced by parenchyma cells).

Plants can cope with drought at the cellular level by altering the shape or features of single cells, at the tissue level by partitioning or altering proportions of certain tissue functions, and at the organ or plant level by altering proportions of tissues within an organ (i.e. root to shoot ratios).

Depending on water supply and structural differences, top dieback can occur in a tree but a similar neighbor may suffer no apparent damage. Trees with higher leaf area such as those in fertilized or thinned stands can have larger leaf areas and thereby lose more water.

TREE DROUGHT STRESS AND INSECT AND PATHOGEN INCIDENCE IN WESTERN YELLOW PINE

Nancy Grulke, USDA Forest Service Western Wildland Environmental Threat Assessment Center, Prineville, Oregon.

Grulke investigated tree response to a severe 1999-2002 drought in Southern CALIFORNIA that caused some areas to lose 40-80% of its ponderosa pine (*Pinus ponderosa*). The trees died from a combination of drought stress and bark beetle attack and there was a need to translate environmental drought to the physiological consequences inside trees. The study used a series of Jeffery pine (*Pinus jefferyi*) trials rather than ponderosa pines as Jeffery pines were subject to less confounding biotic damage agents. Needle elongation was used as a simple proxy for physiological tree drought stress. Trees that had lower cambial total water potential and cambial turgor potential also had reduced resin exudate production. The analysis is on-going.

ANNUAL TRENDS OF ARMILLARIA ROOT DISEASE IN SOUTHERN BC

Michael Murray, British Columbia Ministry of Forests, Lands, and Natural Resource Operations, Nelson, BC.

Armillaria is the top mortality agent in Southeast BC where stand volumes can be reduced by as much as 50 percent. Measurements of 3,000 permanently marked trees found that 2/3 of all dead trees were killed by Armillaria. The study involved looking at years of infection as identified by ring widths. less than 50 percent of the previous year's growth. Murray used local weather data from within 15 km of trial sites and found pulses of Armillaria mortality coincided with drought years, particularly 2003 and 2007.

GLOBAL CLIMATE CHANGE AND WIDE-SCALE MORTALITY

Richard Cobb, US Davis, Plant Pathology Department, Davis, CALIFORNIA.

Cobb is part of an interdisciplinary research team developing an ecological, mechanistic model of tree mortality. All forested continents have experienced large scale tree die-off events but predictions of where these events will occur are very coarse. The causes of tree mortality are

complex and there are multiple interactions but in general the role of biological agents is poorly studied. Landscape level mortality events change ecological function in a variety of ways. In some cases there is a single ecological transition from a single agent; the ecosystem transitions and stabilizes at a new state. In other cases there is an ecological cascade with multiple steps and in other cases there is a multi-pathway response and a variety of new states. Land-use is a major driver of mortality patterns, areas with even-aged silviculture can suffer significant costs from these dieback events (i.e BC and mountain pine beetle). All examples of mass forest die-offs used in model development have included the influence of climate change, as well as other drivers.

CLIMATE CHANGE COMMITTEE MEETING

Wednesday, September 23rd, evening meeting

The Climate Change Committee meeting, held September 23, featured **Michael Murray**, BC Ministry of Forests, Lands, and Natural Resource Operations, Nelson on **Birch decline in Southeastern BC**. Birch decline has progressed in southern BC over the last ten years. Michael has been working with two graduate student projects looking into the phenomenon (Carlo Sarmiento, Univ. of BC and David Jordan, Trinity Western University). Sarmiento has found a number of pathogens associated with the dead-topped trees including *Fomes fomentarius*, *Armillaria*, *Cerrena unicolor* and a vascular wilt caused by *Cryptosporella tomentella*. Tree ring analysis conducted by Trinity Western University indicates that a variety of mature age classes have died. Alex Woods related observations in the Shuswap region of southern BC where paper birch (*Betula papyrifera*) has largely been removed from its position in mixed-species stands. North Idaho and Southern BC are pretty much the southern range extension of birch so it is possible that the decline is the result of increasing summer droughts. The conditions in the Shuswap area were apparently triggered by the 1998 drought.

ROUND ROBIN CONTRIBUTIONS

Dave Shaw: Recent high profile scientific papers from the ecology community (i.e Allen, CD; Breshears, DD; and McDowell, N G. 2015. On underestimation of global vulnerability to tree mortality and forest die-off from hotter drought in the Anthropocene. *Ecosphere*, 6(8), art129) have covered large-scale forest declines but fail to discuss the role of pathogens in those decline events. Dave Shaw contacted the ecologists, and they have asked forest pathologists to propose how to add pathogens into the literature. D. Shaw posed a challenge back to the forest pathology community to consider “How will forest diseases and insect pests interact with increasingly hotter drought conditions to influence tree mortality patterns?”

Kristen Chadwick, Holly Kearns and Amy Ramsey:. Experience with bigleaf maple (*Acer macrophyllum*) decline in Washington seems similar to the situation with paper birch in BC. In both cases the species involved is one that is not actively managed so few resources have gone into understanding the decline. A suggestion was made that a panel for a future WIFDWC cover “Signals

of climate change from underappreciated species”. Mike McWilliams suggested both the bigleaf maple and birch declines could be associated with Phytophthoras.

Nari Williams:. Most of the forest disease problems observed in New Zealand are the result of introduced pathogens rather than climate change impacts on the resident diseases.

Ned Klopfenstein: The incidence and severity of *Dothistroma* needle blight has been increasing in ponderosa pine in natural stands near Moscow, Idaho.

The remainder of the discussion centered on how best to capture changing disease conditions. Richard Cobb noted that it is important to make sure disease data is collected in a standardized fashion so that it could be used for meta-analyses. Forest Inventory and Analysis (FIA) plots in the US were recommended since they are standardized and although they are not very detailed they can be supplemented with additional info. Records of pathogen presence and damage are limited in FIA plots.





2015 DWARF MISTLETOE COMMITTEE REPORT

Committee Chair: Dave Shaw, Department of Forest Engineering, Resources and Management, Oregon State University, Corvallis, Oregon.

Announcements of Conferences/Meetings:

Mistletoes: Pathogens, Keystone Resource, and Medicinal Wonder July 17-22, 2016 Southern Oregon University, Ashland Oregon Sponsored by IUFRO Unit 7.02.11 Parasitic flowering plants in forests <http://blogs.oregonstate.edu/mistletoe/>

Parasitic Plant Societies:

The International Parasitic Plant Society: <http://www.parasiticplants.org/>, They have a great Newsletter called: The Haustorium.

International Union of Forest Research Organizations (IUFRO). Unit 7.02.11, Parasitic flowering plants in forests: <http://www.iufro.org/science/divisions/division-7/70000/70200/70211/> Meet irregularly, but planning future meetings and gatherings at IUFRO World Congresses and collaborations with other Units.

New-ish journal publications:

Agne, M., D. Shaw, T. Woolley, M. Bolanos. 2014. Effects of dwarf mistletoe on stand structure of lodgepole pine forests 21-28 years post-mountain pine beetle mortality in Central Oregon. PLoS ONE 9: e107532 Doi: 10.1371/journal.pone.0107532.

Mathiasen, R.L. and S.C. Kenaley. 2015. A morphometric analysis of *Arceuthobium campylopodum* – *occidentale* complex (Viscaeeae) Madrono 62: 1-20.

Mathiasen, R.L. and S.C. Kenaley. 2015. A morphometric analysis of *Arceuthobium campylopodum*, *A. laricis*, and *A. tsugense* (Viscaeeae) Phytologia 97: 200 – 218.

Mehl, H.K., S.R. Mori, S.J. Frankel, D.M. Rizzo. 2013. Mortality and growth of dwarf mistletoe-infected red and white fir and the efficacy of

thinning for reducing associated losses. Forest Pathology 43: 193-203.

Mortenson, L.A., A.N. Gray, and D.C. Shaw. 2015. A forest health inventory assessment of red fir (*Abies magnifica*) in upper montane California Ecoscience <http://dx.doi.org/10.1080/11956860.2015.1047142>

Oblinger, B.W. 2015. First report of white fir dwarf mistletoe (*Arcuthobium abietinum* f. species *concoloris*) on Engelmann spruce (*Picea engelmannii*) from Oregon. Plant Disease 99: 1041.

Reif, B.P., R.L. Mathiasen, S.C. Kenaley, and G.J. Allan. 2015. Genetic structure and morphological differentiation of three western North American dwarf mistletoes (*Arceuthobium*: Viscaceae). Systematic Botany 40: 191-207.

Smith, L., R. Hofstetter, and R. Mathiasen. 2013. Insect communities associated with Douglas-fir dwarf mistletoe witches' brooms in Northern Arizona. The Southwestern Naturalist 58(4):395-402.

New Reports:

Blodgett, J.T. 2015. Road/Plot survey of dwarf mistletoe and Comandra blister rust diseases in lodgepole pine on the Bighorn National Forest: 2013 Biological Evaluation R2-15-01.

Beatty, J.S. and J.C. Melgar. 2014. Protection against Mistletoe Infestations in the Central Region of Honduras. USDA Forest Service, International Programs.

Thesis:

Pritchard, K.R. 2015. Bird abundance and microhabitat association with oak mistletoe in Willamette Valley Oak Woodlands. M.S. Thesis, Oregon State University.

Book Chapters:

Nicholls, T.H. 2014. Evolution of a short-term study of lodgepole pine dwarf mistletoe vectors that turned into a long-term study of the remarkable gray jay on the Fraser Experimental Forest, Colorado, 1982-2009 Chapter 11, in; Hayes, D.C., S.L. Stout, R.H. Crawford, A.P. Hoover (eds.) USDA Forest Service Experimental Forests and Ranges Research for the Long Term Springer.

Shaw, D.C., and R.L. Mathiasen. 2013. Plant Diseases Caused by Higher Parasitic Plants: Mistletoes Chapter 5, in: Gonthier, P. and G. Nicolotti (editors), Infectious Forest Diseases CABI, Oxfordshire, UK.

New Developments:

Mathiasen, R.L. and Kenaley have a new paper coming that is an alternative to the recent changes in taxonomy of *Arceuthobium* and *Phoradendron* in North America and California THE CLASSIFICATION OF CALIFORNIA VISCACEAE: AN ALTERNATIVE PERSPECTIVE Madroño.



2015 FOLIAGE AND TWIG COMMITTEE REPORT

Committee Chair: Harry Kope, British Columbia Ministry of Forests, Lands and Natural Resource Operations, Victoria, BC. **2015 Acting Committee Chair: Stefan Zeglen**, British Columbia Ministry of Forests, Lands and Natural Resource Operations, Nanaimo, BC.

The committee met the morning of September 24, 2015. Three presentations (abstracted below) were given followed by a round-the-room discussion of several different foliar and twig pathogens. The acting chair's impression is that since this committee was formed the diseases discussed are becoming more notable in their diversity, frequency of occurrence and in their impact on forests.

Hybrid poplar leaf infection by *Sphaerulina musiva* (J. M. LeBoldus)

Sphaerulina musiva is a fungal pathogen of *Populus* spp that causes characteristic stem canker and leaf spot symptoms. In order to determine how this pathogen penetrates and infects leaves of susceptible hosts a greenhouse inoculation experiment was conducted. Resistant and susceptible genotypes were inoculated with a spore suspension of *S. musiva*. Inoculated leaves were collected at 24 h, 48 h, 72 h, 1 week, 2 weeks, and 3 weeks post inoculation and prepared for examination with both a scanning electron microscope (SEM) and a laser-scanning confocal microscope (LCM). Analysis of the SEM images indicated that the pathogen is able to penetrate leaf tissue through stomata; as well as, directly through the leaf epidermis by enzymatic action. Furthermore, there were no differences in the mode of penetration used by the fungus on either the resistant or susceptible genotype, indicating that the resistance response likely occurs post penetration. Using LCM imagery the fungus appears to produce a structure inside stomatal cavities following penetration. The fungus is currently being transformed with red fluorescing protein in order to improve visualization post penetration.

Douglas-fir canker development at Lake Cushman, Shelton, Washington (Will Littke, Amy Ramsey, Anna Leon, and John Browning).

Several 8-year old Douglas-fir plantations situated on gravel outwash soils experienced dieback symptoms prior to severe drought conditions during 2015. Cankers developed on boles, branches, and tops of many widely scattered trees. Trees of all vigor classes appear to be impacted. Examination of cankers failed to discover any associated insect activity such as Douglas-fir twig weevil (*Cylindrocopturus furnissi*), but similar cankers near Rochester, Washington showed evidence of *Phomopsis*.

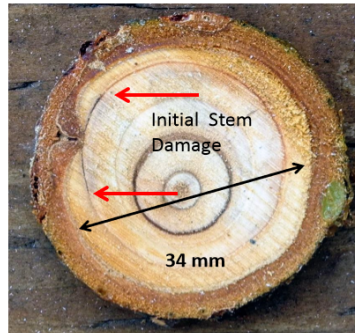
A second field trip to the site with Washington Department of Natural Resources and Weyerhaeuser pathologists collected data on canker development. Several hypotheses were examined: 1) cankers have developed following short severe summer drought events (2012-2014); 2) cold damage occurring either during fall or spring caused damage; and 3) cankers are initiated by pathogens. Five trees with the most severe canker symptoms on the bole were selected and cross sectioned to develop a chronology of damage initiation. The rationale being: summer drought effects would disrupt wood formation during otherwise growth periods, winter damage would occur after latewood formation, but prior to spring wood initiation, and pathogen activity would likely be random in occurrence. The affected stand is in a flat gravel basin surrounded by higher topography. This topography lends itself to drought susceptibility and cold air drainage. Weather data from the Shelton airport was examined for possible "events" during the 2012-2015 timeframe. Stem cross sections were examined and individual cankers dated. In all cases, the largest cankers initiated in fully developed latewood produced in 2014, but prior to the first early wood of 2015. It

was also apparent that callus growth was initiated from considerable larger wounds than what appeared to be due to the canker.

Tree-3

Callus growth initiated after damage occurring after the 2014 growing season.

Year	Radial Growth mm
2015	5
2014	5
2013	5
2012	5



All large cankers examined appear to be initiated during the same time interval. A spring cold event (8° F) is currently suspected as the damage “event”. However, there does appear to be secondary cankers (possibly *Phomopsis*) developing from this same event. Climatic damage events may precede and initiate secondary pathological events and lead to further stand damage. Potential solutions include recognition of topographic cold air pockets and selection of tree progeny with better cold tolerance for planting stock. Pathogen and climate work remains to be done to link this event with similar cankers reported elsewhere.

Leaf rust of black cottonwood in Boise County, Idaho (Angel Saavedra)

In the Pacific Northwest, a number of fungal pathogens cause foliar diseases on black cottonwood (*Populus trichocarpa* Torr. & Gray). These diseases are characterized by discoloration spots, blackening of entire infected leaves, blister of foliage tissue, or powdery fungal growth on affected leaves. Leaf rust of black cottonwood, a disease caused by *Melampsora occidentalis* is a common occurrence on black cottonwood. This disease can limit photosynthetic activity on infected leaves and causes leaves to fall prematurely. Symptoms in the field include yellow

spots in the undersurface of infected black cottonwood leaves.

US Forest Service staff recently observed a large number of black cottonwood trees with symptoms resembling leaf rust of black cottonwood infection. The observations were noted occurring along a 10 mile stretch of Idaho State Highway 21 (SH-21) in Boise County, Idaho. Surveys conducted confirmed the presence of leaf rust of black cottonwood infections. The undersurface of examined black cottonwood leaves had fungal reproductive structures characteristic of *M. occidentalis* (Figure 1).



Figure 1. *Melampsora occidentalis* on black cottonwood leaves.



Figure 2. Leaf rust of black cottonwood infected trees along SH-21.

Several leaf samples were collected and associated fruiting bodies to infections were examined under a light microscope. Examined fruiting bodies were yellow to orange in color and occupied most of the lower surface of infected black cottonwood leaves. Spores contained within the fruiting bodies were observed and measured. The spores were obovoid to periform in morphology, and their wall thickness presented bilateral symmetry. These observations further confirmed the presence of *Melampsora occidentalis* on affected black cottonwood trees. This pathogen requires an alternate host to complete its life cycle. In the Pacific Northwest, its alternate host includes Douglas-fir, western larch, and pines. Usually, this disease causes more damage on black cottonwood than its conifer host.

The survey estimated that 80-90 percent of black cottonwood trees were infected (Figure 2), and the degree of the disease varied within trees (from 25 percent to 75 percent of the crown). Usually, foliar diseases are issues when cool, wet spring weather patterns occur. This year's wet spring in Idaho has likely favored the development of this disease. In general, this foliar disease does not impact the long term health of infected trees. However, severe infections lead to shriveled leaves that will drop prematurely; hence, affecting the aesthetic of this corridor along SH-21.





2015 HAZARD TREE COMMITTEE REPORT

Meeting hosted by: Alan Kanaskie, Oregon Department of Forestry, Salem, Oregon.

Outgoing Chairperson: Pete Angwin (not able to attend due to US Forest Service travel restrictions), USDA Forest Service, Forest Health Protection, Region 5, Redding, CALIFORNIA. **New Chairperson:** Kristen Chadwick, USDA Forest Service, Forest Health Protection, Region 6, Sandy, Oregon. **Meeting notes:** Danny Norlander, Oregon Department of Forestry, Salem, Oregon and Kristen Chadwick, USDA Forest Service, Forest Health Protection, Region 6, Sandy, Oregon.

The committee would like to thank Pete Angwin for his service as chair of the committee.

Alan Kanaskie brought the meeting to order. The first business of the committee was to fill the committee chair position and the workshop chair position following the resignation of Pete Angwin on August 10, 2015. Kristen Chadwick volunteered to fill the role of both positions.

Greg DeNitto nominated Kristen Chadwick and Alan Kanaskie asked for any other nominations.

Mike McWilliams began a discussion about having a federal employee serve as the chair person. This was followed by discussion about federal employees needing support of their supervisor. The point was also made that some of the ethics involved would be more of an issue if the committee or chairperson were involved in potentially lobbying congress, as has been an issue for organizations such as the Wildlife Society. In the case of WIFDWC, the same ethics aren't involved since we do not act as lobbyists. Alan Kanaskie followed the discussion up with "that's a Forest Service issue and I don't think that we are going to solve that problem here."

Will Littke moved that nominations be closed with a second from Blakey Lockman. Motion carried and Kristen Chadwick became the new chair.

Alan mentioned that for the Workshops held every three years, traditionally the same person holds the committee and workshop chair roles. Discussion may be held during future planning calls for Sisters, Oregon meeting in passing the chair of the workshop around. Pete Angwin passed on the message through Alan that he can still participate as a USFS liaison and can help with planning the work conferences.

The rest of the meeting involved a round robin and discussion from the attendees.

International Tree Failure Database

David Hunter brought up support of the International Tree Failure Database (ITFD) and its current access (off line) and the concern of losing support of the FS maintaining the database. West coast arborists are concerned that the information that they put into the database is not accessible and that there is no movement forward to maintain the database.

Kristen mentioned she is the regional representative for the ITFD. Talked about how it is held by FHTET, and that the message has been delivered that FHTET was not interested in maintaining the database and it would need to be hosted by someone else, this was several years ago. Unfortunately, most outside of R5 were not proactive about putting data into the system. Much of the information came from a previous database from California. Kristen stated that the database is worthwhile and would be useful for court cases and issues that arise. Gary Man answered the questions regarding the servers. Currently the server where the database is housed is down and the contractors are not available to work on and maintain the servers at FHTET; this issue should be resolved soon.

Kristen Chadwick stated that we still need to look for a long-term solution for who manages and maintains the database. This has been a question since the Western Hazard Tree Workshop was last held in Oregon. It was asked if the International Society of Arboriculture (ISA) would be interested in hosting the database were raised and no one

present knew if they were ever approached. As discussion moved towards questions of what FHTET would do about the database, Gary Man pointed out that “it is the pathologists/arborists data base and not FHTETs and “we” need to figure out what it is used for and why it exists”. The hazard tree committee needs to decide why it is needed and where it goes.

Questions were asked about what was in the database and Kristen Chadwick and Alan Kanaskie responded that within the system it is reporting tree failures with a variety of conditions included. The database does not include data on trees that haven’t failed. The data has been used in the past to create failure profiles for different species. Blakey Lockman mentioned that the database was used in the National Root disease paper that is due out this winter.

In the end we all need to use it, incorporate its use into trainings, and support it if we are to keep it as a tool. The Forest Service will follow up with FHTET on this and we can take it to ISA to see if they are interested. We should also bring the representatives from all the regions together again to talk about where to go from here.

Danger Tree Field Guide

Greg Filip mentioned that Region Six FHP is revising the Field Guide for Danger Tree Identification and Response. It should be out before too long and just need one more revision.

USDA Forest Service AgLearn module for hazard tree awareness

Greg Filip brought up that an AgLearn module that has come out for hazard tree awareness in the Forest Service. Kristen Chadwick mentioned that her name is on the credits for reviewing it, however the developers did not take the recommendations of her and several other pathologists into account. There are several things that are not accurate in the video trainings. Overall the video misses the key points of what field-going personnel should be looking for. Gregg DeNitto mentioned that this is

not good for a stand-alone training. It is an awareness tool for employees. Some of the technical information is inaccurate. There is interest in having it corrected, however that is not urgent. There are concerns over the fact that it is for people that don’t have access to FHP for more information. There is no certification associated with it. Overall several people are concerned that it might be construed as a certification process. Blakey Lockman suggested that maybe they could say that the user needs to contact FHP for certification or further training.

There was some discussion about the tree falling certification and what level of hazard tree awareness is in the trainings. Mike McWilliams has talked to C-fallers and there is not a formal part on hazard trees in the C-faller training. Danny Norlander said in his experience recognizing hazard trees isn’t part of any of the falling levels. Basically it says that if it looks hazardous, walk away and call in a higher level. The higher levels are just supposed to “know what to do.” David Hunter mentioned that he was a C-faller and sometimes you just have to experiment in the field.

There was a lot of discussion about training fallers and firefighters in hazard tree awareness. Both Kristen Chadwick and Marcus Jackson have worked with fallers in recognizing hazard trees. Firefighters do an annual refresher and many years they make hazard trees a topic in the training.

Tree failure leads to Injury

Angel Saavedra reported on a tree failure that resulted in the victim being paralyzed. The 12.5 inch diameter at breast height (dbh) lodgepole pine involved in the incident was wind throw with and had no signs of decay at the lower bole or the root system. The issue that the Sawtooth National Forest will have is that the campground has had a history of wind throw. The campground had been thinning because of bark beetle mortality in the area; the treatment has opened the stand and made it more susceptible to wind throw incidences.

Campgrounds in western Oregon and Washington closed due to laminated root rot

Kristen Chadwick reported about continuing to close campgrounds or parts of campgrounds because of laminated root rot. In the area that she and Holly Kearns work, various campgrounds have been closed due to root disease issues, many of which our fantastic pathologists over the years have recommended not building. Most importantly, Indian Henry CG was built in the late 1970s against the recommendations of Greg Filip, Don Goheen, Jim Hadfield, and Craig Schmidt. Today, $\frac{3}{4}$ of it is closed and has been for the last several years. The District has finally made the decision to walk away from two loops that have laminated root rot impacting the Douglas-fir and older western hemlock that have been failing with significant decay in the roots from *Heterobasidion* root disease and *Perenniporia subacida*. They have decided to maintain the loop that is outside of the LRR center and treat a portion of the CG by removing both Douglas-fir and western hemlock.

Tomentosus Root Disease

Blakey Lockman briefly mentioned Tomentosus root rot continuing to have impacts east of the continental divide. She found that many of the spruce in one campground (Fairly Lake CG) had one to two inches of sound rind in ~18" diameter stumps. One campground (Jumping Creek CG) fell apart after a wind event. They are continuing to do vegetation management planning training and provide funds for campgrounds to start the vegetation management planning process. They are pushing hard to get in front of the hazard tree issues. Mike McWilliams asked if there is a template for the veg management plans. Blakey mentioned that Marcus Jackson and Brytten Steed worked with the recreation staff to create a template. It is on the Northern Region FHP website and there is a user's guide.

A Database for the USFS for Documenting Hazard Tree Assessments

Brent Oblinger mentioned that we all should be documenting hazard tree assessments so that there

are records for lawsuits. In his experience the reality is that there is not much documenting going on. The question was raised about who is requiring the documentation. Brent mentioned that it is in the policy for R6 recreation sites He has worked with the recreation staff to create a database based in Arc that users can enter their assessment into and can use with a Trimble gps unit. This way, you don't have to worry about losing paper copies and it automatically updates to the database. The database is designed to handle both the tree and campground level information. Unfortunately, they have had problems in their area with people not wanting to use this technology. They have been learning a lot about getting people to document yet alone learn a new technology. Alan suggested that they use an Ipad. At this point the humor of the FS came up with Ellen Goheen saying "we don't have Ipads." And then the suggestion of using their phones followed up with "we don't have iPhones".

Brent carried on the discussion with letting folks know that the database developed in central Oregon is intended to be used region wide and use could be part of the training sessions.

Kristen brought up the lack of consistency across Forests as to who is responsible for doing the hazard tree surveys, the recreation folks or concessionaires. In Brent's area the concessionaires are only supposed to deal with dead trees, in other places they have larger responsibilities.

Gregg DeNitto suggested that the database needed to become a corporate database if it was going to persist long-term. Brent responded that he was asked to create the database with support from the Regional office. The hope is that it will become a corporate database. Documenting hazard tree assessments is in the Region 6 policy, so direction is coming from above. Brent noted that the recreation staff he works with are starting to learn that record keeping is easier to do with the digital technology.

The session ended at this point.



2015 NURSERY PATHOLOGY COMMITTEE REPORT

Committee Chair: Anna Leon, Weyerhaeuser Forestry, Federal Way, Washington

The nursery committee was well attended and featured updates on nursery problems and technology updates from participants shared in a roundtable setting. We started with a brief presentation from Susan Frankel on recent detections of *Phytophthora tentaculata*, a pathogen causing stem rot, in native plant nurseries and restoration sites. It's believed that the pathogen is being moved throughout California through plant sales and restoration work. Samples suspected to be infected with *P. tentaculata* are being processed by the California Department of Food and Agriculture, but in this process they have found 33 different species of *Phytophthora* in the submitted samples.

Josh Bronson updated the group on the status of Stone Nursery, where the plants look healthy overall and all of the greenhouses are full of plants and there are many new employees working. They have been having problems with *Phylloxera*, an insect pest of oak, and also with *Fusiform* rust in container seedlings from Coeur d'Alene. Stone Nursery is still using Basamid fumigation, which requires a significant buffer.

Ellen Goheen is concerned about native plant nurseries that are used for restoration purposes. Foresters contract out to independent nurseries for native plants where no information is provided about plant health or potential disease transmission. An effort should be made to develop a set of best management practices for contractors; it was suggested that perhaps nurseries and water sources should be inspected as a part of this process. It was also suggested that foresters no longer order native plants from online nurseries without nursery inspections. An agreement would need to be established with the Department of Agriculture to set up these surveys and process the samples. It was also suggested that nurseries can be established as preferred growers through a process involving a site visit and meeting a set of

established criteria. Ellen would like any information on problems with native plants to be passed along to her or this committee.

Anna Leon updated the group of the completion of her Ph.D. work, in which she developed a real-time quantitative PCR (qPCR) assay for the detection and quantification of *Fusarium commune* and *Fusarium oxysporum* in nursery soils. This assay will allow us to quantify pathogenic *F. commune* without also including samples of the morphologically indistinguishable saprophyte *F. oxysporum*. The ultimate goal of improved *Fusarium* diagnostics is to decrease the amount of unnecessary fumigation due to a misunderstanding of soil *F. commune* levels.

Ned Klopfenstein and his lab with the Rocky Mountain Research Station are interested in soil metagenomics in relation to root disease. The newest next generation sequencing techniques can identify microbes ranging from the level of species to operational taxonomic unit. Understanding soil microbiomes is an emerging area of research and nurseries may be a good model in which we can observe how shifting microbe populations impact root disease. Will Littke brought up the known suppressive bacteria *Pseudomonas* as an example of an organism we would look for in suppressive soil, although he also mentioned that while it is suppressive against some fungi, it appears to be synergistic with *Cylindrocarpon*. This further emphasizes the need to gain a more comprehensive understanding of soil microorganisms.

John Browning and Anna Leon updated the committee with recent problems with *Phomopsis* in a Douglas-fir seed orchard. The *Phomopsis* is likely a secondary pathogen presenting itself as a result of abiotic stress. It is currently believed that the problems at the orchard were caused by a combination of drought and stimulation, a process by which trees are partially girdled to promote the

production of a stress cone crop. The cankers have been removed and the orchard is working to remove plant competition for resources and improve irrigation.

They also reported on summer cold damage reported at a nursery near Olympia, Washington. Nighttime temperatures dropped to just below freezing in mid-June, shortly after trees were transplanted. The seedlings with the earliest transplant dates did not appear to be affected, but the more recently transplanted seedlings were still vulnerable to fluxes in temperature. Frost protection is not traditionally used in the summer months, but will now become a part of the operating strategy if temperatures dip below freezing.

Weyerhaeuser continues to use methyl bromide fumigation, but is researching a new chemical, allyl

isothiocyanate (trade name Dominus), in conjunction with the Washington DNR Webster nursery. This chemical has a similar mode of action to brassica, is being marketed as cleaner than methyl bromide, and requires the minimum fumigation buffer of 25 feet. Webster is also doing research on chloropicrin alone and green manures at biocontrols. Weyerhaeuser is in the process of making an operational shift in which they will no longer sell bareroot material to the public. Their container seedlings will still be available.

Finally, John Browning updated the group on stunting of loblolly pine seedlings at a southern nursery. The seed are sown in double rows, one of which is stunted or yellow in a wave pattern down the field. The leading theory is that gypsum is not being uniformly distributed across the fields, but the problem remains a mystery.



2015 ROOT DISEASE COMMITTEE REPORT

Committee Chair: Blakey Lockman, USDA Forest Service, Forest Health Protection, Region 1, Missoula, MT.

In attendance: Alan Kanaskie, Alex Woods, Amy Ramsey, Anna Leon, Blakey Lockman, Josh Bronson, Kristen Chadwick, Christy Cleaver, Clive Brasier, Dan Omdal, Danny Norlander, David Hunter, David Rusch, Dave Shaw, Don Goheen, Gary Man, Gregg DeNitto, Greg Filip, Gabriela Ritokova, Ellen Goheen, Betsy Goodrich, Jafa, Jane Stewart, Jared LeBoldus, John Browning, Kathy Lewis, Holly Kearns, Ned Klopfenstein, Gary Man, Mike McWilliams, Mee-Sook Kim, Nari Williams, Natalia R. Fonseca, Nicholas Wilhelmi, Brent Oblinger, Paul Reeser, Ron Rhatigan, Angel Saavedra, Richard Sniezko, Stefan Zeglen, Tod Ramsfield, Walt Thies, John Hanna.

Blakey opened the meeting by asking everyone to give some thought on how we might better use this committee meeting. Should we be doing more than sharing information with each other (though that is very beneficial)? Stay tuned as she starts brainstorming during the interim between this WIFDWC meeting and the 2016 WIFDWC meeting in Sitka.

Short Presentations

Holly Kearns- update on the US National Root Disease Paper

- It's been a long process. All the parts are there: maps have been completed, sections are all written, and Holly and Blakey are working on edits with the contract editor from RMRS. Paper will be published as a GTR through RMRS with Ned Klopfenstein as the sponsor. R1 has obligated funds for the printing. We're hoping for it to hit the ground in late 2015.

Richard Sniezko- Phytophthoras in Scotland (Richard provided a handout for those in attendance)

Three Phytophthoras continue to have a major impact on tree species in UK.

1) Larch death from *P. ramorum*. and *Phytophthora ramorum* resistance 'exploratory' team. Richard Sniezko, Steve Lee, Sarah Green, Katy Hayden, Clive Braiser, Joan Webber.



For larch, some stands show >98% mortality – an effort is being made to find some resistant trees. This species has also been known to kill some of our PNW species (in Scotland, Sarah Green, per. comm).

2) *P. austrocedri* is taking a heavy toll on the native juniper. It also has impacted some of our native PNW species.



- 3) *P. lateralis* mortality has been noted on *Thuja plicata* (Sarah Green).



Chamaecyparis lawsoniana mortality at Dumbarton cemetery, Scotland (Photo credit: Sarah Green)

ROUND ROBIN

Blakey Lockman

- Continue to wrap up the US National Root Disease paper.
- Brief update on Heterobasidion hybrid-after WIFDWC last year, backpacked into the site with Gregg and Matteo and made more root collections, did some spore trapping, and stem mapped the obvious root disease pocket
 - o All but one of the root isolations appear to be first generation hybrids
 - o Successfully trapped a few spores- both *H. irregulare* and *H. occidentale* present
 - o Matteo Garbelotto presenting at IUFRO Root and Butt Rot meeting in Turkey in October, and is taking the lead on writing a paper
- Continue to find Tomentosus root rot as a destructive agent in spruce in eastern Montana, especially impacting recreation sites, has contributed to the closing and/or heavy management (clearcutting) in several campgrounds.

Brent Oblinger

- Recently (July 2015) remeasured long term Armillaria root disease plots (20 years). Preliminary results include:
 - o Thinning made no difference

- o Group selection (?)
- o Trees continue to die
- o Harvesting captured volume and reduced fuels
- o In summary, can't manage white fir/grand fir on Armillaria sites
- Working on using aerial imagery to identify areas with root disease through image classification and by mapping patterns of dead trees. Looking at possibly using LiDAR data also.

Ron Rhatigan

- Resurrecting abandoned Upper Chetco P.O.C. provenance test installed 1996.
- This test was one of four including Althouse Creek, (abandoned) Trinity Lake (ongoing) and, Humboldt Nursery (ongoing).
- Each test site originally had over 9000 planted test trees from most of the range of POC including some resistant selections.
- Original planting maps and first year mortality data is in GenDat.

Michael Murray

- Remeasuring stump removal trials (~13 trials).
- Armillaria main root disease on the sites.
- ~30,000 permanently marked trees.
- Preliminary results:
 - o Significant difference in survivorship and volume
 - o Incidence of root disease is about 1/2 in stumped sites
 - o Basal area volume is ~50% higher in stumped sites

Greg Filip

- Twenty year Armillaria study (same project as mentioned by Brent Oblinger)
- Finished a 30-year measurement of our Armillaria/Heterobasidion-PCT plots, and it was just published in the Oct. issue of Forest Science.

- Armillaria FIDL- it's focused on the West, and should be coming out early 2016.

Holly Kearns

- Working with Kristen Chadwick and others on re-monumenting and remeasuring Walt Thies' long term plots on laminated root rot.
- Also looking at Walt's old plots on species trials in laminated root rot areas.

Dan Omdal and Amy Ramsey

- Armillaria root disease pockets are appearing in the Seattle municipal watershed.
- No Douglas-fir beetle, but something triggered this onset of mortality.

Stefan Zeglen

- Working on a side project looking at Armillaria populations on Queen Charlotte Islands- *A. ostoyae* is the only confirmed Armillaria on the islands but there may be from 1-3 species of saprophytic A.o. on the islands that have never been identified or mapped.

Don Goheen

- Just glad to be here!

Mike McWilliams

- Mike is new to his zone (LaGrande, Oregon), so working on figuring out where root disease is at in his zone.
- Wanting to establish a few long term plots- random assignment-just getting it organized.

Josh Bronson

- Discussion about GPS points versus polygons for recording root disease on the ground.
- Recommend to get what you can while you're there (comment from Amy Ramsey).
- Others added- FHTET planning to "provide" units for recording (tablets), but it's just not quite there.

Kathy Lewis

- PhD student studying Douglas-fir beetle and Western spruce budworm, and schweinitzii root and butt rot keeps popping up in the DF.

Will Littke

- Seeing mortality of DF and Valley ponderosa pine- dying from Armillaria (Will did not clarify where).

Ned Klopfenstein

- Continuing use of DNA sequence-based identification of Armillaria species to document species occurrence, and hope to obtain funding for surveys in the coastal areas of Oregon, Washington, and the Alaskan panhandle.
- Collaborations are ongoing to identify Armillaria in Mexico, Iran, and other global regions. Soil metagenomics studies, which characterize microbial communities, are planned and underway toward developing novel management approaches for forest root disease, such as Armillaria root disease or brown root-rot disease, in collaboration with Amy Ross-Davis, Jane Stewart *et al.*
- Genomic sequencing efforts are underway for *Armillaria solidipes* (= *A. ostoyae*).
- The Moscow Forest Pathology lab is presenting nine posters at the meeting.

Mee Sook Kim

- Conducting genetic characterization of the koa-wilt pathogen (*Fusarium oxysporum*) in Hawaii with **Jane Stewart et al.**
- Conducting population genetic analyses of 100 isolates of the invasive brown root-rot pathogen (*Phellinus noxius*) isolates from eastern Asia, Australia, and Pacific islands- with **Jane Stewart et al.**
- Conducting phylogenetic analyses and bioclimatic modeling to determine area at risk from the invasive brown root-rot pathogen (*P. noxius*) under current and changing climates.

- Collaborative work with Region 5 (Phil Cannon) and Pacific islands to examine management approaches for *P.noxius*.

Danny Norlander

- Working on a geodatabase of surveys on ODF managed lands. Looking for ideas on ways to use almost 30 years' worth of root disease survey data besides where to plant alternate species.

Gregg DeNitto

- We're losing registration for Sporax, and movement towards the development of Rotstop.
- Black stain root disease-no one has mentioned it, has it gone away? (Don Goheen says there still lots in southwest Oregon; Will Littke says it will become a big problem again when we start to do more thinning).

Richard Snieszko

- P.O.C. resistant stock
 - o NEW TRIAL IN 2015 planted ON private land on Nickel Mountain IN SOUTHERN Oregon (?)
 - o Starker Forest- progeny tests
 - o Lots of non-federal planting of resistant P.O.C – HIGH INTEREST BY REDWOOD STATE AND NATIONAL PARKS

- Pacific Madrone/Phytophthora studies-poster at the meeting.

Gary Man

- Personnel changes
 - o Anne Hoover retiring in November
 - o FHM position (vice Boris Tkcaz)-moving on filling it, but not sure when
 - o Just hired a new budget analyst
- 2016 and 2017 budget planning- it doesn't look too good. If fires keep going, it could affect our 2016 budget. Oct/Nov travel plans may be affected- wait and see.
- Director of FHP- Monica Leer
 - o Pathologist by training
 - o Need to present your case in a compelling way
 - o Go through your Directors to get word out regarding root disease, and for word to get to the WO. Educate your Directors!

We briefly discussed the Root Disease Committee sponsoring a panel on mapping root disease across landscapes, or maybe more broadly, tools for mapping distribution of root disease across the landscape. Mapping the distribution of root diseases is an identified need in the National Root Disease Paper.

Notes taken and transcribed by Blakey Lockman and edited by all!

2015 RUST COMMITTEE REPORT

Committee Chair: Helen Maffei, USDA Forest Service, Forest Health Protection, Region 6, Bend, OR.

The meeting included a round table discussion of rust related projects. Only members who submitted reports post-meeting are included in this committee report.

Five-Needle Pine Hi5DB Database

Gregg DeNitto, USDA Forest Service, Forest Health Protection, Regions 1 and 4, Missoula, MT.

A new database for compiling plot summary information on high elevation five-needle pines is under development. This database is a successor to the Whitebark-Limber Pine Information System (WLIS) that was developed in the mid-2000s. Hi5DB has several changes and improvements to the original WLIS. One change is the expansion to include all of the high-elevation 5-needle pines, including foxtail, Great Basin bristlecone, limber, Rocky Mountain bristlecone, southwestern white, and whitebark pines. Most of the variables included in WLIS are in Hi5DB. Additional data fields are being provided for more information on regeneration. A major change will be the availability of Hi5DB for viewing to everyone by being available over the internet. Data can only be entered or changed by those with approval from data stewards. Over 4,600 records have been entered into the database and it is expected for this number to increase substantially once the database is completed and available. Although there have been some issues with the completion of Hi5DB, it is hoped that a working version will be available by the end of 2016.

Resistance of lodgepole pine and jack pine to western gall rust

Tod Ramsfield, Natural Resources Canada, Canadian Forest Service, Northern Forestry Centre, Edmonton, AB.

A new collaborative project has been established at the University of Alberta (Prof. Janice Cooke) with assistance from the Canadian Forest Service, Northern Forestry Centre. The project is investigating the host / pathogen interaction between lodgepole pine and jack pine and *Endocronartium harknessii* (= *Peridermium harknessii*) with the objective of identifying resistance in these species to the pathogen. The study will also include a population genetics analysis of the pathogen. The population genetics study requires samples of *E. harknessii* from a broad geographic distribution and we will be seeking samples of *E. harknessii* from throughout range of the pathogen in the USA. Michael Mbenoun, a post-doctoral researcher who is working on the project, is planning to contact WIFDWC members to assist with pathogen collection and we are hoping that WIFDWC members will be able to help us to assemble a sample population with a broad geographic distribution. Additionally, we are hoping that a partner in the US will be able to receive samples and provide laboratory space to host Michael so that he can extract DNA outside of Canada.

Whitebark Pine Direct Seeding Results in Northern Idaho and Montana, 5-Year Summary

John Schwandt¹ and Christy Cleaver²

¹USDA Forest Service, Forest Health Protection, Region 1, retired. ²USDA Forest Service, Forest Health Protection, Region 1, Coeur d'Alene, ID.

Whitebark pine direct seeding field trials were established on six whitebark pine sites in northern Idaho and Montana in the fall of 2009 and 2010 to determine if direct seeding is practicable and if seed or seedling treatments can enhance germination and survival. Eight hundred seeds collected from local seed sources were planted at each site in a randomized complete block design to

test four seed treatments. Treatments included: 30-day warm stratification at 21° C.; seed scarification by sanding; a combination of warm stratification plus scarification and an untreated control. One half of the seed in each treatment was covered with a wire mesh cage to minimize rodent predation for the first two years. The 2009 sites were: Fairy Lake on the Gallatin National Forest near Bozeman, MT, and Pioneer Mountain west of Big Sky, MT,

Thompson Peak on the Lolo National Forest near Plains, MT, Ulm Peak on the Idaho-Montana state line west of Thompson Falls, MT, and Gold Pass on the Idaho-Montana state line west of St. Regis, MT. In 2010, additional trials were established on Toboggan Ridge in the Clearwater National Forest north of Powell, ID, and Pioneer Mountain west of Big Sky, MT (Figure 1).

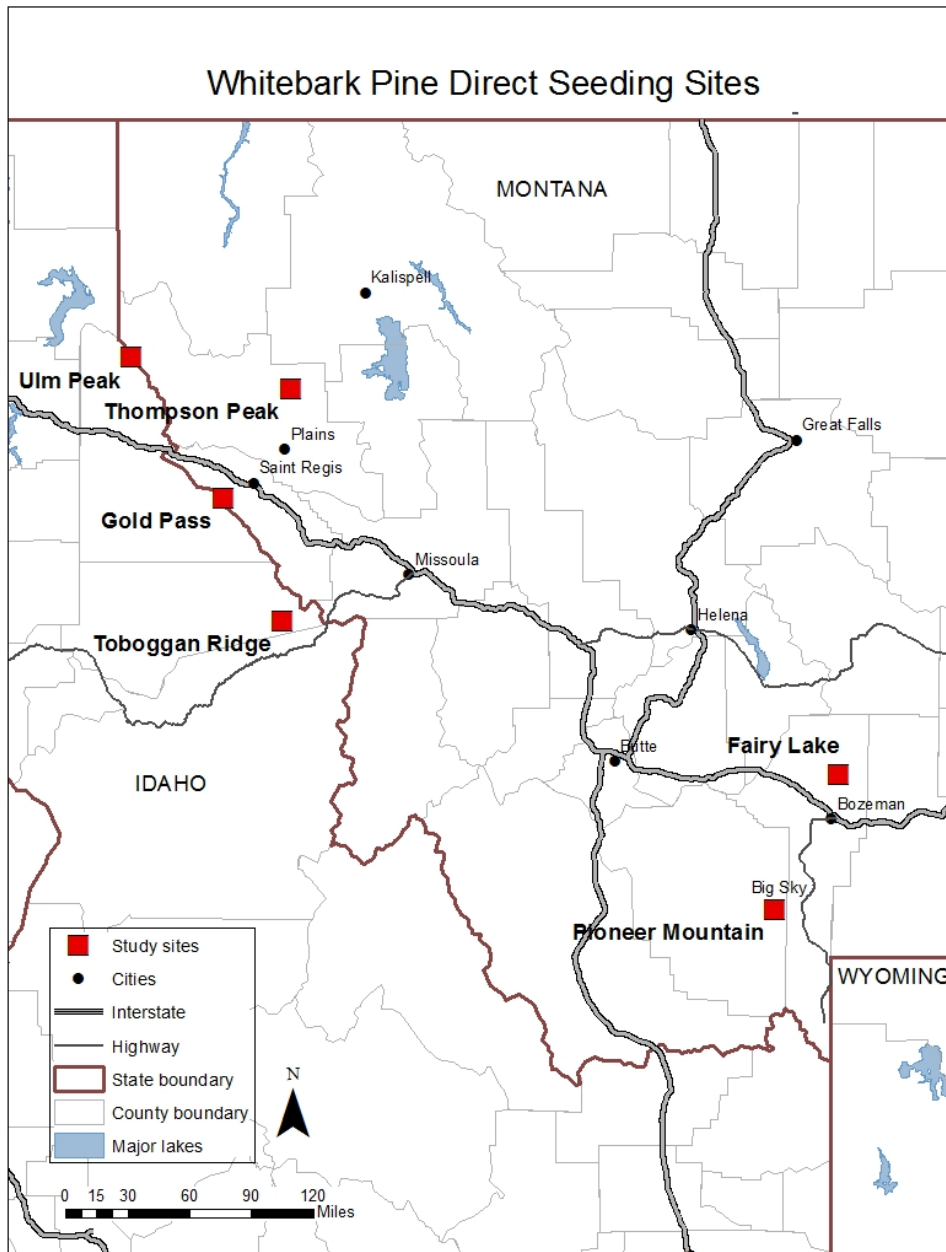


Figure 1. Map of the whitebark pine direct seeding sites established in Idaho and Montana in 2009 and 2010.

In addition, 3-seed caches were planted next to two-year old (2-0) nursery seedlings at each site to compare seed germination success and survival with survival of seedlings in the same microenvironment. This also provided an opportunity to compare 3-seed caches with individual seed planting at each site. The 2009 trials used untreated seed for the caches while the cached seed in 2010 were all given a 30-day warm stratification prior to planting. One half of the 2010 cached seed were coated with a mycorrhizal powder prior to planting. About 34 seedlings and 3-seed caches were established at the 2009 trials and about 100 seedlings and 3-seed caches were planted at the 2010 trial locations. Half of the 2-0 seedlings planted in 2010 were also given a mycorrhizal treatment about 30 days prior to planting. The third year after planting, the wire cages were removed and a sample of non-emerged seed in each treatment was dug up to record seed condition if found. Heights and diameters of all surviving seedlings were recorded in 2015.

Successful restoration requires good germination of seeds followed by successful seedling establishment. These sites have been monitored for at least 5 years to document germination success and seedling establishment.

Germination (seedling emergence)³ results (Table 1):

Although there was wide variation in germination levels observed between sites, some seed treatments consistently outperformed others:

- Warm stratified seed germinated best at 5 of 6 sites (> 50% average germination at 4 of 6 sites).
- Seed scarification was only beneficial at Pioneer Mountain and was less successful than the untreated control seed on all other sites.
- The benefit of warm stratification was reduced when combined with scarification at 5 of 6 sites.
- Germination of treated seed primarily occurred the first spring following planting; (e.g. 97% of warm stratified seed germinated the first year) while nearly half (42%) of the untreated seed didn't germinate until the second year.
- No seed germinated after the second year.
- Very little rodent predation of planted seed was observed either inside or outside cages.

Table 1. Total and average percent germination by site and treatment.

Site	Warm Strat				Ave Germ.
	Warm Strat	Scarify	Warm Strat & Scarify	Control	
Ulm Peak	52.3	11.1	43.0	30.0	34.1
Thompson Peak	56.0	24.1	58.8	37.9	44.2
Gold Pass	72.9	27.5	50.5	41.5	48.2
Toboggan Ridge	41.4	42.5	38.5	45.5	42.0
Fairy Lake ¹	24.0	9.0	20.5	18.5	18.0
Pioneer Mtn.	51.0	48.5	40.0	29.5	42.1
Ave (w/o Fairy Lk)	54.7	30.7	46.2	36.9	42.1

¹Fairy Lake was determined to be a limber pine site and was excluded from further analysis.

Table 2. Number of 3-seed caches, total seeds planted, and percent germination of each.

Site	# of 3-seed caches	% of 3-seed caches with 1 germinated seed	Total # of cached seeds	% of total seeds that germinated
Ulm Peak	27	37.0	81	22.2
Thompson Peak	34	88.2	100	66.0
Gold Pass	36	50.0	108	32.4
Toboggan Ridge	100	49.0	300	29.3
Pioneer Mountain	99	49.5	297	36.0

Results from 3-seed caches (Table 2):

- The percent of seed that germinated from 3-seed caches was less than individual warm stratified seed germination at all sites except Thompson Peak. This indicates that caching three seeds usually did not improve germination on the same sites.
- The percent of 3-seed caches with one or more seedlings was always greater than the percent of cached seed that germinated.

Survival and establishment of seedlings from direct seeding after germination:

- Once germinated, seed treatments had little effect on survival.
- An average of about 50% of seedlings from individually planted seed survived at least five years, but this varied widely by site.
- Survival increased with seedling size; minimal losses were observed in five year old seedlings that were greater than 15 cm tall.

Planting success of nursery seedlings and direct seeding (Figure 2):

- Survival and establishment of nursery stock on all sites was much better than seedlings from direct seeding of warm stratified or cached seed. Many of the 2-0 nursery seedlings were more than 61 cm tall and were well established.
- Survival of seedlings from all sources was best on good sites (Toboggan Ridge and

Thompson Peak,) and declined with harshness of the site (Gold Pass and Ulm Peak).

- Pioneer Mountain had good germination but seedling survival was severely impacted by gophers after the 4th year. Rodent damage was minimal on other sites.
- Rodents did not appear to seek out planted seeds.
- Mycorrhizal effects are still being monitored, but did not appear to be very effective in increasing germination or seedling survival.

Planting Success of Nursery Seedlings and Direct Seeding After 5 years at each site

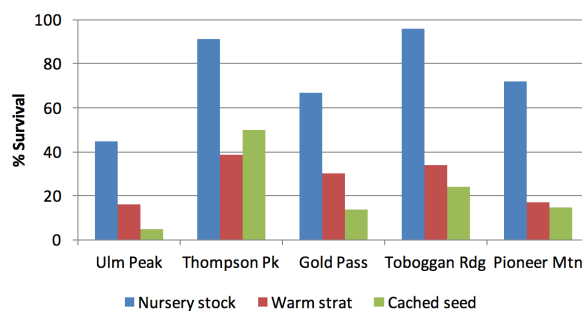


Figure 2. Percent planting success of seedlings and direct seeding at each site after five years.

Conclusions:

Early germination success with warm stratified seed at some sites indicated that direct seeding might be a potential tool for whitebark pine restoration. Treating seeds with 30 days of warm

stratification just prior to late fall planting appears to enhance germination, but declines if combined with seed scarification.

However, successful restoration of whitebark pine requires successful survival and seedling establishment as well as germination. Even though planted seedlings consistently outperformed the directly planted seed, these trials indicate that direct seeding may be a viable restoration tool especially on good sites where planting nursery

stock may be logistically challenging or restricted. A cost-benefit analysis could be performed if seed germination and site quality can be determined as well as costs for seed, seedlings, and planting.

The patterns observed in these direct seeding trials provide insight into the practicality of direct seeding, however, the wide variation observed between sites and treatments indicates the need for additional testing before direct seeding can be widely implemented or rejected.



2015 STUDENT AWARDS COMMITTEE REPORT

Committee: **Robin Mulvey**, USDA Forest Service, Forest Health Protection, Region 10, Juneau, Alaska. **Dave Shaw**, Department of Forest Engineering, Resources and Management, Oregon State University, Corvallis, Oregon. **Harry Kope**, Lands and Natural Resource Operations, British Columbia Ministry of Forests, Victoria, BC. **Alex Woods**, Lands and Natural Resource Operations, British Columbia Ministry of Forests, Smithers, BC.

The Student Travel Award Committee was quite busy in the months leading up to WIFDWC. It may have had something to do with the location of the conference being so close to Oregon State University and the territory of the BEAVERS but there was a great contingent of graduate students at WIFDWC this year. Many of them submitted excellent applications so our committee had quite a time deciding how to rank the applications and make the awards. In the end we gave out nine travel awards for a total of \$2,768.

Congratulations once again to the following students:

Brandon Alveshire,
Zolton Bair,
Christina Benemann,
Patrick Ian Bennett,
Dixie Daniels,
Meg Dudley,
Kelsey L Dunnell,
Yung-Hsiang (Sky) Lan,
and Nicholas Wilhelmi

Thanks again to all of those people who donated items for the auction. Through their generous donations and the excellent participation of the attendees the silent auction raised \$1,000. In addition, there were 48 regular WIFDWC member registrations which added \$720.00 to the student travel account which now has a balance of \$2,112.00.

Betsy Goodrich has now joined the committee and Robin Mulvey has cycled off. Thanks again to Robin for her time on this committee and to Betsy for stepping forward.



BUSINESS MEETING MINUTES

Secretary (acting): Amy Ramsey

The WIFDWC business meeting was called to order by the Conference Chair Alan Kanaskie at 10:30 AM on Friday, September 25th, 2015.

OLD BUSINESS

A motion to adopt the 2014 business meeting minutes without revision was made by Dave Shaw (then seconded). Motion passed.

Holly Kearns reported a summary of the Treasurer's report, including that there were 90 registrants for this meeting. She also reported that \$720 from 48 regular member registrants and \$1000 raised at the silent auction will go to the Student Awards Committee. A complete report will be included in the Proceedings.

Stefan Zeglen confirmed that the 2017 WIFDWC meeting will be held in either Campbell River, BC or Courtney, BC, which will be the third coastal location in a row. A straw poll vote resulted in a decision to have the meeting in October.

NEW BUSINESS

The Railroad Committee presented its slate of candidates for the 2016 meeting executives: Conference Chair (Paul Hennon), Brent Oblinger*(Secretary), Josh Bronson (Interim Program Chair), Robin Mulvey (Local Arrangements). Ellen Goheen made a motion to accept slate (seconded). Motion passed.

Brief reports were then given from the standing committees.

Allen Kanaskie was the standing chair, Kristen Chadwick was the interim chair and now chair of the hazard tree committee. The Hazard Tree Workshop is WIFDWC sponsored and will be held in October 2016. Historically the chair of the committee is the chair of the Workshop, which

used to be Pete Angwin. The Workshop consists of the chair, someone helping to get the local arrangements in place, three people working on the program. There will be conference call to discuss more details of the Hazard Tree Workshop.

Pete Angwin resigned as committee chair of the hazard tree committee earlier this year due to ethics issue. Greg DeNitto offered an explanation stating that the resignation was due to the interpretation of a letter from higher up within the USDA Forest Service and it specifically affected Region 5 personnel. This issue also came up with the WIFWC members, as to whether or not USDA Forest Service personnel can be on committees and it was determined that it shouldn't be a problem as long as there are no financial issues. Kristen Chadwick had more information saying that the ethics issue started with a circumstance relating to the California Pest Council, since they lobby the state legislature, and since WIFDWC does not lobby, then USDA Forest Service personnel should be able to be on WIFDWC committees without any problems. Terry Shaw also added that a new letter was located regarding ethics, so things may be okay now and that the ethics officer made a recommendation based on an old CFR.

Dave Shaw reported on the dwarf mistletoe meeting. Everyone had breakfast, discussed regional reports and noted that people are paying attention to dwarf mistletoes. It was reported that the Jepson Flora Manual says there are three species of dwarf mistletoes in California, but the committee does not agree. There still doesn't seem to be an agreement among individuals working on dwarf mistletoes about taxonomic clumping and splitting. Work is being done to keep the Hawksworth identification system in place. There will be an IUFRO meeting on mistletoes around the globe in Ashland, OR in July, 2016.

Blakey Lockman reported on the root disease meeting. 40 people were in attendance at the lunch meeting. Holly Kearns provided an update on the National Root Disease paper. Richard Sniezko presented his observations from Scotland of *Phytophthora*'s and associated damage. A discussion was held about the importance of long term monitoring of root disease nationwide. A suggestion was made to potentially sponsor a panel at the next WIFDWC regarding tools for mapping root disease. Gary Man mentioned that the USDA Forest Service deputy director will be retiring in November and that there needs to be a raised awareness of root diseases to the agency directors to hopefully raise the awareness of root diseases to a national level.

Stefan Zeglen reported on the foliage and twig disease meeting. Approximately 40 people were in attendance at the meeting. Jared LeBoldus and Will Littke presented work and observations, including cold damage associated cankers. There seems to be more diversity of pathogens and more interest in disease than ever before.

Anna Leon reported on the nursery pathology committee meeting. She reported that basically, nursery pathogens are affecting all of us and there was some discussion of the difficulties in identifying soil-borne pathogens.

Alex Woods reported on the climate change committee meeting, which occurred on Wednesday after the field trip. Michael Murray began the meeting by talking about birch decline in BC, which was then followed by a discussion about bigleaf maple and pathogen behavior in unappreciated species. Ideas for moving forward include making the most of what already exists in term of monitoring systems and if climate changing continues with current trends, then we may need to scale back in details and try to establish some course scale detection systems. The committee was unable to discuss a response to Dave Shaw's recommended climate change paper.

Holly Kearns reported on the student awards committee reminding members that the student award committee members cycle out periodically. Robin Mulvey, Alex Woods, Harry Kope and Dave Shaw are current members. Robin Mulvey has been on the committee the longest and will be cycling out. WIFDWC members nominated Betsy Goodrich to replace Robin Mulvey and she accepted.

The outstanding achievement award committee also contains members that cycle out every three years. Current members included Ellen Goheen (2015), Mike Cruickshank (2013) and Kathy Lewis (2014). Mike Cruickshank will be cycling out and Jared LeBoldus will be replacing him. Jared LeBoldus is a professor and Canadian, fulfilling the need to have academic and international representation on the committee.

Another discussion was had about the timing of WIFDWC. All organizations and agencies face different challenges with meeting attendance. For US federal employees, October is the beginning of the fiscal year, but sometimes Congress shuts down in early October. Kristen Chadwick pointed out that the WIFDWC bylaws say that the executive committee will decide when the meeting will be held. The academic school year is different across membership, and Kathy Lewis suggested that the meeting should not necessarily be planned with regards to the school year. Fiscal years are different across members. Walt Thies pointed out that again, let the planning committee decide. Blakey Lockman mentioned that it's increasingly difficult to travel late in the fiscal year. Then Greg DeNitto seconded what Walt, and basically Kristen said, ending the discussion on the time of year that the meeting should occur.

There was another discussion about a joint meeting with the Western Forest Insect Work Conference (WFIWC) occurred. Mike McWilliams will check in with members of WFIWC about a potential joint meeting in 2018. WFIWC members were supposed to discuss this line item at their spring, 2015

meeting, but no one from WIFDWC has heard back.

Everett Hansen initiated a discussion about having a *Phytophthora* workshop. He asked WIFDWC members about potential objectives, an agenda and logistics, with a commitment from Oregon State University to host. Dave Shaw suggested creating an ad hoc committee to plan the details. Terry Shaw suggested a questionnaire to determine the needs of the *Phytophthora* workshop. Mike McWilliams reminded the group that identification of *Phytophthora* spp should be a key component of the workshop. Everett Hansen wondered if the group was talking about culturing and identification in each group's own lab, or how to interpret results if the identification was done by a diagnostician. Danny Norlander brought up the idea that the WIFDWC community may be losing old lab methods due to the ability to send samples to a more experienced lab, but maybe WIFDWC could sponsor a general pathogen identification training to get those skills passed on to the new cohort of pathologists. Holly Kearns said that WIFDWC does have money to spend on a training and that there may be the ability to make money on a training. Stefan Zeglen suggested using the Hazard Tree Workshop model for training and establish a committee to plan the training. Melody Putnam said that the cost for a *Fusarium* training was \$1500. She is connected to the *Phytophthora* community and knows of *Phytophthora* trainings that happen approximately every other year. Ellen Goheen volunteered to be chair of the ad hoc training committee and will be taking the lead on sending out a questionnaire to potentially interested parties about desired objectives and organizing the training.

Kristen Chadwick pointed out that the bylaws need to be updated to include the extra \$15 student award fee added onto annual meeting registration costs. The bylaws are currently updated through 2011. Amy Ramsey will update the bylaws through 2015.

Dave Shaw brought up an interesting conversation that he had with Gary Man. Basically, the WIFDWC community is not communicating with Washington, DC very well and we need to start promoting ourselves as necessary, relevant and solution oriented. Dave Shaw suggested that maybe the chair of each academic institution write a letter to the head of the USDA Forest Health Protection or the USDA Forest Service chief. Beth Willhite shared that the bark beetle working group developed a mission statement and that maybe WIFDWC could do the same, while also listing all the collaborative projects that WIFDWC is involved in. Terry Shaw and Richard Sniezko suggested inviting relevant people working in Washington, DC to our meetings and possibly having them provide an overview perspective. Blakey Lockman mentioned that Gary Man offered some ideas on how to move the root disease paper up the chain and that he wants us to be successful. Danny Norlander suggested having more pathologists attend the WFIWC meetings each year.

Nari Williams announced that there are some jobs and PhD opportunities at her organization in New Zealand.

At the close of the new business, a motion was made to end the business meeting and the motion was passed. The business meeting ended by 11:30 AM on Friday, September 25th.

TREASURER'S REPORT, 63rd WIFDWC

Submitted by Holly Kearns

The 63rd annual WIFDWC in Newport, Oregon had 84 attendees including 48 regular members, 14 graduate students, 8 retirees, 5 single day attendees, and 9 guests. The following is a summary of transactions for the WIFDWC accounts from 1/1/2015 through 11/30/2015. The WIFDWC Federal Tax Identification Number is available upon request.

	Income / Expense	Balance	Total Account
All WIFDWC Accounts balance 12/31/14			\$48,930.42
WIFDWC Meeting Account balance 12/31/14		\$28,719.48	
Total registration	19,719.23		
Hotel meeting rooms, meals & breaks	-8,211.59		
Banquet	-4,418.32		
Field trip transportation	-3,949.00		
Field trip supplies and snacks	-352.88		
Souvenirs and awards	-1,341.11		
Office supplies	-18.15		
Invited speakers expenses	-103.87		
Regular member registration fees to STA Fund	-720.00		
Other Account Activity			
2014 Proceedings (printing and formatting)	-1,896.32		
WIFDWC Meeting Account balance 11/30/15		\$27,427.47	
Hazard Tree Committee Account balance 12/31/14		\$7,666.94	
No Activity			
Hazard Tree Committee Account balance 11/30/15		\$7,666.94	
Student Travel Award Fund balance 12/31/14		\$3,160.00	
2015 Student Travel Awards	-2,768.00		
2015 Silent auction proceeds	1,000.00		
2015 Regular registration fees (48 @ \$15)	720.00		
Student Travel Award Fund balance 11/30/15		\$2,112.00	
International Sponsorship Fund balance 12/31/14		\$9,384.00	
Registration, meals, hotel for two sponsored pathologists	-1,627.36		
International Sponsorship Fund balance 11/30/15		\$7,756.64	
All WIFDWC Accounts balance 11/30/15			\$44,963.05

WIFDWC OUTSTANDING ACHIEVEMENT AWARD RECIPIENTS

Year	Recipient	Meeting	Comments
2000	Lew Roth	Kailua---Kona, HI	For pioneering work on <i>Phytophthora lateralis</i> , <i>Armillaria</i> and dwarf mistletoes, and for inspiration and leadership of a generation of plant pathology students and colleagues.
2000	Duncan Morrison	Kailua---Kona, HI	For long-standing contributions to forest pathology research, especially in relation to roots diseases and tree hazards.
2001	Bob Gilbertson	Carmel, CA	For contributions to the taxonomy and identification of wood-inhabiting basidiomycete fungi.
2002	No award given.		
2003	Everett Hansen.	Grants Pass, OR	For strong leadership in forest pathology including research on the biology and management of tree and seedling diseases of western conifers.
2004	Bob James	San Diego, CA	For strong leadership in forest pathology especially technology transfer and research on the biology and management of forest nursery diseases for growers and nursery pathologists throughout the West.
2005	Walt Thies	Jackson, WY	For sustained long---term high quality research on laminated root rot and other root diseases of forest trees.
2006	Bart van der	Smithers, BC	In recognition of outstanding lifetime contribution to tree disease research and for inspiring a generation of students and colleagues in the field of forest pathology.
2006	Alan Kanaskie	Smithers, BC	For outstanding leadership, as a practicing forest pathologist, in the management of Swiss Needle Cast.
2007	Richard Hunt	Sedona, AZ	In recognition of his valuable research and extension efforts on white pine blister rust, along with many other contributions to forest pathology and biology.
2008	No award given		
2009	Bill Jacobi	Durango, CO	In recognition of your 30-plus years as an educator, researcher, organizer, advocate and practitioner of forest pathology.
2009	Bob Edmonds	Durango, Co	In recognition of 40-plus years as an educator, researcher, organizer, advocate and practitioner of forest pathology and ecology.
2010	Paul Hennon	Valemount, BC	For sustained, significant contributions to our knowledge and understanding of forest disease dynamics and ecology.
2011	Susan Frankel & Ellen Goheen	Leavenworth, WA	For leadership in the science and practice of forest pathology and for critical contributions to the management of Sudden Oak Death.
2012	John Schwandt	Lake Tahoe, CA	For the energy, enthusiasm, and integrity which he has invested in the professions of forest pathology and forest management.

WIFDWC OUTSTANDING ACHIEVEMENT AWARD RECIPIENTS CONTINUED

Year	Recipient	Meeting	Comments
2013	Don Goheen	Waterton Lakes, AB	In honor of your 35 years of dedicated service to forest pathology as a researcher, leader and mentor of others.
2014	Terry Shaw III	Cedar City, UT	In recognition of broad western U.S. and international experiences, and dedicated mentoring and storytelling.
2014	Willis R. Littke	Cedar City, UT	In recognition of a valuable industry perspective, support for WIFDWC Nursery Committee, international experience, mentoring and storytelling.
2015	Brian Geils	Newport, OR	In recognition of a creative scientist with a broad range of interests, a high level of enthusiasm and curiosity, and a great guy to be with in the field.

WIFDWC OUTSTANDING ACHIEVEMENT AWARD MEMBERS

Year	Members		
2000	J. Byler	W. Littke	B. van der Kamp
2001	W. Littke	B. van der Kamp	R. Sturrock
2002	B. van der Kamp	R. Sturrock	G. Filip
2003	R. Sturrock	G. Filip	
2004	G. Filip	D. Goheen	S. Zeglen
2005	D. Goheen	S. Zeglen	D. Shaw
2006	S. Zeglen	D. Shaw	B. Ferguson
2007	D. Shaw	B. Ferguson	R. Reich
2008	B. Ferguson	R. Reich	E. Goheen
2009	R. Reich	E. Goheen	P. Angwin
2010	E. Goheen	P. Angwin	H. Kope
2011	P. Angwin	H. Kope	B. Jacobi
2012	H. Kope	B. Jacobi	P. Hennon
2013	B. Jacobi	P. Hennon	M. Cruickshank
2014	P. Hennon	M. Cruickshank	K. Lewis
2015	M. Cruickshank	K. Lewis	E. Goheen

STANDING COMMITTEES AND CHAIRS, 1994—2015

Committee	Chairperson	Term
Hazard Trees	J. Pronos	1994—2005
	P. Angwin	2006—2015
	K. Chadwick	2016—present
Dwarf Mistletoe	R. Mathiasen	1994—2000
	K. Marshall	2001—2003
	F. Baker	2004—2013
	D. Shaw	2014—present
Root Disease	G. Filip	1994—1995
	E. Michaels Goheen	1996—2005
	B. Ferguson	2006—2009
	M. Cleary	2010—2011
	B. Lockman	2012—present
Rust	J. Schwandt	1994, 2005
	R. Hunt	1995—2004
	H. Kearns	2006—2011
	H. Maffei	2012—present
Disease Control ^a	B. James	1995—2002
Nursery Pathology	B. James	2002—2005
	K. Mallams	2007—2010
	W. Littke	2011—2014
	A. Leon	2015—present
Foliar and Twig Diseases ^b	H. Kope	2007—present
Climate Change ^c	S. Frankel	2007—2008
	S. Frankel & D. Shaw	2009—2014
	S. Frankel, D. Shaw & A. Woods	2015—present

^aDisease Control committee was disbanded in 2002.

^bFoliar and Twig Diseases committee was made full charter member in 2009.

^cClimate Change committee was made full charter member in 2010.



BYLAWS OF THE WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE

Passed by a vote of the Membership at the Business Meeting of September 25, 2015.

Article I

Objectives_____

The Western International Forest Disease Work Conference (WIFDWC) was formed in 1953 to provide a forum for information exchange among forest pathologists in western North America. The primary objectives of the organization are:

- To exchange information on forest pests and related matters through periodic meetings and other appropriate means,
- To promote education, research and extension activities in forest pathology, and
- To sustain and improve the health of western North America's forests.

Article 2

Membership_____

Membership is open to individuals who are engaged in forest pathology related endeavors in western North America. These include but are not limited to: research, survey, management, teaching or extension activities pertaining to tree diseases, forest health, or deterioration of forest products.

Western North America is defined as Canada: British Columbia, Yukon, Alberta, Manitoba, Saskatchewan; United States: Washington, Oregon, California, Idaho, Nevada, Utah, Arizona, Montana, Wyoming, Colorado, New Mexico, North Dakota, South Dakota, Nebraska, Kansas, Alaska, Hawaii, Guam, the Commonwealth of the Northern Mariana Islands and other Pacific Islands in Micronesia; and all of Mexico.

Membership is established after attending one Western International Forest Disease Work Conference. Members must attend another Western International Forest Disease Work Conference within 5 years or their membership is no longer valid.

Honorary Life membership will be automatically awarded to those members of WIFDWC (as defined above) who have attended at least 5 previous meetings of WIFDWC and have retired. Newly retired members who meet these criteria should notify the current WIFDWC Secretary of their status. Other members who have retired but do not meet the attendance criteria or other outstanding contributors to the field of Forest Pathology may request, or be proposed for, Honorary Life Membership by members present at an annual business meeting.

A list of Honorary Life Members will be published in the Proceedings of each meeting.

A 50% or more reduction in the registration fees for Honorary Life Members, to include a copy of the Proceedings, should be considered by the Executive Committee, as per Article 7.

Article 3

Officers_____

WIFDWC officers will include a Conference Chairperson, Secretary, Treasurer, Program Chairperson, Historian and Web Coordinator. The Conference Chairperson and Secretary will be elected by majority vote of the membership at the annual business meeting. If there is no majority, an acting Chairperson will be appointed by the current Conference Chairperson. The tenure of the Conference Chairperson and Secretary begins at the conclusion of the WIFDWC meeting where they were elected and ends

when all business from the next WIFDWC is completed. The Treasurer, Historian and Webmaster will be elected every five years, to serve for the following 5 years.

Duties of the Conference Chairperson

At each WIFDWC, the Conference Chairperson will run the general and business meetings. The Conference Chairperson will appoint an interim Program Chairperson at the start of each WIFDWC to gather suggestions and opinions to guide the conference in the planning of next year's conference. The Conference Chairperson will also appoint three members to serve as the "railroad committee" to nominate candidates for next year's Conference Chairperson and Secretary (and every fifth year, Treasurer, Historian and Web Coordinator). The Conference Chairperson may appoint members to assist in conducting the affairs of the Conference including, but not limited, to Local Arrangements representative(s) and Program Chairperson. The Conference Chairperson may also appoint ad hoc committees and their chairpersons as deemed necessary to assist in carrying out the mission of WIFDWC.

In the event that a new Conference Chairperson cannot carry out their duties, the previous Chairperson will carry them out. If another member of the Executive Committee cannot or will not carry out their duties the Conference Chairperson may appoint a replacement.

Duties of the Secretary

The Secretary shall maintain the membership and mailing lists. The Secretary shall send out meeting notices to the membership, take minutes at the business meeting, and compile and distribute the Conference proceedings.

The secretary will query all Honorary Life Members to determine if they want to receive a free copy of the proceedings and only those responding in the affirmative will receive a copy.

Duties of the Treasurer

The Treasurer shall receive all payments, be custodian of WIFDWC funds, keep an account of all moneys received and expended, and make commitments and disbursements authorized by the Conference Chairperson. At the annual business meeting the Treasurer shall make a report covering the financial affairs of WIFDWC. All funds, records and vouchers in the Treasurer's control should be subject to inspection by the Executive Committee.

Duties of the Program Chairperson

The Program Chairperson is appointed by the Conference Chairperson. The Program Chairperson is responsible for all aspects of the conference agenda including arranging the format and timing of the meeting, selecting panel chairpersons or moderators, selecting the poster session coordinator, assigning subject matter committee meeting times, and arranging keynote, contributing paper and other speakers.

Duties of the Historian

The Historian will keep a complete set of WIFDWC proceedings and answer any inquires as needed. The Historian will contact the WIFDWC Secretary and provide the address for mailing the archival copy of the proceedings.

Duties of the Web Coordinator

The Web Coordinator will create and manage the WIFDWC website. The Web Coordinator will supervise the hosting, security and access of the website. Content for the website will be provided by the Executive Committee for each meeting. The Web Coordinator will ensure that previous WIFDWC meeting websites and their proceedings are archived and linked to the current website.

Compensation

Officers will not be compensated for their services.

Non-liability of Officers

The officers shall not be personally liable for the debts, liabilities or other obligations of the WIFDWC.

Article 4

Decision Making Process_____

The business meeting will be run under Roberts Rules of Order. Meetings are open to the public and non-members may participate in meetings. Only members may vote.

Decisions will be made by majority, with each member granted one vote. Votes may be called for at the annual business meeting or via electronic ballot (i.e., e-mail ballot, web poll, etc.). A quorum is reached when more than 25 members are present.

Article 5

Finances_____

Expenditures

The Conference Chairperson may authorize expenditures of WIFDWC funds. Standing Committee Chairs may similarly authorize the expenditure of funds that are generated by their standing committees (e.g., Hazard Trees Committee). Checks, orders for payment, etc. may be signed by the Treasurer, or other person designated by the Chairperson. The Executive Committee may determine which and how many outside speakers they want to invite, and travel costs for such speakers can be paid from registration fees.

Contracts

The Conference Chairperson may authorize any officer or agent of WIFDWC to enter into a contract on behalf of WIFDWC. Standing Committee Chairs may similarly authorize any agent of their standing committee to enter into a contract on behalf of their committee. Unless so authorized, no person shall have any authority to bind WIFDWC or any standing committee to any contract.

Gifts

The Conference Chairperson or the Treasurer may accept on behalf of the WIFDWC any contribution, gift, or bequest. Commercial sponsorship of conference special events is not allowed.

Fiscal year

The WIFDWC fiscal year shall begin on the first of January and end on the last day of December.

Article 6

Bylaws_____

Amendments

Changes to bylaws shall be made available to all WIFDWC members for review at least one month prior to the next business meeting. A two-thirds majority is required to pass a motion to amend existing bylaws if the vote is held at a business meeting. An affirmative vote from at least 26 members is required to approve a motion voted on by electronic balloting (i.e., e-mail ballot, web poll, etc.).

Article 7

Meetings

Frequency

The WIFDWC endorses holding annual meetings but will, on vote of the membership, change the time of any particular meeting when circumstances dictate that such action be taken.

Date

WIFDWC endorses holding meetings in late summer but will change the interval between any two meetings when circumstances dictate that such an action be taken. Meeting dates will be set by the Executive Committee for each meeting.

Registration

Registration will be reduced by half, if possible, for graduate students and Honorary Life Members. It will be at the discretion of the WIFDWC Executive Committee for each meeting to offer a further reduction in fees to graduate students and Honorary Life Members and to offer further reduced fees to others such as retired professionals and visitors.

Article 8

Committees

There shall be two types of committees, namely

- a) Standing Committees – as designated in the by-laws, and
- b) Ad Hoc Committees – as appointed by the Conference Chairperson to serve for a term specified by the Chairperson.

The chair of each standing committee shall prepare a report of the committee activities for the membership. The report will be submitted by the publication deadline to the Secretary for inclusion in the proceedings.

The following are WIFDWC standing committees:

- Executive Committee
 - o Composed of the elected Conference Chairperson, Secretary, Treasurer, Historian and Web Coordinator.
 - o The Conference Chairperson may appoint a Program Chair, Local Arrangements representative(s) and other persons as necessary to carry out the business of the next WIFDWC meeting.
 - o The Executive Committee may invite non-member speakers to the annual meeting and pay their travel expenses from conference registration fees.
- Awards Committee
 - o Composed of three members with the longest serving member designated as chair.
 - o Committee will be comprised of a representative from each of the following – a university employee, a public agency employee, and one member at large. At least one member should be from Canada.
 - o The chair's term will be completed at the end of the annual business meeting and a new junior member will be appointed by the Conference Chairperson. The most senior serving member will assume the chair for the next year.
 - o The chair will provide a report of activities at the annual business meeting.
 - o Responsible for accepting and evaluating nominations and determining recipients of the WIFDWC Outstanding Achievement Award as outlined in Article 10.
- Student Scholarship Committee
 - o Composed of four members with the longest serving member designated as chair.
 - o The chair will provide a report of activities at the annual business meeting.
 - o The committee will be comprised of at least one representative from a university.
 - o Replacement of committee members will be by election at the annual business meeting.

- The committee is responsible for fundraising to finance any awards given by the committee.
- The committee is responsible for determining and advertising the award application criteria, receiving and evaluating applications and determining recipients of the WIFDWC Student Travel Awards as outlined in Article 10.
- Hazard Trees Committee,
- Dwarf Mistletoe Committee,
- Root Disease Committee,
- Rust Committee,
- ~~Disease Control Committee~~ [disbanded 2002],
- Nursery Pathology Committee [approved 2002],
- Foliage and Twig Diseases Committee [established 2007, approved 2009],
- Climate Change Committee [established 2007, approved 2010].

Ad hoc committees are established by the Conference Chairperson to carry out various functional needs (e.g., the annual Nominating Committee). Ad hoc committees carry out specific, normally short term, tasks required by the membership. The terms of reference for ad hoc committees will be determined by the Conference Chairperson in consultation with the membership.

Article 9

Proceedings_____

Papers for each year's proceedings must be submitted to the Secretary by the deadline set for each conference by the Secretary.

Distribution of proceedings is made to all paid registrants and honorary members who have indicated a desire to receive them and will be made available to others at cost.

Article 10

Awards_____

Outstanding Achievement Award

Members may recognize outstanding achievement in the field of forest pathology by bestowing the WIFDWC Outstanding Achievement Award. The award will recognize an individual that has, in the opinion of the membership, contributed significantly to the field of forest pathology in western North America.

The award will be presented during the conference by the chair of the Awards Committee or designate. The recipient will receive a framed certificate or plaque. The recipient will present a keynote address at the following year's WIFDWC. A list of recipients will be published in the proceedings.

Members may nominate other current or active members for the award; they may not nominate themselves. A member may only make one nomination each year. A nomination must include: a short introductory letter, a narrative of the nominee's qualifications, educational background, work history, etc., letters of support from other members and organizations, and copies of a few of the nominee's published works. Nominations are due no later than three months prior to the start of next year's conference and must be sent to the Awards Committee chair.

The Awards Committee may decide to not make an award if no suitable candidates are nominated.

Student Travel Awards

Members encourage participation in the annual conference by students engaged in studies in the field of forest pathology by bestowing the WIFDWC Student Travel Awards to enable their attendance.

The awards are intended for students currently enrolled in a university graduate level program with a thesis or dissertation topic relevant to the field of forest pathology. The awards are intended to assist with conference-related expenses.

Criteria for application and selection of award recipients will be determined by the committee and made public at least four months prior to the early registration date for the meeting or by the first WIFDWC mailing. Completed applications are due by the deadline set by the committee. The awards will be presented at least four weeks prior to the early registration date for the conference by the chair of the committee or designate. The recipients will receive an award of up to US\$500 depending on funding availability. Recipients will be required to make an oral or poster presentation at the meeting for which they received the award. Oral presentations are preferred. The committee may decide to not make an award if no suitable candidates apply.

Select Motions and Decisions _____

1998

Outstanding Achievement Award—established.

1999

Honorary Life Members—members added and provisions discussed (see 1996 Proceedings for historic retrospective on HLM).

Assisting Outside Speakers—amendment passed.

Website—Committee Reports and Meeting synopsis by the Chairperson would be posted; web committee (Baker, Muir, and Adams) formed.

2000

Outstanding Achievement Award—staggered committee established and recommendations made.

Joint Meetings with WFIWC—motions passed to meet in 2004, have dual program chairs, form a planning committee in 2001 for the joint meeting.

2001

Standing Committees—proposal to reorganize Disease Control Committee tabled.

2002

Standing Committees—motion passed to disband the Disease Control Committee and establish a Nursery Pathology Committee.

2004

Outstanding Achievement Award—changes to the Bylaws for this award were proposed and accepted by the membership.

Executive Committee—motion to make Webmaster an official position on the committee was approved.

2007

Standing Committees—motion passed to create both an ad hoc Foliar and Shoot Diseases Committee and a Climate Change Committee.

2008

Digital Proceedings—motion to make WIFDWC proceedings available on the website was approved.

2009

Standing Committees—motion passed to confirm the Foliage and Twig Diseases Committee as a standing committee.

2010

Standing Committees—motion passed to confirm the Climate Change Committee as a standing committee.

Fund Raising—the first WIFDWC Silent Auction was held to raise funds for graduate student travel awards.

2011

Standing Committees—motion passed to add the Student Scholarship Committee as a standing committee.

Business Meeting—motion passed outlining requirements needed to pass a motion by means of an electronic ballot.

2012

Finances—motion passed to hire a tax consultant for WIFDWC taxes.

Student Travel Award—motion passed to recommend to the program chair of each meeting to allow time in the program for each student receiving a travel award to present their work.

Deceased members – a moment of silence or tribute will be given for deceased members.

Regional Reports – motion passed for the Secretary to request regional reports in a standard format prior to the meeting and distribute reports at the meeting.

Joint Meetings with WFIWC- motion passed for the fall 2016 Executive Committee to consider having joint meeting with WFIWC.

2013

Officers- motion passed for Kristen Chadwick to maintain mailing and member list up to date, not the Secretary as specified in the bylaws.

Fund Raising- motion passed to increase regular registration rates by \$15 to go to student travel award.

2014

Joint Meetings with WFIWC- conference chair will send an invitation to the WFIWC chair to hold a joint meeting in 2018 at a location in the US.

2015

No New Motions Passed



PAST ANNUAL MEETING LOCATIONS AND OFFICERS
Meetings and Officers, 1953—2015

#	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	HarrisonHotSpring BC	R. Scharpf	H. Toko	A. Harvey	J. Roff
19	1971	Medford, OR	J. Baranyay	D. Graham	R. Smith	H. Bynum
20	1972	Victoria, BC	P. Lightle	A. McCain	L. Weir	D. Morrison
21	1973	Estes Park, CO	E. Wicker	R. Loomis	R. Gilbertson	J. Laut
22	1974	Monterey, CA	R. Bega	D. Hocking	J. Parmeter	
23	1975	Missoula, MT	H. Whitney	J. Byler	E. Wicker	O. Dooling
24	1976	Coos Bay, OR	L. Roth	K. Russell	L. Weir	J. Hadfield
25	1977	Victoria, BC	D. Graham	J. Laut	E. Nelson	W. Bloomberg
26	1978	Tucson, AZ	R. Smith	D. Drummond	L. Weir	R. Gilbertson
27	1979	Salem, OR	T. Laurent	T. Hinds	B. van der Kamp	L. Weir
28	1980	Pingree Park, CO	R. Gilbertson	O. Dooling	J. Laut	M. Schomaker
29	1981	Vernon, BC	L. Weir	C.G. Shaw III	J. Schwandt	D. Morrison R. Hunt
30	1982	Fallen Leaf Lake, CA	W. Bloomberg	W. Jacobi	E. Hansen	F. Cobb J. Parmeter
31	1983	Coeur d'Alene, ID	J. Laut	S. Dubreuil	D. Johnson	J. Schwandt J. Byler
32	1984	Taos, NM	T. Hinds	R. Hunt	J. Byler	J. Beatty E. Wood
33	1985	Olympia, WA	F. Cobb	W. Thies	R. Edmonds	K. Russell
34	1986	Juneau, AK	K. Russell	S. Cooley	J. Laut	C.G. Shaw III
35	1987	Nanaimo, BC	J. Muir	G. DeNitto	J. Beatty	J. Kumi
36	1988	Park City, UT	J. Byler	B. van der Kamp	J. Pronos	F. Baker
37	1989	Bend, OR	D. Goheen	R. James	E. Hansen	A. Kanaskie

Meetings and Officers, 1953–2015 (cont.)

Annual	Year	Location	Chair-person	Secretary	Treasurer	Program Chair	Local Arrangements	Historian	Web Coordinator
38	1990	Redding, CA	R. Hunt	J. Hoffman	K. Russell	M. Marosy	G. DeNitto		
39	1991	Vernon, BC	A. McCain	J. Muir	K. Russell	R. Hunt	H. Merler		
40	1992	Durango, CO	D. Morrison	S. Frankel	K. Russell	C.G. Shaw III	P. Angwin		
41	1993	Boise, ID	W. Littke	J. Allison	K. Russell	F. Baker	J. Hoffman		
42	1994	Albuquerque, NM	C.G. Shaw III	G. Filip	K. Russell	M. Schultz	D. Conklin T. Rodgers		
43	1995	Whitefish, MT	S. Frankel	R. Mathiasen	K. Russell	R. Mathiasen	J. Taylor J. Schwandt		
44	1996	Hood River, OR	J. Kliejunas	J. Beatty	J. Schwandt	S. Campbell	J. Beatty K. Russel		
45	1997	Prince George, BC	W. Thies	R. Sturrock	J. Schwandt	K. Lewis	R. Reich K. Lewis		
46	1998	Reno, NV	B. Edmonds	L. Trummer	J. Schwandt	G. Filip	J. Hoffman J. Guyon	D. Morrison	J. Adams
47	1999	Breckenridge, CO	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		
56	2008	Missoula, MT	G. DeNitto	F. Baker	J. Schwandt	W. Littke	B. Lockman M. Jackson		

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.

Meetings and Officers, 1953—2015 (cont.)

Annual	Year	Location	Chair-person	Secretary	Treasurer	Program Chair	Local Arrangements	Historian	Web Coordinator
57	2009	Durango, CO	G. Filip	J. Adams	J. Schwandt	D. Shaw	K. Burns B. Jacobi J. Worrall R. Mask J. Blodgett	R. Sturrock	J. Adams
58	2010	Valemount, BC	R. Sturrock	M. Fairweather	J. Schwandt	D. Goheen	M. Cleary R. Reich		
59	2011	Leavenworth, WA	P. Angwin	S. Zeglen	H. Kearns	A. Kanaskie	G. Filip A. Saavedra A. Ramsey-Kroll D. Omdal		
60	2012	Tahoe City, CA	A. Woods	J. Browning	H. Kearns	P Hennon	P. Cannon B. Woodruff		
61	2013	Waterton Lakes National Park, AB	R. Reich	K. Chadwick	H. Kearns	B. Lockman	T. Ramsfield		
62	2014	Cedar City, UT	M. McWilliams	M. Murray	H. Kearns	J. Worrall	J. Guyon		
63	2015	Newport, OR	A. Kanaskie	A. Ramsey	H. Kearns	E. Goheen	K. Chadwick A. Kanaskie G. Filip D. Shaw	S. Romero	

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

In Memory of Eugene P. Van Arsdel

Eugene P. Van Arsdel, PhD. of Tijeras, New Mexico, passed away on March 30, 2015. A memorial for him will appear in the 2017 WIFDWC Proceedings.

Eugene was born in Emmaus, Pennsylvania, on December 4, 1925.

Dr. Van Arsdel worked as a Forester, Pathologist, and Meteorological Epidemiologist, for his entire adult life. He authored and co-authored numerous books and publications, relating to his fields of study. Eugene enjoyed spending time with his dog, reading, asking questions, wood-working, painting, and exploring mountain tops, just to name a few of his many hobbies.

Eugene was a long time resident of Tijeras, and is survived by his son Jonathan van Arsdel and wife Luz Mari-a Avalos of Ri-o Rancho, NM; his daughter Elizabeth VanArsdel of Santa Fe, NM; and other family and friends.

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2015 WIFDWC MEMBERS

Alex Abair

2082 Cordley Hall
Corvallis OR 97331
541-737-5242
abaira@onid.oregonstate.edu
Last Attended 2015

Michelle Agne

Oregon State University
Dept of Forest Engineering, Resources
& Management, 280 Peavy Hall
Corvallis, OR 97331
206-384-9804
Michelle.Agne@oregonstate.edu
Last Attended 2015

Brandon Alveshere

5315 Prairiewood Dr
Bismarck ND 58504
701-426-9115
alvesheb@onid.oregonstate.edu
Last Attended 2015

Peter Angwin

USDA Forest Service
Forest Health Protection
Pacific Southwest Region
3644 Avtech Parkway
Redding, CA 96002
530-226-2436
pangwin@fs.fed.us
Last Attended 2014

Sara Ashiglar

University of Idaho
Dept of Forest, Rangeland,
& Fire Sciences
875 Perimeter Drive MS 1133
Moscow, ID 83844
ashiglar@gmail.com
Last Attended 2014

Tara Barrett

USFS 1133 N. Western Ave
Wenatchee WA 98801
509-664-1715
tbarrett@fs.fed.us
Last Attended 2012

Elisa Becker

Canadian Forest Service NRC
Pacific Forestry Centre
506 West Burnside Road
Victoria, BC V8Z 1M5
250-298-2382
Elisa.Becker@NRCan-RNCan.gc.ca
Last Attended 2015

Peter Beedlow

200 SW 35th Street
Corvallis OR 97333
541-754-4567
beedlow.peter@epa.gov
Last Attended 2015

Maia Beh

University of California, Davis
Division of Agriculture & Natural
Resources
371 Hutchison Hall, 1 Shields Avenue
Davis, CA 95616
530-754-9894
mmbbeh@ucdavis.edu
Last Attended 2012

Christina Benemann

Dept. Botany and Plant Pathology
2082 Cordley Hall
Corvallis OR 97330
925-301-6680
benemans@science.oregonstate.edu
Last Attended 2015

Patrick Bennett

Dept. Botany and Plant Pathology
2029 SE Crystal Cir.
Corvallis OR 97333
951-750-3771
bennetpa@science.oregonstate.edu
Last Attended 2015

James T. Blodgett

USDA Forest Service
Forest Health Protection
Rapid City Service Center
8221 S. Highway 16
Rapid City, SD 57702
605-716-2783
jblodgett@fs.fed.us
Last Attended 2014

Tyler Bourret

One Shields Ave
Davis CA 95616
(206) 351-3953
tbourret@ucdavis.edu
Last Attended 2015

Simren Brar

University of British Columbia
Dept of Forest & Conservation Sciences
3041-2424 Main Mall
Vancouver, BC V6T 1Z4
604-607-4758
brar.simren@gmail.com
Last Attended 2013

Josh Bronson

USDA Forest Service
Forest Health Protection
J. Herbert Stone Nursery
2606 Old Stage Road
Central Point, OR 97502
jjbronson@fs.fed.us
Last Attended 2015

John Browning

Weyerhaeuser Forestry
505 N Pearl St, PO Box 420
Centralia, WA 98531
360-330-1721
john.browning@weyerhaeuser.com
Last Attended 2015

Christine Buhl

2600 State St.
Salem OR 97310
503-945-7396
christine.j.buhl@oregon.gov
Last Attended 2015

Beverly Bulaon

Stanislaus National Forest
19777 Greenley Road
Sonora CA 95370
209-532-3681 X323
bbulaon@fs.fed.us
Last Attended 2012

Kelly Burns

USDA Forest Service
Forest Health Protection
Rocky Mountain Region
740 Simms Street, Golden, CO 80401
303-236-8006
ksburns@fs.fed.us
Last Attended 2014

Kim Camilli

California Dept of Forestry & Fire
Protection
635 N. Santa Rosa
San Luis Obispo, CA 93405
805-550-8583
kim.corella@fire.ca.gov
Last Attended 2012

Phil Cannon

USDA Forest Service
Forest Health Protection
Pacific Southwest Region
1323 Club Drive, Vallejo, CA 94592
707-562-8913
pcannon@fs.fed.us
Last Attended 2012

Kristen L. Chadwick
USDA Forest Service
Forest Health Protection
Westside Service Center
16400 Champion Way
Sandy, OR 97055
503-668-1474
klchadwick@fs.fed.us
Last Attended 2015

Art Chappelka
Auburn University
School of Forestry & Wildlife Sciences
602 Duncan Drive
Auburn, AL 36849
334-844-1047
chappah@auburn.edu
Last Attended 2013

Gary Chastagner
Washington State University
Puyallup Research & Extension Center
2606 West Pioneer
Puyallup, WA 98371
253-445-4528
chastag@wsu.edu
Last Attended 2011

Michelle Cleary
University of Agricultural Sciences
Southern Forest Research Centre
Box 49, 23053 ALNARP, Sweden
040-415181
michelle.cleary@slu.se
Last Attended 2011

Christy Cleaver
Colorado State University
Bioagricultural Sciences & Pest
Management
P.O. Box 310 458 Streamside Drive
Glen Haven, CO 80532
303-907-8718
ccleaver@rams.colostate.edu
Last Attended 2015

Mike Cruickshank
Canadian Forest Service NRC
Pacific Forestry Centre
506 West Burnside Road
Victoria, BC V8Z1M5
250-363-0641
mcruicks@nrcan.gc.ca
Last Attended 2013

Richard Cobb
University of California Davis
530-754-9894
rccobb@ucdavis.edu
Last Attended 2015

Robert Cruz
USDA Forest Service
Forest Health Protection
Intermountain Region
324 25th Street
Ogden, UT 84401
801-625-5162
rcruz@fs.fed.us
Last Attended 2014

Ruben Damian
Colegio de Postgraduados
Km 36.5 Carretera Mexico-Texcoco
56230 Texcoco, Mexico
edamian@colpos.mx
Last Attended 2012

Dixie Daniels
1755 SE Lilly Place
Corvallis OR 97333
865-896-9457
danieldi@onid.oregonstate.edu
Last Attended 2015

Gregg DeNitto
USDA Forest Service
Forest Health Protection
Northern Region, P.O. Box 7669
Missoula, MT 59807
406-329-3637
gdenitto@fs.fed.us
Last Attended 2015

Alvarad Dionicio
Texcoco Mexico
dionicioyganoderma@gmail.com
Last Attended 2012

Megan Dudley
Colorado State University
Bioagricultural Sciences & Pest
Management, 307 University Ave.
Fort Collins, CO 80523
970-222-9558
meg.dudley@rams.colostate.edu
Last Attended 2015

Nick Dudley
Hawaii Agriculture Research Center
70 Loop Road
Kailua, HI 96734
808-262-4041
ndudley@harc-hspa.com
Last Attended 2012

Kelsey Dunnell
2002 35th Street Circle S
Moorhead MN 56560
701-361-8612
dunnellk@onid.oregonstate.edu
Last Attended 2015

Marianne Elliott
Washington State University
Puyallup Research & Extension Center
2606 West Pioneer, Puyallup, WA
98371
253-445-4596
melliott2@wsu.edu
Last Attended 2011

MaryLou Fairweather
USDA Forest Service
Forest Health Protection
Arizona Zone Office
Southwest Forest Science Complex
2500 S. Pine Knoll Drive
Flagstaff, AZ 86001
928-556-2075
mfairweather@fs.fed.us
Last Attended 2014

Lina Farfan
University of British Columbia
Dept Forest Sciences
2424 Main Mall
Vancouver, BC V6T 1Z4
778-928-4346
linafarfan@hotmail.com
Last Attended 2011

Nicolas Feau
University of British Columbia
Forest Sciences Centre
2424 Main Mall
Vancouver, BC V6T 1Z4
feaunico@mail.ubc.ca
Last Attended 2013

Brennan Ferguson
USDA Forest Service FHP
1133 N Western Ave.
Wenatchee WA 98001
509-664-9215
brennanferguson@fs.fed.us
Last Attended 2014

Gregory Filip
USDA Forest Service
Forest Health Protection
Pacific Northwest Region
PO Box 3623, Portland, OR 97204
503-808-2997
gmfilip@fs.fed.us
Last Attended 2015

Risso Fonseca
Universidade Federal de Viçosa (UFV)
Viçosa, Estado de Minas Gerais, Brazil
natalia.smile@yahoo.com.br
Last Attended 2015

Susan Frankel
USDA Forest Service
Pacific Southwest Research Station
800 Buchanan Street, Albany, CA
94710
510-559-6472
sfrankel@fs.fed.us
Last Attended 2015

Martin Frye
University of California - Davis
Tahoe Environmental Research Center
291 Country Club Dr., 3rd Floor TCES
Incline Village, NV 89451
530-546-3014
martinjfrye@gmail.com
Last Attended 2012

Amy Gannon
Montana Dept of Natural Resources &
Conservation, 2705 Spurgin Road
Missoula, MT 59804
406-542-4283
agannon@mt.gov
Last Attended 2012

Matteo Garbelotto
University of California - Berkeley
Forest Pathology & Mycology Lab
54 Mulford Hall
Berkeley, CA 94720
510-643-4282
matteog@berkeley.edu
Last Attended 2013

Matthew Gelderman
University of Alberta
Dept of Renewable Resources
751 General Services Building
Edmonton, AB T6G 2H1
780-993-6938
mgelderm@ualberta.ca
Last Attended 2013

Ellen Michaels Goheen
USDA Forest Health Protection
Pacific Northwest Region
2606 Old Stage Road
Central Point, OR 97502
541-858-6126
egoheen@fs.fed.us
Last Attended 2015

Betsy Goodrich
USDA Forest Health Protection
PNW Forestry Sciences Laboratory
1133 N. Western Avenue
Wenatchee, WA 98801-1229
509-664-9223
agoodrich@fs.fed.us
Last Attended 2015

Tom Gordon
University of California - Davis
Dept of Plant Pathology
One Shields Ave
Davis, CA 95616
530-754-9893
trgordon@ucdavis.edu
Last Attended 2012

Joyce Gould
Alberta Parks Environment &
Sustainable Resource Development
2nd Floor Oxbridge Place 9820-106
Edmonton, AB T5K 2J6
780-427-7702
joyce.gould@gov.ab.ca
Last Attended 2013

Laura Gray
University of Victoria
Centre for Forest Biology
PO Box 3020, STN CSC
Victoria, BC V8W 3N5
250-857-0966
lkgray@gmail.com
Last Attended 2012

Nancy Grulke
USDA Forest Service
Western Wildland Environmental
Threat Assessment Center, 3160 NE 3rd
Street, Prineville, OR 97754
541-416-6583
ngrulke@fs.fed.us
Last Attended 2015

John C. Guyon II
USDA Forest Service
Forest Health Protection
Intermountain Region
4746 S 1900 E, Ogden, UT 84403
801-476-9720 ext 218
jguyon@fs.fed.us
Last Attended 2014

Richard Hamelin
University of British Columbia
Forest Sciences Centre 3032
2424 Main Mall
Vancouver, BC V6T 1Z4
Richard.Hamelin@ubc.ca

John Hanna
USDA Forest Service
Rocky Mountain Research Station
1221 South Main Street
Moscow, ID 83843
208-883-2337
jhanna@fs.fed.us
Last Attended 2015

Chris Hansen
University of Northern British
Columbia, 3333 University Way
Prince George, BC V2N 4Z9
250-960-6659
chansen@unbc.ca
Last Attended 2010

Paul Hennon
USDA Forest Service
PNW Research Station Forestry
Sciences Lab
11305 Glacier Highway
Juneau, AK 99801
907-586-8769
phennon@fs.fed.us
Last Attended 2012

Ted (E.H.) Hogg
Canadian Forest Service NRC
Northern Forestry Centre
5320 122 Street Northwest
Edmonton, Alberta, T6H 3S5
780-435-7225
ted.hogg@NRCan-RNCan.gc.ca
Last Attended 2013

Abbey Hudler
Utah State University
Dept of Wildland Resources
5230 Old Main Hill
Logan, UT 84322
abbeyhudler@yahoo.com
Last Attended 2012

David Hunter
PO Box 324
Forest Grove OR 97116
503-319-0380
ddhunterarborist@aol.com
Last Attended 2015

Karen Hutten
University of Washington
School of Forest Resources
1492 NE Boat Street
Seattle, WA 98105
360-460-5718
huttenk@u.washington.edu
Last Attended 2012

Louis Iverson
USDA Forest Service
Northern Research Station
359 Main Road
Delaware, OH 43015
740-368-0097
liverson@fs.fed.us
Last Attended 2012

Marcus Jackson
USDA Forest Service
Forest Health Protection
Northern Region, 200 E. Broadway
P.O. Box 7669
Missoula, MT 59807
406-329-3282
mbjackson@fs.fed.us
Last Attended 2014

William Jacobi
Colorado State University
Bioagricultural Sciences & Pest
Management, 307 University Ave.
Fort Collins, CO 80523
970-491-6927
william.jacobi@colostate.edu
Last Attended 2013

James Jacobs
USDA Forest Service
Forest Health Protection
New Mexico Zone
333 Broadway Blvd., SE
Albuquerque, NM 87102
505-842-3288
jamesjjacobs@fs.fed.us
Last Attended 2014

Saeideh Jafarpour
Department of Plant Protection, Faculty
of Agricultural Science and
Engineering, College of Agriculture and
Natural Resources, University of Tehran
Karaj Iran 31587-77871
0098 261 2224022-4
sjafarpour@ut.ac.ir
Last Attended 2015

Chamille Jenson
University of California - Davis
Tahoe Environmental Research Center
291 Country Club Drive
Incline Village, NV 89451
530-400-3037
jensencamille@gmail.com
Last Attended 2012

Randy Johnson
USDA Research & Development
1601 North Kent Street
Rosslyn Plaza C 4th Floor
Arlington, VA 22209
703-605-5178
randyjohnson@fs.fed.us
Last Attended 2012

Nathan Johnson
University of Washington
School of Environmental & Forest
Sciences
PO Box 352100, Seattle, WA 98195
781-927-5341
ngjohnso@gmail.com
Last Attended 2011

Tyler Jones
Hawaii Agriculture Research Center
PO Box 100, Kunia, HI 96759
808-927-7508
tylercjohnes@gmail.com
Last Attended 2012

William Jones
USDA Forest Service
Southern Research Station
200 Weaver Blvd
Asheville, NC 28801
828-259-0526
wejones@fs.fed.us
Last Attended 2011

Alan Kanaskie
Oregon Dept of Forestry
Forest Health Management
2600 State Street Bldg D
Salem, OR 97310
503-945-7397
akanaskie@odf.state.or.us
Last Attended 2015

Cynthia Kanner
Montana Dept of Natural Resources &
Conservation, PO Box 489
Darby, MT 59829
cynkanner@gmail.com
Last Attended 2011

Holly Kearns
USDA Forest Health Protection
Pacific Northwest Region
16400 Champion Way
Sandy, OR 97055
503-668-1475
hkearns@fs.fed.us
Last Attended 2015

Rick Kelsey
PNW Research Station
3200 Jefferson Way
Corvallis OR 97331
541-750-7368
rkelsey@fs.fed.us
Last Attended 2015

Mahsa Khorasani
University of Washington
School of Environmental & Forest
Sciences
PO Box 352100, Seattle, WA 98195
mkh2612@u.washington.edu
Last Attended 2012

Mee-Sook Kim
Rocky Mountain Research Station
1221 s. Main Street
Moscow ID 83843
208-883-2362
mkim@fs.fed.us
Last Attended 2015

Jim Kiser
College of Forestry
280 Peavy Hall
Corvallis OR 97331
541-737-2192
jim.kiser@oregonstate.edu
Last Attended 2015

Ned Klopfenstein
USDA Forest Service
Northern Research Station
1221 S. Main Street
Moscow, ID 83843
208-883-2310
nklopfenstein@fs.fed.us
Last Attended 2015

Jennifer Klutsch
University of Alberta
Dept of Renewable Resources
751 General Services Building
Edmonton, AB T6G2H1
780-802-1554
jklutsch@gmail.com
Last Attended 2013

Harry H. Kope
British Columbia Ministry of Forests,
Lands & Natural Resource Operations
9th floor, 727 Fisgard Street
Victoria, BC V8W 1R8
250-387-5225
harry.kope@gov.bc.ca
Last Attended 2014

Barbara Lachenbruch
Dept. of Forest Ecosystems & Society
321 Richardson Hall
Corvallis OR 97331
541-737-4213
barb.lachenbruch@oregonstate.edu
Last Attended 2015

Thomas Lawson
1224 Navellier St El Cerrito
El Cerrito CA 94530
drthomlawson@gmail.com
Last Attended 2015

Jared Leboldus
Dept. of Botany and Plant Pathology
2082 Cordley Hall
Corvallis OR 97331
701-566-4608
lebolduj@science.oregonstate.edu
Last Attended 2015

Anna Leon
Weyerhaeuser Forestry
32901 Weyerhaeuser Way S
Federal Way, WA 98001
253-924-6318
anna_leon@wsu.edu
Last Attended 2015

Kathy Lewis
University of Northern British
Columbia
Ecosystem Science & Management
3333 University Way
Prince George, BC V2N 4Z9
250-960-6659
kathy.lewis@unbc.ca
Last Attended 2015

Blakey Lockman
USDA Forest Health Protection
Northern Region - 1
200 E. Broadway, P.O. Box 7669
Missoula, MT 59807
406-329-3189
blockman@fs.fed.us
Last Attended 2015

Martin MacKenzie
USDA Forest Health Protection
South Sierra – Pacific Southwest
Region, 19777 Greenley Road
Sonora, CA 95370
209-532-3671 ext 242
mmackenzie@fs.fed.us
Last Attended 2012

Tom Maertens
University of British Columbia
Dept of Forest Sciences
2424 Main Mall
Vancouver, BC V6T1Z4
604-710-3410
maertens@forestmail.com
Last Attended 2011

Helen Maffei
USDA Forest Service
Forest Health Protection
Deschutes - Pacific Northwest Region
63095 Deschutes Market
Bend, OR 97701
541-383-5591
hmaffei@fs.fed.us
Last Attended 2015

Beh Maia
University of California – Davis
Division of Agriculture & Natural
Resources, 371 Hutchinson Hall
Davis, CA 95616
530-754-9894
mmbbeh@ucdavis.edu
Last Attended 2011

Patricia Maloney
University of California – Davis
Division of Agriculture & Natural
Resources, 371 Hutchinson Hall
Davis, CA 95616
530-754-9894
pemaloney@ucdavis.edu
Last Attended 2012

Gary Man
FHP 201 14th St SW
Washington DC 20250
703-605-5339
gman@fs.fed.us
Last Attended 2015

Suzanne Marchetti
USDA Forest Health Protection
Gunnison Service Center
216 N Colorado Street
Gunnison, CO 81230
970-642-4448
sbmarchetti@fs.fed.us
Last Attended 2014

Danielle Martin
USDA Forest Health Protection
Morgantown Field Office
180 Canfield Street
Morgantown, WV 26505
304-285-1531
dkmartin@fs.fed.us
Last Attended 2014

Roy Mask
USDA Forest Service
Forest Health Service Center
216 North Colorado
Gunnison, CO 81230
970-642-4446
rmask@fs.fed.us
Last Attended 2011

Robert Mathiasen
Northern Arizona University
School of Forestry
200 East Pine Knoll Drive
PO Box: 15018, Flagstaff, AZ 86011
928-523-0882
robert.mathiasen@nau.edu
Last Attended 2011

Kathleen McKeever
Washington State University
Puyallup Research & Extension Center
2606 West Pioneer
Puyallup, WA 98371
253-445-4623
kmmckeev@wsu.edu
Last Attended 2014

Michael McWilliams
USDA Forest Health Protection
Blue Mountains Forest Insect & Disease
1401 Gekeler Lane,
LaGrande, OR 97850
541-962-6544
michaelgmcwilliams@fs.fed.us
Last Attended 2015

Rosario Medel
University in Xalapa
Forest Research Institute
CP 91070, Xalapa, Veracruz, Mexico
+52 2288421700 ext 13962
Medel.rosario@gmail.com
Last Attended 2012

Heather Mehl
University of California – Davis
Division of Agriculture & Natural
Resources, 371 Hutchinson Hall
Davis, CA 95616
530-320-5475
hkmehl@ucdavis.edu
Last Attended 2012

Pam Melnick
Alberta Environment & Sustainable
Resource Development, 2nd fl
Provincial Building, 4919 - 51 Street
Rocky Mountain House, AB T4T 1B3
403-845-8277
pam.melnick@gov.ab.ca
Last Attended 2013

Jessie Micales-Glaser
USDA Forest Service
Forest Products lab
One Gifford Pinchot Drive
Madison, WI 53726
608-231-9215
jglaeser@fs.fed.us
Last Attended 2012

Maria Mircheva
Sugar Pine Foundation
1458 Mt Rainier Drive
South Lake Tahoe, CA 96150
650-814-9565
maria.mircheva@gmail.com
Last Attended 2012

Bruce Moltzan
USDA Forest Health Protection
National Headquarters
1601 North Kent Street RPC,
7th Floor (FHP)
Arlington, VA 22209
703-605-5344
bmoltzan@fs.fed.us
Last Attended 2012

Jeff Moore
USDA Forest Health Protection
Region 5 – Pacific Southwest Region
1323 Club Drive, Vallejo, CA 94592
530-759-1753
jwmoore@fs.fed.us
Last Attended 2012

Leif Mortenson
Oregon State University
College of Forestry, 204 Peavy Hall
Corvallis, OR 97331
503-901-5666
singletrack82@yahoo.com
Last Attended 2011

Robin Mulvey
USDA Forest Service
Forest Health Protection
Juneau Forestry Sciences Lab-USFS
11175 Auke Lake Way
Juneau, AK 99801
907-586-7971
rlmulvey@fs.fed.us
Last Attended 2014

Michael Murray
British Columbia Ministry of Forests,
Lands & Natural Resource Operations
401-333 Victoria Street
Nelson, BC V1L 4K3
250-825-1173
michael.murray@gov.bc.ca
Last Attended 2015

Colin Myrholm
Canadian Forest Service NRC
Northern Forestry Centre
5320 122 Street Northwest
Edmonton, AB T6H 3S5
780-435-7379
cmyrholm@nrcan.gc.ca
Last Attended 2013

Ahmed Najar
University of Alberta
Dept of Renewable Resources
751 General Services Building
Edmonton, AB T6G 2H1
587-710-0424
najar@ualberta.ca
Last Attended 2013

Sarah Navarro
Oregon State University
Dept of Botany & Plant Pathology
2082 Cordley Hall
Corvallis, OR 97331
541-737-5242
navarros@science.oregonstate.edu
Last Attended 2015

Danny Norlander
Oregon Dept of Forestry
Forest Health Management
2600 State St., Bldg D
Salem, OR 97310
503-945-7310
Danny.norlander@state.or.us
Last Attended 2015

Estelle Noyer
Centre INRA de Nancy-Lorraine
Site de Champenoux
Rue d'Amance - 54280 Champenoux
541-207-2299
enoyer@nancy.inra.fr
Last Attended 2015

Brent Oblinger
USDA Forest Health Protection
Deschutes National Forest
63095 Deschutes Market Rd.
Bend, OR 97701
541-383-5788
boblinger@fs.fed.us
Last Attended 2015

Paul Oester
10507 N McAlister Rd Rm 9
La Grande OR 97850
541-963-1010
paul.t.oester@oregonstate.edu
Last Attended 2015

Forrest L. Oliveria
USDA Forest Health Protection
Alexandria Field Office
2500 Shreveport Highway
Pineville, LA 71360
318-473-7294
foliveria@fs.fed.us
Last Attended 2011

Daniel Omdal
Washington State Dept of Natural
Resources
PO Box 47037 1111 Washington St. SE
Olympia, WA 98504
360-902-1692
dan.omdal@dnr.wa.gov
Last Attended 2015

Donald R. Owen
State of California
Dept of Forestry & Fire Protection
6105 Airport Rd.
Redding, CA 96002
530-224-2494
don.owen@fire.ca.gov
Last Attended 2012

Jennifer Parke
Oregon State University
Dept of Crop & Soil Science
3017 Agriculture & Life Sciences
Building, Corvallis, OR 97331-7306
541-737-8170
Jennifer.Parke@oregonstate.edu
Last Attended 2015

Ebba Peterson
Oregon State University
Dept of Botany & Plant Pathology
2082 Cordley Hall
Corvallis, OR 97331
541-737-5242
petersoe@science.oregonstate.edu
Last Attended 2015

Pamela Phillips
18813 SE Lake Holm Road
Auburn, WA 98092
253-217-8447
pafphillips@comcast.net
Last Attended 2011

Rebecca Powell
Colorado State University
Bioagricultural Sciences & Pest
Management
307 University Avenue
Fort Collins, CO 80523
720-480-8214
rebeccashifler@yahoo.com
Last Attended 2012

Jay Pscheidt
Oregon State University
Dept of Botany & Plant Pathology
2082 Cordley Hall
Corvallis, OR 97331
pscheidj@science.oregonstate.edu
Last Attended 2011

Melodie Putnam
Oregon State University
Dept of Botany & Plant Pathology
2082 Cordley Hall
Corvallis, OR 97331
putnamm@science.oregonstate.edu

Amy Ramsey
Washington State Dept of Natural
Resources, PO Box 47037
1111 Washington St. SE
Olympia, WA 98504
360-902-1309
amy.ramsey@dnr.wa.gov
Last Attended 2015

Tod Ramsfield
Canadian Forest Service NRC
Northern Forestry Centre
5320 122 Street Northwest
Edmonton, AB T6H 3S5
780-435-7394
Tod.Ramsfield@nrcan.gc.ca
Last Attended 2015

Paul Reeser
2082 Cordley Hall
Corvallis OR 97331
541-737-5242
reeserp@science.oregonstate.edu
Last Attended 2015

Richard Reich
British Columbia Ministry of Forests,
Lands & Natural Resource Operations
1044 Fifth Avenue, 5th Floor
Prince George, BC V2L 5G4
250-565-6203
Richard.Reich@gov.bc.ca
Last Attended 2013

Ron Rhatigan
Rogue River Siskiyou NF
Gold Beach RD, 29279 Ellensburg
Avenue, Gold Beach OR 97444
541 247 3683
rrhatigan@fs.fed.us
Last Attended 2015

Gabriela Ritokova
Oregon State University
Dept of Forest Engineering, Resources
& Management, 280 Peavy Hall
Corvallis, OR 97331
415-640-9663
gabriela.ritokova@oregonstate.edu
Last Attended 2015

Medel Rosario
Universidad Veracruzana
Xalapa de Enriquez, Estado de
Veracruz-Llave, Mexico
Medel.rosario@gmail.com
Last Attended 2012

Amy Ross-Davis
USDA Forest Service
Rocky Mountain Research Station
1221 South Main Street
Moscow, ID 83843
208-883-2310
arossdavis@fs.fed.us
Last Attended 2012

Damian Ruben
Texcoco Mexico
edamian@colpos.mx
Last Attended 2012

David Rusch
British Columbia Ministry of Forests,
Lands & Natural Resource Operations
400 - 640 Borland St.
Williams Lake, BC V2G 5G1
250-392-4404
david.rusch@gov.bc.ca
Last Attended 2015

Angel Saavedra
USDA Forest Health Protection
Boise Field Office
1249 S. Vinnell Way, Suite 200
Boise, ID 83709
208-373-4221
alsaavedra@fs.fed.us
Last Attended 2015

Hugh Safford
Region 5 1323 Club Dr.
Vallejo CA 94592
530-219-0898
hughsafford@fs.fed.us
Last Attended 2012

Denis Sandanov
Institute of General & Experimental
Biology, Siberian Branch of Russian
Academy of Sciences, Sakh'yanovoi
str., 6, 670047 Ulan-Ude, Russian
Federation
707-365-3726
denis.sandanov@gmail.com
Last Attended 2012

Bob Schlub
University of Guam
College of Natural & Applied Sciences
Unibetsedat Guahan, UOG Station
Mangilao, Guam 96923
671-735-2089
671-734-3346H
rlschlub@gmail.com
Last Attended 2012

Simon Shamoun
Canadian Forest Service NRC
Pacific Forestry Centre
506 West Burnside Road
Victoria, BC V8Z 1M5
250-298-2358
sshamoun@nrcan.gc.ca
Last Attended 2011

David C. Shaw
Oregon State University
Dept of Forest Engineering, Resources
& Management, FERM 204 Peavy Hall
Corvallis, OR 97331
541-737-2845
dave.shaw@oregonstate.edu
Last Attended 2014

Michael Simpson
USDA Forest Health Protection
Central Oregon Service Center
1001 SW Emkay Drive
Bend, OR 97702
541-383-5575
mlsimpson@fs.fed.us
Last Attended 2012

Laura Sims
University of California, Berkeley
Dept of Environmental Science, Policy,
& Management, 130 Mulford Hall
#3114, Berkeley, CA 94720-3114
simslaura@berkeley.edu
Last Attended 2013

Tom Smith
State of California, Forestry & Fire
Protection, PO Box 944246
Sacramento, CA 94266-2460
916-599-6882
tom.smith@fire.ca.gov
Last Attended 2012

Richard Sniezko
USDA Forest Service
Dorena Tree Improvement Center
34963 Shoreview Road
Cottage Grove, OR 97424
541-767-5716
rsniezko@fs.fed.us
Last Attended 2015

Jane Stewart

Colorado State University
C034 Plant Sciences Building
Fort Collins CO 80523-1177
Jane.stewart@colostate.edu
Last Attended 2015

Jeff Stone

Oregon State University
Dept of Botany & Plant Pathology
2082 Cordley Hall
Corvallis, OR 97331
541-737-5260
stonej@science.oregonstate.edu
Last Attended 2015

Rona Sturrock

Canadian Forest Service NRC
Pacific Forestry Centre
506 Burnside Road West
Victoria, BC V8Z 1M5
250-298-2376
Rona.Sturrock@nrcan.gc.ca
Last Attended 2014

Kat Sweeney

Oregon State University
Dept of Botany & Plant Pathology
2082 Cordley Hall
Corvallis, OR 97331
541-737-3573
Last Attended 2012

Cassandra Swett

University of Maryland
Plant Science & Landscape Architecture
2106 Plant Science Building
College Park, MD 20742
301-405-5586
clswett@umd.edu
Last Attended 2012

Borys Tkacz

USDA Forest Service
Pacific Northwest Research Station
1220 SW 3rd Avenue
Portland, OR 97204
503-808-2115
btkacz@fs.fed.us
Last Attended 2011

Yana Valachovic

University of California Cooperative
Extension, Humboldt County Office
5630 South Broadway
Eureka, CA 95503
707-445-7351
yvala@ucdavis.edu
Last Attended 2012

Rimvys Vasaitis

Swedish Agricultural University
Dept of Forest Mycology & Plant
Pathology, Skoglig mykologi och
växtpatologi, Box 7026, 750 07
Uppsala, Sweden
018-671874
Rimvys.Vasaitis@slu.se
Last Attended 2011

Detlev Vogler

USDA Forest Service
PSW Station, Institute of Forest
Genetics, 2480 Carson Road
Placerville, CA 95667-5107
530-621-6881
dvogler@fs.fed.us
Last Attended 2012

Joan Webber

Forest Research - Forestry Commission
Farnham, United Kingdom
Last Attended 2015

Cedar Welsh

University of Victoria
Pacific Institute for Climate Solutions
4741 Soucie Ave
Terrace, BC V8G 2E9
250-641-1770
cedar.welsh@unbc.ca
Last Attended 2014

Nicholas Wilhelmi

804 SW 11th
Corvallis OR 97330
707-407-9325
nicholas.wilhelmi@oregonstate.edu
Last Attended 2015

Beth Willhite

USDA Forest Health Protection
Westside Service Center
16400 Champion Way
Sandy, OR 97055
503-668-1477
bwillhite@fs.fed.us
Last Attended 2015

Nari Williams

Te Papa Tipu Innovation Park,
49 Sala Street, Rotorua
+64 7 343 5535
nari.williams@scionresearch.com
Last Attended 2015

Bill Woodruff

USDA Forest Health Protection
NE CA Shared Service Area
2550 Riverside Drive
Susanville, CA 96130
530-252-6680
wwoodruff@fs.fed.us
Last Attended 2014

Alex Woods

British Columbia Ministry of Forests,
Lands & Natural Resource Operations
PO Box #5000, 3726 Alfred Avenue
Smithers, BC V0J 2N0
250-847-6382
Alex.Woods@gov.bc.ca
Last Attended 2015

Jim Worrall

USDA Forest Health Protection
Rocky Mountain Region
216 North Colorado
Gunnison, CO 81230
970-642-1166
jworrall@fs.fed.us
Last Attended 2014

Jessica Wright

USDA Forest Service
Pacific Southwest Research Station
1731 Research Park Drive
Davis, CA 95618
530-759-1742
jessicawwright@fs.fed.us
Last Attended 2012

Yun Wu

USDA Forest Health Protection
Northeastern Region
180 Canfield Street
Morgantown, WV 26505
304-285-1594
ywu@fs.fed.us
Last Attended 2012

Lan "Sky" Yung-Hsiang

Dept. of Forest Ecosystems & Society
322 Richardson Hall
Corvallis OR 97331
541-602-4555
skylan12@gmail.com
Last Attended 2015

Paul Zambino

USDA Forest Health Protection
Northern Region
3815 N Schreiber Way
Coeur d'Alene, ID 83815
208-765-7493
pzambino@fs.fed.us
Last Attended 2014

Stefan Zeglen
British Columbia Ministry of Forests,
Lands & Natural Resource Operations
2080 Labieux Road
Nanaimo, BC V9T 6J9
250-751-7108
stefan.zeglen@gov.bc.ca
Last Attended 2015

HONORARY LIFE MEMBERS 2015

Judy Adams
Fort Collins, CO
970-834-2413
tjmmadams@aol.com

Paul Aho
Corvallis, OR
541-752-3204

Norm Alexander
Chilliwack BC
604-824-2516
normalex@telus.net

Ed Andrews

Fred Baker
Max, MN 56659
218-659-2612
fab384@gmail.com

Jerry Beatty
Portland, OR 97202
503-810-8723
jeromebeatty@gmail.com

Clive Brasier
Farnham Surrey, UK GU10 4LH

Jim Byler
Dalton Gardens, ID 83815
208-972-7442
jjbyler@aol.com

Sally Campbell
Arlington, OR 97202
503-810-8717
sallyjcampbell51@gmail.com

David Conklin
Albuquerque, NM 87102
505-842-3288
david.conklin@lifeboatearth.org

Robert L. Edmonds
Seattle, WA 98105
206-523-6913
bobe@u.washington.edu

David Etheridge
Victoria, BC V8N 3B6

Lowell Farmer
Mount Vernon WA

Mike Finnis
Victoria, BC V8S 1V3

Brian Geils
Flagstaff, AZ 86001
928-556-2076
bgeils@npgcable.com

Linnea Gillman
Denver, CO 80236

James Ginns
Penticton, BC V2A 8T8
250-492-9610
ginnsj@shaw.ca

Don Goheen
Central Point, OR 97530
541-899-1449
edgoheen@jeffnet.org

Don Graham
Vancouver, WA 98662
360-892-8811

Jim Hadfield
East Wenatchee, WA 98802
509-884-4732
nitaandjim@msn.com

Susan K. Hagle
Harper, ID 83552
208-926-4545
2lazyh@wildblue.net

Everett Hansen
Corvallis, OR 97331
541-737-5243
hansene@science.oregonstate.edu

John H. Hart
Cheyenne, WY 82009
307-630-5202
huntwyoming@aol.com

Alan Harvey
West Richland, WA 99353
509-628-3124
asharvey100@msn.com

Bob Harvey
Frenchtown Township Maine

Diane Hildebrand
Arvada, CO 80005
360-903-2891
hildebranddiane@yahoo.com

Ray Hoff
Moscow, ID 83483

James T. Hoffman
Boise, ID 83703
208 373-4221

John Hopkins
Victoria, BC
250-595-5739

Richard Hunt
Hunt, BC V8P 5M5
778-430-5904
ribicola@gmail.com

Bob James
Vancouver, WA 98661
360-936-5658
treejpathman66@yahoo.com

David Johnson
Lakewood, CO 80288

John King
Victoria, BC V8W 9C2
250-387-6476
king.forgen@gmail.com

John Kliejunas
Kent, WA 98042
925-682-4825
kliejunas@comcast.net

John Laut
Mesa, AZ 85205
480-620-3402
johnlaut@gmail.com

Leon Lemadeleine
Morgan, UT 84050
801-845-9173

Paul Lightle

Will Littke
Fall City, WA 98024
425-443-2685
will.littke@comcast.net

Katy Mallams
Central Point, OR 97502
541-664-4615
k.mallams1@gmail.com

Otis Maloy
Moscow, ID 83843
208-883-0940
otismaloy@cpinternet.com

Walter Mark
La Pine, OR 97739
805-305-2553
wmark@calpoly.edu

Neil E. Martin
Moscow, ID 83843
208-882-7049
jandmart@moscow.com

Geral McDonald
Pullman, WA 99163
509-332-0352
geral.mcdonald4@gmail.com

Duncan Morrison
Saainchton, BC V8M 1S8
250-652-3281
armillaria@shaw.ca

John Muir
Victoria, BC V9A 2J6
250-477-1805
johnmuir@consultant.com

Earl Nelson
Redmond, OR 97756
541-504-0685
bigearl35@aol.com

Thomas H. Nicholls
Fifield, WI 54524
715 762-3076
nicho002@umn.edu

Vidar Nordin
Ottawa, ON K1P 5W5
613-234-7478
vidar.nordin@gmail.com

Don Norris

Steve Oak
Asheville, NC 28803
828-298-1045
swoclo@charter.net

Art Partridge

Glenn Peterson
Lincoln, NE 68503
402-464-3696

Roger S. Peterson
Santa Fe, NM 87505-7502
505-983-7559
RogPete@aol.com

John Pronos
Sonora, CA 95370
209-532-6221
johnpronos@gmail.com

Jerry W. Riffle
Syracuse, IN 46567
574-457-3065

Kenelm Russell
Olympia, WA 98506
360-943-8199
kenelmrussell@msn.com

Robert Scharpf
Placerville, CA 95667
530-622-8315
quartzhillvineyard@gmail.com

Craig Schmitt
La Grande, OR 97850
541-962-6544
schmitc@eoni.com

Mike Schomaker
LaPorte, CO 80535
970-420-8658
Michael.Schomaker@colostate.edu

John W. Schwandt
Coeur d'Alene, ID 83815
208-765-3403
jwschwandt@gmail.com

Mike Sharon

Terry Shaw
Powell Butte, OR
541-447-5836
cgsarchxx@aol.com

Wayne Sinclair
Ithaca, NY 14850
was1@cornell.edu

Pritam Singh
Orleans Ontario
250-442-2419

Eric L. Smith
Fort Collins, CO 80526-8121
970-295-5841
elsmith@fs.fed.us

Richard B Smith
Victoria, BC
250-477-2801

Richard S Smith
Livermore CA

Michael Srago
El Cerrito, CA 93540
510-232-7092
msrago@comcast.net

Jack Sutherland
Victoria, BC V8R 5V9
JackSutherland@shaw.ca

Al Tegethoff
Tucson, AZ 85749

Walt Thies
Corvallis, OR 97330
541-752-5214
wgthies@gmail.com

Bob Tinnin
Portland, OR
Last Attended 2003

Jim Trappe
Corvallis, OR

Lori Trummer
sirisundri@gmail.com

Bart Van Der Kamp
Delta, BC V4K2C5
604 946-4673
vdkamp@telus.net

Allen Van Sickle
Victoria, BC V8N 5L6
250-721-0734

Eugene VanArsdel
Tijeras, NM 87059
505-286-4116
epvan@q.com

Gordon Wallis
Victoria, BC
250-479-6581
Last Attended 1983

Conrad Wessela

Roy Whitney
Calgary, AB T2K 1S8
403-284-5650
drroot@telus.net

Stuart Whitney
Victoria, BC V9A 7K2
250-642-5546
stuwitney@shaw.ca

Ralph Williams
Nampa, ID 83686-9408

Ed Wood

Bratislav Zak

DECEASED MEMBERS

Stuart "Stuie" Andrews
Jesse Bedwell
Robert Bega
Warren Benedict
John Bier
Richard Bingham
Bill Bloomberg
Roy Bloomstrom
Thomas "Buck" Buchannan
Don Buckland
Hubert "Hart" Bynum
Elmer Canfield
Fields Cobb
Ross Davidson
Oscar Dooling
Charles Driver
Norm Engelhart
Ray Foster
Dave French
Alvin Funk
Robert Lee Gilbertson
Lake S. Gill
Clarence "Clancy" Gordon

John Gynn
John Hansbrough
Hans Hansen
Homer Hartman
George Harvey
Frank G. Hawksworth
Dwight Hester
Tommy Hinds
Yasuyuki Hiratsuka
Brenton Howard
John Hunt
Paul Keener
James Kimmey
Andrea Koonce
Tom Laurent
Don Leaphart
Tom McGrath
Neil E. McGregor
Jim Mielke
D. Reed Miller
Alex Molnar
Vergil Moss
Harrold Offord

Nagy Oshima
Lee Paine
John Palmer
John "Dick" Parmeter
Fred Peet
Clarence Quick
Jack Roff
Lew Roth
Keith Schea
Dave Schultz
Charles G. Shaw
Albert Slipp
Willhelm Solheim
Albert Stage
James Stewart
Phil Thomas
Willis Wagener
Charles "Doc" Waters
Larry Weir
Ed Wicker
John Woo
Ernest Wright
Wolf Ziller



Back Row (L-R): Walter Thies, Richard Sniezko, Greg Filip, Ron Rhatigan, Dan Omdal, Gregg DeNitto
Front Row (L-R): David Hunter, Amy Ramsey, Nari Williams, Jane Stewart



Back Row (L-R): Zolton Bair, Michelle Agne, Mike McWilliams, Patrick Bennett
Middle Row (L-R): Tod Ramsfield, Peter Beedlow, Yung-Hsiang Lan (Sky), Christine Benemann, Sarah Navarro, Anna Leon
Front Row (L-R): Natalia Risso Fonseca, Mee-Sook Kim



Back Row: David Rusch

Middle Row (L-R): Nicholas Wilhelmi, Brent Oblinger, Ellen Goheen, Stefan Zeglen, Dave Shaw, Willis Littke, Michael Murray

Front Row (L-R): Christy Cleaver, Meg Dudley, Kathy Lewis, Joshua Bronson, Gabriela Ritokova



Back Row (L-R): Clive Brasier, Brandon Alveshere, Everett Hansen, Alan Kanaskie, Alex Woods
Middle Row (L-R): Jared LeBoldus, John Hanna, John Browning, Blakey Lockman, Alex Abair
Front Row (L-R): Betsy Goodrich, Kristen Chadwick, Kelsey Dunnel, Estelle Noyer, Joan Webber



Back Row (L-R): Don Goheen, Beth Willhite, Barbara Lachenbruch, Melodie Putnam, Paul Reeser
Middle Row (L-R): Angel Saavedra, Jerry Beatty, Mike McWilliams, Saeideh Jafarpoor, Rick Meinzer, Helen Maffei
Front Row (L-R): Danny Norlander, Dixie Daniels, Sally Campbell, Tyler Bourret, Paul Oester

