

***Proceedings of the 58<sup>th</sup> Annual  
Western International Forest Disease  
Work Conference***

**October 4-8, 2010  
Valemount, British Columbia**





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*October 4-8, 2010  
Valemount, British Columbia  
Best Western Valemount*

**Compiled by:**

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and

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## Chair's Opening Remarks

Rona Sturrock<sup>1</sup>

Good morning WIFDWC members, guests, and friends. On behalf of the 2010 Organizing Committee, I welcome you all to the 58<sup>th</sup> annual meeting of the Western International Forest Disease Work Conference here in beautiful Valemount, British Columbia.

If this is your very first time at WIFDWC and you are engaged somehow in the field of forest pathology, then you are now, according to WIFDWC's bylaws, a member of this great organization. The OC and I would like to give special welcome to all the graduate students, and aspiring graduate students, who've made the long trek here; also to the retired members, both recent retirees and long-timers; and to those who've come an especially long distance and from as far away as Sweden. You may be interested to know that we have a total of 70+ registered participants, representing several agencies and institutions, including the BC Ministry of Forests and Range; US State Forestry Departments; the USDA Forest Service, the Canadian Forest Service, Universities, and some Forest Industry folks.

As Chairperson for this meeting I want to personally acknowledge the efforts and hard work of all my fellow members of the Organizing Committee including Michelle Cleary and Richard Reich and their legion of 'volunteers', Mary Lou Fairweather, John Schwandt, Don Goheen, Michael Murray, Simren Brar, and Judy Adams. These folks have made this meeting possible.

Many of you may know that, as of January 2011, our very own Michelle Cleary will be taking a one-year leave of absence from the BCMFR to work with Jan Stenlid's group in Uppsala, Sweden on a new dieback disease of common ash trees caused by a fungus called *Chalara fraxinea*. I encourage you to chat with Michelle about this adventure while you're

<sup>1</sup>In: Fairweather, M. Comp. 2011. Proceedings of the 58th Annual Western International Forest Disease Work Conference; 2010 October 4-8; Valemount, BC. US Forest Service, AZ Zone Forest Health, Flagstaff, AZ.

<sup>1</sup>Rona Sturrock, Natural Resources Canada, Canadian Forest Service, Pacific Forestry Centre, Victoria, British Columbia, Canada.

here this week, if you can get her to stay still that is! Also, Don Goheen has made it known that he will be retiring in early 2011 and we want to thank Don for ably handling the program, including some dizzying last minute changes. I cannot mention Don's name without also recognizing and congratulating Don and Ellen on their 26<sup>th</sup> wedding anniversary, which they are celebrating this very day!

At this time I would like to ask everyone to now join me in observing a moment of silence for WIFDWC members who passed away over the last year or two, we know of two such individuals – Tom McGrath from Texas and Andi Koonce from California.

### MOMENT OF SILENCE

Before I move on to some announcements and introduce our special guest speakers, I'd like to propose that, in keeping with the fact that it's 'Random Acts of Poetry Week' here in Canada, we do as much as we can to celebrate and observe 'Random Acts of Pathology', here in the Robson Valley. I have a feeling that Michelle may consider it more like random acts of Mother Nature, right Michelle??!!





# Panel

## Economic, Ecological, and Social Impacts of Forest Diseases



Mount Robson Provincial Park (Wofratz 2007) Wikimedia Commons





## Are Managed Stands in BC Meeting Expectations? If Not, Why Not? And What Are The Implications?

Alex Woods<sup>1</sup>

British Columbia's (BC) forested land-base of 63 million hectares is 95 percent publicly owned. Responsibility for the management of that large forested area is shared between the provincial government and forest licensees. Volume based tenure agreements form the basis of management for the majority of public forest land in the province. Under these agreements, licensees are responsible for reforesting the areas they harvest back to a state called "free-growing", one of the most influential components of forest policy in British Columbia. The policy and practices associated with free-growing essentially dictate the initial species composition and density of managed stands throughout the province. To achieve the free-growing condition, harvested sites must be reforested with a suitable number of well-spaced trees, of minimum height that are ecologically appropriate to the site and free from brush competition. After free-growing is declared, possibly as early as age 7, stands are not likely assessed again until after age 60. Rather than monitoring stand conditions over time, the land owner uses growth and yield models, primarily the Tree and Stand Simulator (TASS) (Mitchell 1975), to project yield post-free-growing. These model outputs drive managed stand assumptions in Timber Supply Reviews (TSRs). So the management decisions associated with the achievement of free-growing can have a large influence on forest conditions in the mid and long-term. There currently is no government lead formal re-evaluation of managed stands post-free-growing in BC.

Since early 2005, the Forest and Range Evaluation Program (FREP) in conjunction with the provincial forest health program has led an investigation of the condition of managed stands post achievement of the free-growing milestone. A series of intensive evaluations in five Timber Supply Areas (TSAs) across the province (figure 1) have been conducted. The essential findings from those studies are presented in this report. The first of these intensive

In: Fairweather, M. Comp. 2011. Proceedings of the 58th Annual Western International Forest Disease Work Conference; 2010 October 4-8; Valemout, BC. US Forest Service, AZ Zone Forest Health, Flagstaff, AZ.

<sup>1</sup>Alex J. Woods, British Columbia Forest Service, British Columbia, Canada.

evaluations has been published (Woods and Bergerud 2008) and serves as a guide for future reports.

As a result of these investigations a young stand monitoring protocol has recently been developed to help fill the current knowledge gap. Stand Development Monitoring (SDM) involves a second look, 10+ years post-free-growing. The overall objectives of SDM are first to look back at previous stand condition as determined at free-growing and see if stands are on assumed growth trajectories. Second, SDM determines current yield in a manner that can be compared to growth and yield model projections. Third, SDM quantifies species specific impacts due to forest damaging agents.



**Figure 1**—Location of five intensively studied Timber Supply Areas where 30-60 samples of post-free-growing stands were assessed.

The SDM protocol has been developed and piloted throughout BC for 2 years. The following five basic stand attributes have been assessed in the intensive studies:

1. Density (stocking of free-growing, well-spaced and total stems)
2. Species composition (based on inventory labels)
3. Volume (current volume as compared to model projections)
4. Site index
5. Forest health

This report covers four of these basic attributes as determined from assessments made in 266 randomly selected post-free-growing stands across 5 TSAs in BC. Volume estimation procedures have been refined in the SDM protocol since these intensive studies were conducted, and are not reported here. This report provides insight into how some of those stand attributes have changed since last assessed as free-growing. Some of the potential implications of changes in average stand conditions discovered through this work include:

- Losses to timber supply
- Unrealized genetic gain
- Uncertain basis for certification of sustainably managed forests
- Carbon sequestration rates found to be less than expected.

Each of these possible implications is discussed in more detail.

## METHODS

### Stand Selection

In this report the term 'stand' has been considered synonymous with 'inventory polygon'. An inventory

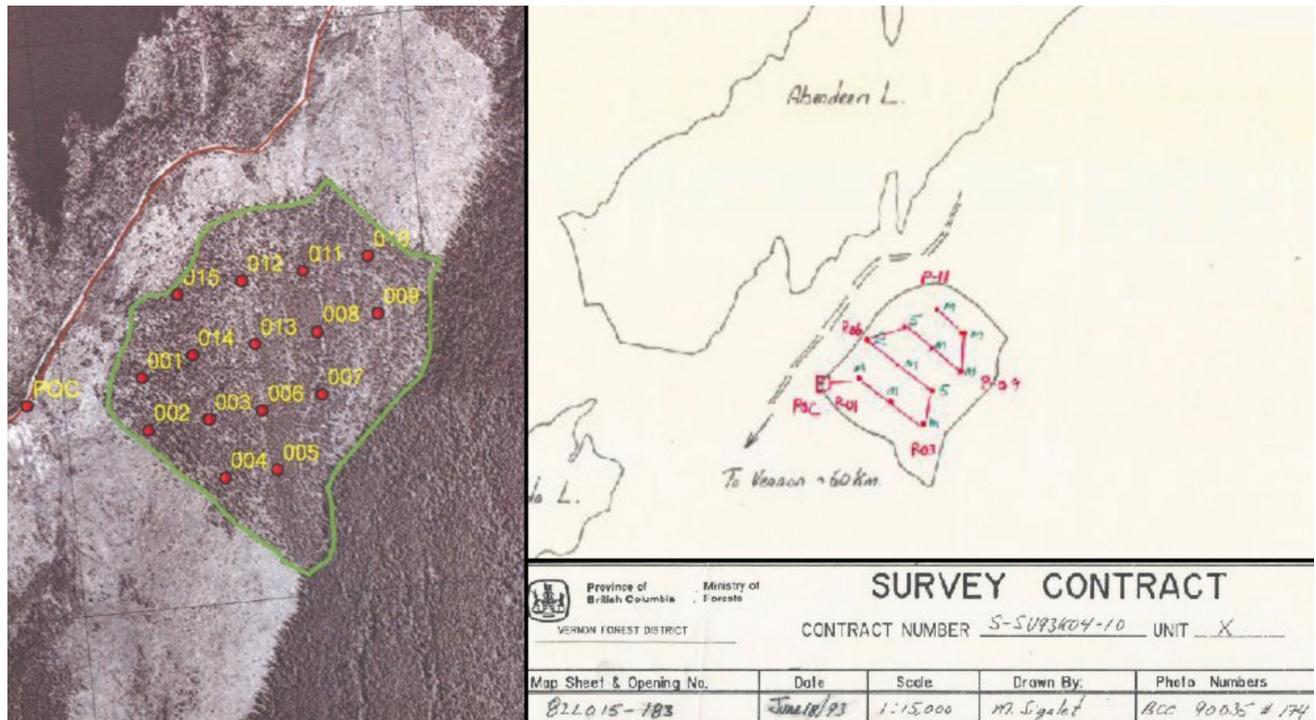
polygon is a homogeneous unit of forest land based on leading species and stocking as determined at declaration. The BC Ministry of Forests and Range's RESULTS database was used to determine the population of all stands (polygons) designated as free-growing in the selected TSAs. Sort procedures were then used to limit the sample population to stands with over the period 1995-2001 (late). In the Headwaters District the 30 stand sample was confined to those stands surveyed for free-growing in the 1995-2001 period.

For each stand, data from the original silviculture survey as reported in RESULTS and the opening file were compared with a follow-up contemporary free-growing survey.

Opening file maps showing silviculture survey location and strata boundaries were referenced to ensure the current survey covered as closely as possible the same areas originally surveyed and declared (figure 2).

### Within Stand Sampling

1. In each stand, a target of fifteen 3.99 m radius plots were located in the largest strata on a systematic 100 x 100 m grid starting from a clearly marked and photo-identifiable point of commencement.



**Figure 2**—Plot map of a surveyed stand illustrating the location of 15 silviculture survey plots established in 2006 (left) and the original silviculture survey plot locations from the pre-stand tending survey that was used to designate this stand as free-growing (right).

2. All trees, including broadleaves, taller than 1.3 m were tallied into one of three height classes (< 2 m, 2-4 m and > 4 m) based on species and estimated height class.

3. Well-spaced and free-growing trees were tallied in the field for both groups of stands. Lower confidence limits about the mean for both well-spaced and free-growing stems were determined using the calculation card for silviculture survey confidence limits, FS 1138A.

4. All healthy well-spaced trees had their height recorded to the nearest 10 cm and diameter at breast height (dbh) recorded to the nearest 0.1 cm. Their species was recorded and their health determined using the current free-growing damage criteria.

5. Pest incidence occurring on all trees by species, height class, and the responsible damaging agent was recorded. All dead conifer trees were identified by species, and where discernable, cause of death and whether they were planted.

6. Strict adherence to the current free-growing damage criteria was applied, including the revised defoliation damage standard and the multiplier rules for all root diseases.

7. For each stand, site index was determined based on one sample tree per plot using a 3.99 m radius plot.

8. All stands were classified using the Biogeoclimatic Ecosystem Classification (BEC) system to the zone level (Pojar et al. 1987).

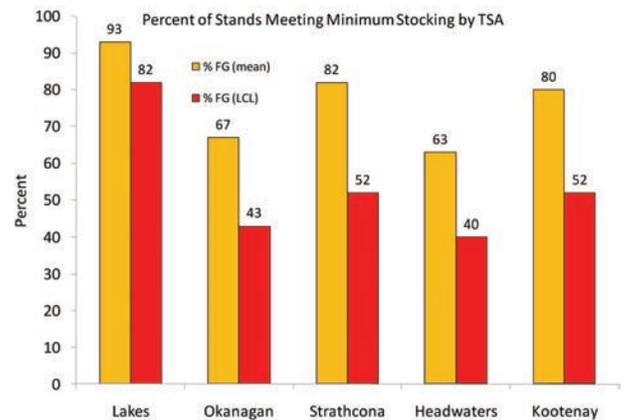
## RESULTS AND DISCUSSION

The average stand age at the time of declaration across all five sampled TSAs was just over 15 years. The average age of stands at the time they were assessed in our survey varied from 23.6 years in the SBS biogeoclimatic zone to 28.2 in the ICH zone with an overall average of 26.5 years.

### Stand Density

Pooled results from the five intensive surveys show considerable differences among BEC zones in stand density trends between the two assessments (table 1). In both the coastal western hemlock (CWH) and sub-boreal spruce (SBS) zones, total stand densities

increased by 9 and 4 percent respectively. In the Engelmann spruce sub-alpine fir (ESSF), interior cedar hemlock (ICH), interior Douglas-fir (IDF) and montane spruce (MS) zones, total densities dropped by 32 percent or more since declaration. Changes in well-spaced densities among BEC zones followed a similar pattern. The proportion of stands that continue to meet the minimum stocking threshold of free-growing stems has varied among TSAs (figure 3) with the SBS dominated Lakes TSA maintaining the most.



**Figure 3**—Proportion of previously declared free-growing stands that continue to meet minimum stocking thresholds of free-growing stems, based on the mean and lower confidence limit (LCL) decision rules, in five sampled TSAs, 10+ years post-declaration.

The growth and yield model TASS (Tree and Stand Simulator) and its derivative TIPSYP (Table Interpolated Projected Stand Yield) are used to project stand yield post-free-growing for managed stands throughout the province. Based on an alpha version of TIPSYP that includes well-spaced density as an output (TIPSYP Version 4.2a: 14 October 16, 2009) a slight decrease in WS density and an increase in FG density are projected for stands between ages 15-30 years (figure 4). The TIPSYP projections presented are for a theoretical lodgepole pine leading stand and although the species is extensively managed in BC it may not be appropriate to extrapolate to other species. We have not yet investigated differences in density relationships by tree species within the model. If, however, those modelled relationships are similar to that of lodgepole pine, there may be a basic divergence between actual stand conditions and those modelled in our provincial system. In other words, according to the growth and yield model that is used in nearly every timber supply review in the province, all of the bars in Figure 3 should be at or close to 100 percent.

**Table 1**—Stand density (sph) attributes compared by BEC Zone at Declaration and at FREP Survey.

	BEC Zone						Total or Mean
	CWH	ESSF	ICH	IDF	MS	SBS	
<b>Number of Stands</b>	58	44	74	3	27	60	266
<b>Total at Declaration</b>	2700	3860	5289	9250	4274	3544	4036
<b>Total Density (Survey)</b>	2947	2263	3596	4394	2908	3671	3190
<b>Percent Change in Total Density</b>	+9%	-41%	-32%	-52%	-32%	+4%	-21%
<b>Well-spaced Change from Declaration</b>	+6*	-154	-241	-351	-252	+35*	-113
<b>Free-growing Change from Declaration</b>	-80	-33*	-138	-116*	-188	+80	-63

\* Value not statistically greater than zero.

These are all simple means. Positive differences (+) mean that the density has increased from declaration to time of survey, negative numbers (-) means that the total density has decreased.

The stands surveyed in these five TSAs were all at or above the minimum density threshold at age 15, or they would not have been reported as free-growing. By age 26, stands within the CWH, ICH, and MS BEC zones experienced a considerable drop in free-growing density (table 1), while stands in the SBS dominant zone in the Lakes TSA experienced an increase.

### Species Composition

With the exception of the lodgepole pine dominated Lakes TSA, there were changes in the leading species of inventory labels in approximately 20 percent of sampled stands (table 2). Changes in leading species were due to both natural ingress and to species specific forest health agents such as the mountain pine beetle. Such changes in leading species are not typically projected in managed stand productivity estimates.

Hemlock leading stands at the time of declaration underwent the least changes (9 percent) while stands dominated by western red cedar (CWR) and coastal Douglas-fir (FDC) at the time of declaration had the greatest change (36 percent). Pine and spruce leading stands in the interior lost their leading species role in 11 percent and 17 percent of the stands respectively (data not shown).

### Site Index

Site index estimates have not been consistently reported in the past silvicultural records. Many earlier estimates have been either qualitative (i.e. good, medium or poor), or appear to have been converted from tables. Our field sampled site index estimates based on the growth intercept method have tended to

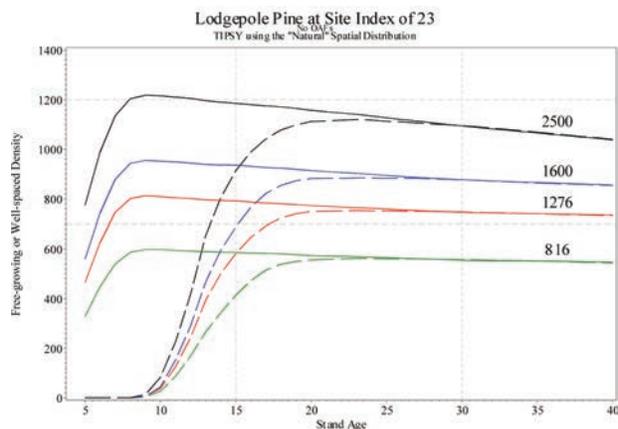
be higher than those previously reported in TSRs for the five sampled TSAs.

**Table 2**—Change in leading species based on inventory labels by Timber Supply Area.

Timber Supply Area:	Did Leading Species Change?			Total Count
	No	Yes	Percent	
<b>Lakes</b>	60	0	0	60
<b>Okanagan</b>	48	12	20%	60
<b>Strathcona</b>	47	11	19%	58
<b>Kootenay</b>	45	13	22%	58
<b>Headwaters</b>	22	8	27%	30
<b>Total</b>	222	44	17%	266

### Forest Health

A wide variety of forest health agents were found in the five TSAs. In order to simplify analyses, forest health agents were placed into groups. These groups were formed loosely on a combination of their biologic mode of action and on the extent of damage they would cause. Root diseases, arguably the most serious damaging agent of managed stands given their persistence and impact, were found in over 38 percent of the sampled stands over all five TSAs (figure 5). Several biotic forest health agents cause deformities including spruce leader weevil, pine leader weevil, and atopellis canker. These agents were all grouped together as deformities which are not generally responsible for mortality. Abiotic damage including forking due to frost, snow breakage, and hail damage was the most widely distributed damage category occurring in about 66 percent of sampled stands. Death for unknown reasons was reported in 20 percent of sampled stands.



**Figure 4**—TIPSy projections of well-spaced (solid line) and free-growing density (dashed line) for a theoretical lodgepole pine stand, using the natural stand stem distribution, at site index 23, no operational adjustment factors and four nominal densities. Target and minimum free-growing density values are identified as are stand ages 15 and 30 years, for reference (Courtesy of Wendy Bergerud).

The mean percent incidence of each of the damage agent groups both within the stands where the agent occurred as well as over all sampled stands are reported in Table 3. Hard pine rusts were the most widely represented forest health agent across all five sampled TSAs even though the damage agent group was not present in the coastal Strathcona TSA. No single damage agent had a higher incidence in any stand than the combined hard pine rusts. Root diseases were found in over 1.1 percent of all trees in the combined population.

**Table 3**—Mean, maximum and minimum incidence of forest health agent damage group in affected stands and mean incidence overall 266 sampled stands combined for the five intensively sampled TSAs.

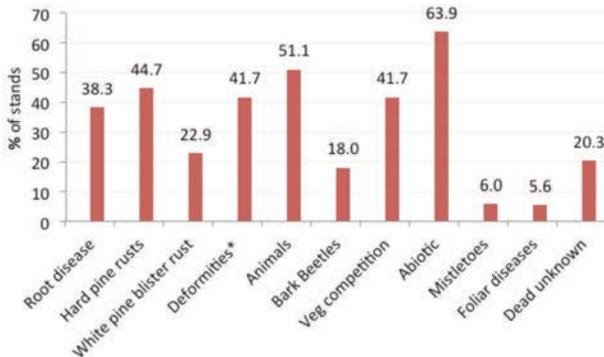
Damage agent group	Root disease	Hard pine rust	Pw blister rust	Deform.	Animal	Bark beetles	Veg. comp.	Abiotic damage	Dwarf mistletoe	Foliar disease	Dead unknown cause
<b>Incidence in affected stands</b>											
# of stands affected	102	119	61	111	136	48	111	170	16	15	54
Mean	2.8	9.3	2.2	3.9	2.6	2.4	8.4	3.4	2.4	1.2	1.1
Max	23	45.9	12.7	26.3	18.5	19.8	50	30.8	10.4	5.1	4.4
Min	0.1	0.3	0.2	0.3	0.2	0.2	0.3	0.1	0.3	0.2	0.1
<b>Incidence in all stands, whether affected by health agent or not</b>											
Mean	1.1	4.2	0.5	1.6	1.3	0.4	3.5	2.2	0.1	0.1	0.2

## Implications

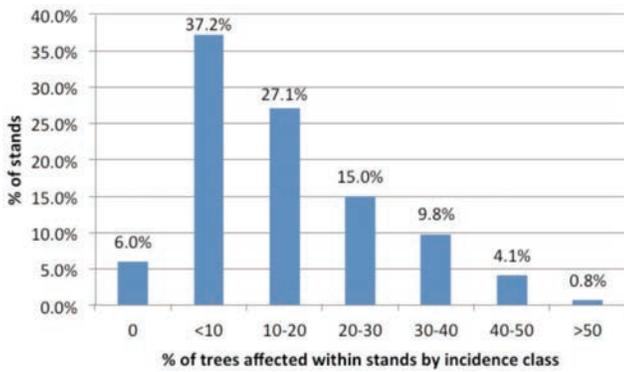
The remainder of this report involves a hypothetical examination of the preliminary results found in the five intensive studies and what they could mean for forest management in BC. Actual stand conditions 10+ years post free-growing may not meet the basic density projections of the growth and yield model used throughout BC. A primary reason why they may not is the incidence and impacts of both biotic and abiotic forest health agents. Those same agents have contributed to changes in leading species in 20 percent of managed stands. If managed stands continue to not meet basic timber productivity expectations the following implications are possible:

1. Losses to timber supply
2. Unrealized genetic gains
3. Certification of sustainably managed forests questioned, and
4. Carbon sequestration less than expected

Six percent of the 266 sampled stands contained no forest health damage agents severe enough to deem the tree unacceptable based on current free-growing damage criteria (figure 6). Over 60 percent of stands had less than 20 percent of the total conifer tree count damaged by forest health agents. Close to five percent of all stands surveyed had 40 percent or more of their trees damaged to a degree that would make them unacceptable based on current free-growing damage criteria.



**Figure 5**—Proportion of 266 stands (sampled over 5 TSAs) affected by forest health damaging agents, grouped on the basis of biological function and severity of damage.



**Figure 6**—Percent of the 266 sampled stands over five TSAs affected by combined damage agents, grouped into 10 percent combined incidence classes. Only the most severe damaging agent was recorded per tree allowing for a summation of damage agent incidence per stand.

### Losses to Timber Supply

A primary reason why managed stands may be less productive than TASS/TIPSY projections involves the calibration of mortality functions. Mortality functions are generally considered as one of the most influential yet challenging aspects of accurate stand growth modeling. No single factor, not site index, species composition, nor growth a rate has a greater influence on modelled stand productivity than unexpected mortality (D. Coates pers. comm. July 28, 2009). It is the holes in the stands that affect productivity more than anything else (K. Mitchell pers. comm. 2002). Mortality functions in growth and yield model applications are driven primarily by competition, and not biological agents. However, competition induced mortality has far less impact on actual managed stand productivity than damage agents like root disease (figure 7).

Accurately modelling forest disease impacts in spatially explicit models such as TASS is not easy, but lack doing so may result in actual timber supplies that are less than forecast. Overly optimistic growth rates due to the omission or underestimation of forest health

agent impacts ultimately results in the potential for overcutting. The general productivity of managed stands was of less concern in timber supply reviews throughout the province when mature age classes dominated our forests. The massive mountain pine beetle epidemic in BC's interior has changed that.

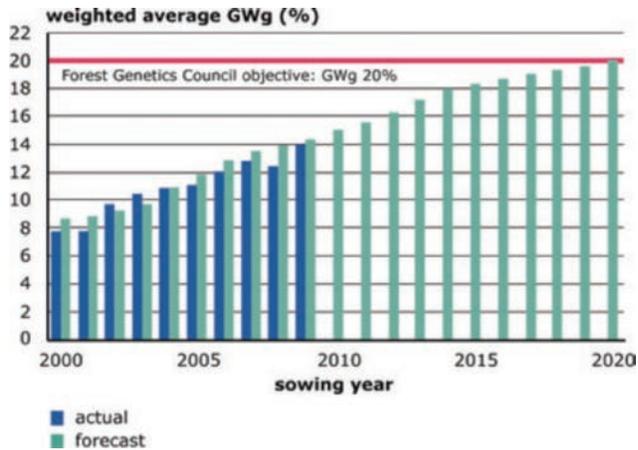


**Figure 7**—Unanticipated gaps in managed stands such as those created by *Armillaria* root disease shown here, have the largest influence on managed stand growth and yield productivity estimates.

### Unrealized Genetic Gains

Reforestation of harvested areas in BC is guided in part by the efforts of the Forest Genetics Council (FGC). The FGC's mandate is to lead a provincial tree improvement and forest genetics resource management program that encompasses the conservation, controlled use, and value-enhancement of the genetic resources of forest tree species and to advise the provincial Chief Forester on forest genetics resource management policies (<http://www.fgcouncil.bc.ca/>). Forest Genetics Council's goals and activities have resulted in over 50 percent of the seed used for reforestation in BC being derived from seed orchards with an average genetic gain of 12 percent (i.e. 12 percent more timber volume at rotation compared to wild seed (J. Snetsinger pers. comm. 2009 12 01) (figure 8).

Stoehr et al. (2004) state that there are three important benefits associated with the genetic worth of orchard produced seed in BC: 1) increased stand volume production; 2) increased timber flow over time through incorporation of GW in timber supply analyses; and 3) plantations grown using orchard seed will reach free-growing earlier. The same authors state that early achievement of free-growing has important implications in BC as adjacent timber stands cannot be harvested until free-growing has been met. It is important to consider however, that genetic gains are determined based on seedlot selections made very early in the life of a tree (figure 9), prior to many forest health agents being expressed.



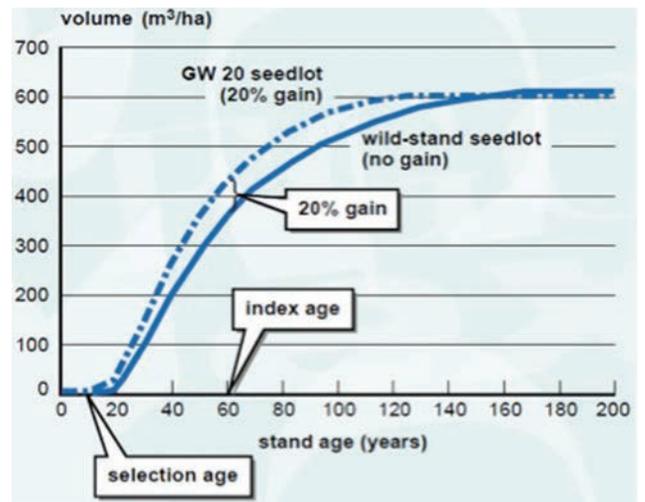
**Figure 8**—Actual and forecast gain in volume (GW) of orchard used seed (2000-2020) (Forest Genetic Councils (FGC) Strategic Plan 2009-2014).

For example, the current timber supply review for the Okanagan TSA includes estimates of genetic gain for lodgepole pine leading stands of 2.5 percent for pine in dry-belt areas and 6.9 percent in wet-belt. Lodgepole pine leading stands in the wet-belt portion of the Okanagan TSA (aka ICH) were found to have suffered some of the greatest losses in free-growing densities since declaration. In order for genetic gains to be realized the trees grown from improved seed must survive to rotation. Given the extent of losses in lodgepole pine leading stands in particular, and the majority of managed stands in general, the concept of projecting future volume gains at age 60 based on assessments made before age 10 should be reassessed.

### Certification of Sustainably Managed Forests

Certification is a voluntary, market-based instrument that gives buyers of forest products assurance that the products come from well-managed forests. Third-party verification is used to increase the credibility of certification. Meeting the requirements of some certification standards is intended to be a guarantee of sustainable forest management. British Columbia considers itself a world leader in forest certification with three-quarters of B.C.'s forest operations being certified (Barker et al. 2006).

Given the importance of forest certification in BC, it is critical that we can stand behind what we claim (P. Bradford pers. comm.). If forest managers have relied too extensively on a growth and yield model that may not adequately capture mortality and if claims of genetic gain are not fully supported by empirical data extending further into the life of a stand than age 10, can BC be a world leader in sustainable forest management?

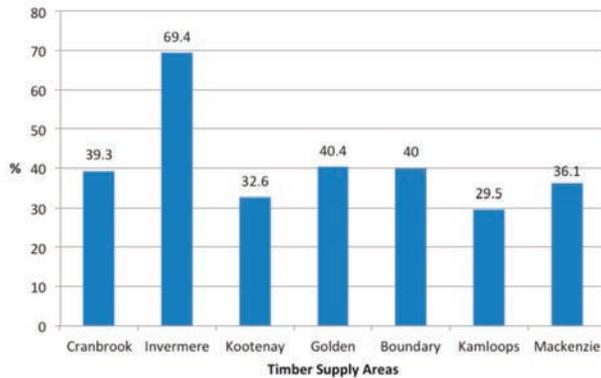


**Figure 9**—A schematic representation of stand volume production over time from an orchard seedlot with a genetic worth (GW) of 20 as compared to a natural stand seedlot (from Tanz, 2001).

### Carbon Sequestration from Managed Stands Could Be Less Than Expected

As carbon based forest management decisions begin to take precedent over the historical timber emphasis, accurate modeling of stand productivity will become all the more important (<http://www.env.gov.bc.ca/cas/mitigation/fcop.html>). The implications of this change in management focus will expand beyond the borders of TSAs, and provinces to a global stage. If BC intends to be a leader in this new forest based economy, the productivity of managed stands will have to be more closely and accurately monitored than they have been to date. Great expectations have been placed upon managed stands as they are converted from unmanaged mature conditions to managed plantations. In a sample of seven TSAs throughout the BC Interior the average yield increase expected from managed stands over that harvested from unmanaged stands was approx. 40 percent (figure 10). Such predictions serve to emphasize the points already made, that we must more closely monitor managed stands to see if indeed these expectations are being met. International agreements on reducing atmospheric CO<sub>2</sub> levels will be tied to these managed stand expectations.

Given the extent of recent losses that have occurred in managed stands due to apparent changes in forest health agent behavior under climate change (Woods et al. 2010); it may be overly optimistic to assume that managed stands could match the productivity of the stands currently being harvested. To assume that managed stands could be 40 percent more productive at age 100 given what the forests of BC have already experienced requires an unrealistic sense of optimism.



**Figure 10**—Examples of projected increases in harvest volumes at age 100, for seven TSAs in the BC Interior based on conversion from unmanaged to managed stand yield tables (C. Mulvihill).

## CONCLUSIONS

It is premature to state that managed forests in BC are not meeting the high expectations that have been placed upon them. In the examinations to date there have been examples of areas such as the Lakes TSA that are continuing to meet expectations despite high levels of forest health damage agents. There are also examples, however, where this is not the case. The balance to date suggests that free-growing densities are declining more than has been projected in growth and yield models. The primary reasons for these unforeseen stocking reductions are a combination of abiotic and biotic damage agents.

The most severe damaging agents in terms of mortality are root disease and hard pine rusts, though the overall damaging agents in terms of timber supply impacts depends on the final products expected from these stands. Timber defects may not necessarily compromise carbon sequestration for example.

Given what we have learned so far regarding the current state of managed stands, are there possible changes in forest management that could be made? Losses due to hard pine rusts could have been mitigated by adjusting species composition and increasing planting densities at the time of stand initiation. Those due to root disease could have been reduced by pro-active root disease inoculum reduction prior to planting such as stump removal. Losses due to vegetation competition could have been reduced by more pro-active brushing treatments. Abiotic and biotic caused tree deformities could possibly have been reduced by species selection but to a degree these losses must simply be accounted for in terms of lowered managed stand productivity expectations. Awareness of the fact that in a proportion of managed stands the leading inventory species labels can

change over time should help improve the accuracy of timber supply forecasts.

Quantifying the extent of changes in managed stand conditions through increased monitoring will improve the accuracy of future forest productivity estimates whether those estimates are used to forecast timber supply or carbon stocks. Such recommendations are being made by the Province's Chief Forester in the most recent Timber Supply Reviews in BC (<http://www.for.gov.bc.ca/hts/tsa/tsa24/tsr4/24ts11ra.pdf>). Sustainable forest management certification bodies may need to expand their perspectives if they are to remain relevant, particularly in light of climate change.

## ACKNOWLEDGEMENTS

I would like to acknowledge the input, support and dedication of Wendy Bergerud who even after retirement has continued to work on this overall examination of post-free-growing monitoring and the development of SDM. I would like to thank Peter Bradford and Frank Barber for developing the Forest and Range Evaluation Program that has made this work possible. I would also like to thank Anthony Britneff, Stefan Zeglen and Erin Havard and others in the BC Forest Health program for their dedication to seeing Stand Development Monitoring succeed. I would also like to express many thanks to the regional and district field staff that provided valuable insights and input into the development and piloting of the SDM protocol to be implemented in the summer of 2011.

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## Using a Needle Retention Model and a Log Market Model to Estimate the Economic Impacts of Swiss Needle Cast in Western Oregon

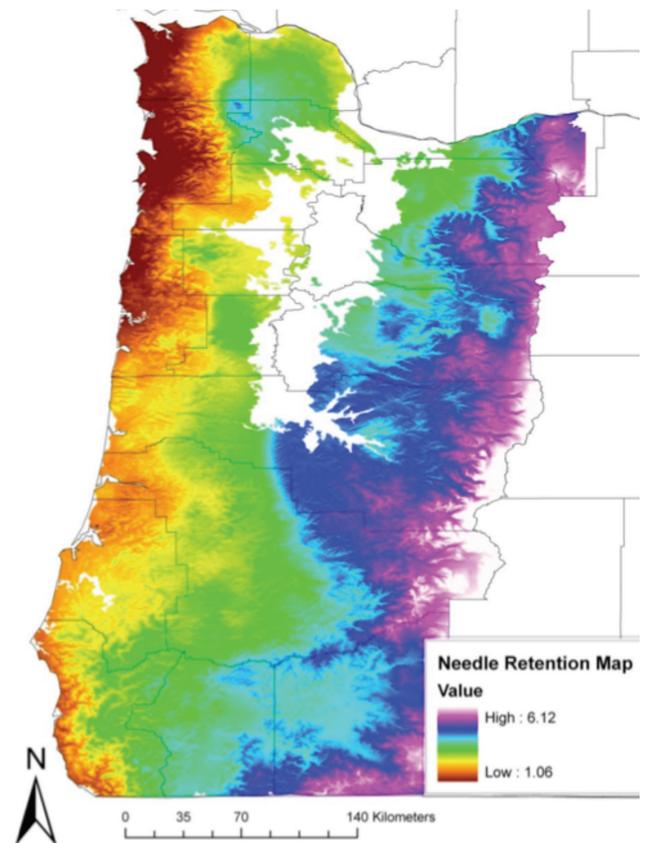
Greg Latta<sup>1</sup>, Darius Adams<sup>1</sup>, and David C. Shaw<sup>1</sup>

Swiss needle cast (SNC) is a foliage disease specific to Douglas-fir that is currently causing a major epidemic on the west coast of Oregon from approximately Coos Bay to Astoria and inland ~ 30 km or so. The 2010 cooperative US Forest Service (USFS) and Oregon Department of Forestry aerial survey identified 393,923 acres with visible symptoms, while growth impact studies have shown an average growth loss between 20 and 30 percent within this area. While mortality of Douglas-fir is rare, growth losses in some heavily impacted stands exceed 50 percent and competitive ability of Douglas-fir is reduced.

One of the key factors associated with growth loss is foliage retention. Normal foliage retention should exceed 3 years of cohorts of needles. Within the epidemic zone of SNC foliage retention can be reduced to less than one year of foliage. The relationship of disease severity, growth loss, and foliage retention has been clearly delineated by multiple studies. In addition, for the field forester, foliage retention is a readily identifiable field characteristic that does not require advanced training to identify disease. Several disease severity models have been developed that use needle retention as a surrogate for disease severity and these models predict needle retention across the landscape.

One such model developed by Greg Latta (figure 1) is being used as a foundation for analysis of the economic and market impacts associated with growth losses across the landscape caused by SNC. Growth reductions are derived from landscape level projections of needle retention (figure 1) and applied to the forest inventory using the USFS Forest Inventory and Analysis (FIA) data for the region. Forest level growth and inventory changes

are combined with models of owner harvest behavior and mill-level log demand to project market responses.



**Figure 1**—A needle retention map based on a model developed by Greg Latta for application to economic and market modeling. The modelling is a work in progress, and this map depicts one of a series of potential models. Note the very low values near the coast, especially the north coast.

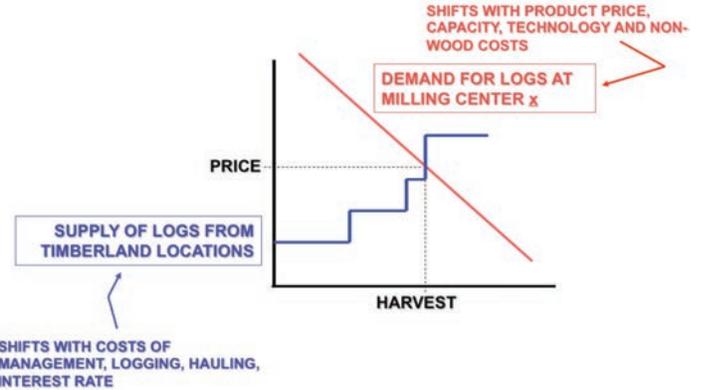
Figure 2 illustrates the basic elements of the market model. On the log supply side, timberland is differentiated by ownership, an array of resource characteristics (site, forest type, management regime, etc.) and geographic location. Owners vary harvest over time in response to prices and other economic conditions (as noted in the diagram) so as to improve their land values. Shifts in the composition of the forest inventory (volume, species, location) influence timber supply. SNC acts on these elements by

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<sup>1</sup>Department of Forest Engineering, Resources, and Management, Oregon State University, Corvallis, OR.

changing growth (of Douglas-fir) and competitive conditions in stands (hence the growth of other species as well). These changes all combine to shift timber supply.

On the demand side of the model, the milling capacity of lumber and plywood producers is identified geographically by milling center in western Oregon and differentiated by costs and production efficiency. Changes in any of these conditions, and the prices of softwood lumber and plywood in national markets, shift regional log demand. Over time, capacity adjusts across milling centers as relative profitability changes. We simulate the effects of SNC by imposing the forest growth reductions described above on the timber supply side and observing overall market adjustments.



**Figure 2**—Model of a regional log market supply and demand. The supply of logs from timberland locations will be influenced by SNC. As supply shifts market prices, harvest, product output and regional product processing capacity will adjust.





## ***Phytophthora ramorum* on Facebook: Culture, Connections, and Communities in the Fight Against Sudden Oak Death**

**Janice Alexander<sup>1</sup>**

Along with the obvious biological and ecological effects, pathologists and foresters should also take into account the social aspect of tree diseases. As examples in Canada have shown – where extensive tree mortality didn't just affect local ecosystems and communities but also international policy that was counting on these trees to be a carbon sink for a climate change agreement – social impacts can be just as important as any biological impacts of disease. The scaling of these social aspects will likely become more and more common with continued globalization, international commerce and travel, etc. There are already some examples in popular culture where people have become motivated to care about and take action around tree diseases or insects, such as: Facebook pages and Halloween costumes for Emerald Ash Borer, school plays in Australia centered on *Phytophthora cinnamomi*, and rock bands named "Sudden Oak Death."

Sudden Oak Death (SOD) is caused by the pathogen *P. ramorum* and is prevalent in the coastal forests of central California. It kills some oaks (*Quercus* spp.) as well as tanoak (*Notholithocarpus densiflorus*) and lives on many others (e.g. California bay laurel). Native California tribes value these species, especially tanoak, beyond their utilitarian – i.e., timber – values. Tanoaks are valued as a food source and spiritual connection, and California bay laurel trees are used in ceremonies. In a more abstract way, the values of our larger Western culture are often expressed through our policies and regulations. In regards to SOD in California, these values manifested themselves as fairly strict regulations against plant movement, to protect against disease spread, until important timber species were affected. Then, regulations were modified to allow for timber to move; a value judgment

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on potential risks. However, there has recently been greater interest in more strongly regulating *P. ramorum* nationally as perceived risks – social values – change.

Looking to the general conditions of what makes a community engage in action, we have three examples from the social sciences literature. Tilly (1973) says that groups who take action (1) have the economic resources necessary to get attention, (2) have members who are in close geographic proximity to each other, and (3) feel like they are lacking a voice. Michaels and others (2006) say that a focusing event is necessary before action can happen. Finally, Flint and Luloff (2007) point to a group's perception of risk and its interactional capacity. Specifically regarding SOD in California, Alexander and Lee (2010) analyzed a dozen different management actions that were taking place and summarized them into 5 case studies. Here I present two of those case studies to highlight how community attributes influenced responses to SOD.

First we have Marin County, just north of San Francisco across the Golden Gate Bridge. It can generally be characterized as urbanized and wealthy, and is generally small in scale in terms of land ownership. In the mid 1990s, local hikers first noticed dying tanoaks and brought the issue to the attention of local officials. Real alarm was raised when neighborhood and backyard oaks started dying in large numbers. There was a push to educate and inform as new information was gathered on the situation. However, at some point people wanted to know what concrete actions were going to be taken. County Supervisor Cynthia Murray took a leadership role early on and convened a widely based task force in January 2000 to coordinate the efforts of myriad local, state, federal and university authorities working to contain the disease. Next, the County mobilized other coastal counties to lobby for research and funding.

An extra benefit to Marin was that one of California's U.S. Senators, Barbara Boxer, hails from the County. She helped with the federal funding efforts which culminated in \$3.5 million from the U.S. Forest Service and more than \$2 million from California to fight the disease. In all, the response from Marin County culminated in a statewide Task Force with funding for 4 employees in 2002; a federally-supported research program in the millions of dollars (on-going); and buy-in from agencies across the spectrum.

In comparison to Marin, Sonoma County, just to the north, is typically more rural, the land holdings are larger in scale, and communities are less centrally connected. Initially Sonoma was active in SOD work: it was included in original disease area; had 1 person on staff at the local Cooperative Extension office to deal with homeowner calls and visits; and took part in State-sponsored hazard tree removal funds. However, since the disease was not found in population centers, no real leadership manifested at the County level, nor was there popular support from public on the ground. Nothing politically happened in Sonoma County in terms of SOD from about 2002-2006, but tree mortality continued and expanded greatly during the wet springs of 2005 and 2006. A community meeting on SOD in 2006 turned contentious as people crowded the event and demanded action to address the fire hazards. Energized individuals began an aggressive letter-writing campaign. In response, Sonoma County agencies convened local, state and federal officials to appeal for assistance in addressing the concerns of affected homeowners. The USDA Forest Service provided funds to the Sonoma County Department of Emergency Services and UCCE Sonoma County offices to respond. Two temporary staff positions were funded to write a strategic response plan for the County and to conduct public outreach on the issue. In addition, more funds were leveraged to address hazard trees and defensible space, and the momentum from these projects brought continued funding to the County through 2010. Ultimately, the Sonoma County Sudden Oak Death Strategic Response Plan was presented to the Sonoma County Board of Supervisors in January 2008. In response, the Board gave SOD and fire management a higher budgetary priority within the

County budget and continued to seek avenues of funding for dealing with issues pertaining to SOD.

Using these two examples and several more from other California communities, Alexander and Lee (2010) came up with three characteristics common to where successful management had occurred: (1) Connections count; (2) Scale matters; and (3) Building capacity is crucial. In the Marin case study, these lessons are apparent in that it is a wealthy, urban community close to San Francisco (many connections) that has many small lots in tight neighborhoods built right into forests (smaller scale), where the community is well-educated, activist, and used to petitioning for government support (larger capacity). This led to a quick and highly visible organization against the disease and its impacts. In contrast, see Sonoma's example, where the community is made up of dispersed rural homesteads in forested valleys (fewer connections), land holdings are large forested lots spread among agriculture and open space (larger scale), and communities are typically less reliant on centralized actions (less capacity). This led to a long delay in addressing SOD in Sonoma as compared to its neighbor Marin.

These lessons from SOD in California might be applied to future pest outbreaks. Predictive modeling of disease outbreaks is typically based on climate, host distribution, and pathogen biology, but perhaps should also include the likelihood of management action based on local community characteristics. Increasing capacity in communities now could increase collaboration and volunteer efforts to address the next pest outbreak. This may be especially important when government budgets alone do not allow for adequate management efforts.

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# Outstanding Achievement Awards



Fireweed on the Klondike Highway - Wikimedia Commons





## 2010 Outstanding Achievement Award Recipient – Paul Hennon

Ellen Michaels Goheen, Pete Angwin, and Harry H. Kope-  
WIFDWC Outstanding Achievement Award Committee 2010

The Outstanding Achievement Award is given by the members of WIFDWC to recognize outstanding achievement in the field of forest pathology in western North America. We are honored to present the 2010 Outstanding Achievement Award to Paul Hennon for sustained, significant contributions to our knowledge and understanding of forest disease dynamics and ecology.

### Paul is recognized for:

- Advancing fundamental knowledge and understanding of forest disease dynamics and ecology in the coastal rainforests of southeast Alaska, with his innovative and trend-setting research on gap dynamics and the roles of decay fungi and dwarf mistletoe in disturbance ecology.

- His elegant elucidation of the extremely complex phenomenon of yellow cedar decline resulting from careful dogged research efforts over many years.

- Moves easily between scales: from some unusual reticulated parenthosomes surrounding the dolipore of a hyphomycete with clamp connections all the way to continental climate science.

- For developing a model that involved association of two US Forest Service units, the Pacific Northwest

Research Station and Forest Health Protection, as well as his successful collaborations with scientists from many other disciplines.

- For actively creating the future of forest pathology through his work.

- And as an astute, thoughtful person with an intelligent perspective who takes his WIFDWC citizenship seriously. Paul encapsulates the best of WIFDWC by doing good science and shepherding it into forest practice.







## 2007 Outstanding Achievement Award Recipient Presentation – Rich Hunt<sup>1</sup>

### Cryptology 101: Lecture 1 – Perspectives

Because I have received this great research honour from WIFDWC, I have been asked to give you a philosophical talk. My immediate reaction is to launch into the dead rabbit experiment. However, I know that several of you would say that that zoological experiment does not belong in a botanical setting, but I have jazzed it up to make it chlorophyll friendly. Consequently, I must first give a pre-amble.

As a Berkeley trained phytopathologist it has been ingrained in me that if one has new scientific information one needs to get it into the public domain. This is because you and others can build on it, thus improving the lot of mankind. Sitting on new information unfairly impairs the lot of mankind. Consequently, when papers are submitted for review, reviewers are obligated to find the golden nuggets within the poorest of submissions and positively emphasize them and strive to help the author get the information into the public domain. The reviewer must not use generalities such as “re-write”, “poor” etc., but make specific concrete improvements. The reviewer should bear in mind Justin’s story, that when he was a lad he once made some disparaging remarks concerning Joe’s daughter for all to hear, particularly his father. Pierre was not amused and scolded him by saying have respect for the person and concentrate on attacking the person’s ideas, not on attacking the person. Reviewers who attack the author, themselves are often made to look like fools when the associate editor has to sum up the comments of all the reviewers. Fortunately for me, I give my written reviews sober second thought, so the author is not criticized; however, when the review comes out of my mouth I have been known to make transgressions.

Consequently, in Cryptology 101 to make it clear that I am attacking ideas, not the authors, I am going to give authors pseudonyms. Also in Cryptology 101, I

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<sup>1</sup>Retired plant pathologist, BC ribicola@gmail.com.

want you to observe the stirring of the pot of hasenpfeffer stew through the eyes of three crusty Cyclopi: Rick Hunt the scientist, Rich Hunt the farmer, and Richard Hunt the religious zealot. Occasionally there may be some hyperbole.

It seems from about the 1200s to 1600s learned men debated strange topics, not related to everyday needs, such as how many angels could stand on the head of a pin ([http://en.wikipedia.org/wiki/How\\_many\\_angels\\_can\\_dance\\_on\\_the\\_head\\_of\\_a\\_pin%3F](http://en.wikipedia.org/wiki/How_many_angels_can_dance_on_the_head_of_a_pin%3F)). Moreover they arrived at some fixed number, which I believe was five. At about age 7, I heard this story and was dismayed that angels could be so small. At about age 9, I decided to get a pin from my mum’s pin cushion to contemplate the subject. I was amazed at the variance in pin-head sizes and concluded that you could not possibly come up with a fixed number of standing angels without first explaining the attributes of the pin-head. Moreover, I knew that pincushions were passed from grandmothers to granddaughters and not from grandfathers to grandsons. Clearly, pins fell into the domain of women’s work, so probably the learned men of the time never observed a pin, yet debated and reached conclusions about it.

In the early 1700s the power of the king and pope were weakening and more realistic topics were debated. Causal observations and anecdotal evidence enter into the debates, but often several conflicting conclusions were reached because the debaters had different perspectives gleaned from their life experience, causal observations and anecdotal stories.

Before we review the year of 1750, let’s quickly kill the rabbit. Farmer Rich builds the restraining cage that keeps the rabbit in a vertical position. The rabbit is put into it only after Rick has determined from ear sampling that the titer is perfect. Then about midday Friday, the dirty deed is done, with subsequent harvesting of blood into beakers that are stored in the refrigerator. By late Friday, rigor has set in and the rabbit cannot be removed from the cage. It is quitting time, so they all head home for the weekend. On

Monday morning Rick arrives just in time to see Richard slide the carcass out of the cage, while he is exclaiming that something is going on here because the rabbit comes out so easily. Rick too is excited and states that he has all the equipment necessary in reviving the rabbit. He gets Richard to start CPR, while he gets his mini defibrillator, little transfusion apparatus etc. About mid-morning Rich comes in from milking the cows and shakes his head. By the end of the day, Rick is exhausted and Rich asks him what he has learnt. He has learnt that the rabbit is very, very dead, and because of his pre-med training he suspects that if this was to be repeated one would get the same result. Rich says the farmers go even further and have a theorem that states: that if you did this every Friday for the rest of your life and all the lives you spawn also did it, that on the following Monday you would have a dead rabbit.

Now in 1750 it was the norm for Europeans to eat gingerbread. This was because much of the grain was infested with stinking smut and the cheap way to make it palatable was to lace it with ginger (Large 1962). The Academy of Arts and Sciences of Bordeaux offered a prize for the best explanation for what caused the stinking grain. Mathieu Tillet was a humble man with a plot of ground on which he conducted experiments in 1751. He took all the ideas as to the cause of stinking smut and worked them into a grand split plot design with replications. He had high hopes that small black insects were the cause of the black stinky grains. In 1752 he took his results to the learned men of Bordeaux. He had a long pre-ambule saying that he was not as wise as they and he had no debatable tenets that he wished to pursue, he merely had some observations from humble experiments he wanted to share. He had various manure treatments and six different sowing days to incorporate differing weather conditions at the time of sowing, parts of these had black insects, parts with clean grain and parts with grain deliberately infested with the contents of black grains. The learned men could not inject their own views into the experiment; the data forced them all to have the same perspective.

Consequently, they all came to the same conclusion: stinking grain grew on plots where the wheat grains were deliberately infested with the black dust and clean grains grew when the initial sowing was with clean grain. None of the manure treatments, sowing dates or little black insects had any effect (Large 1962). So, all good plant pathologists know that from

this day forward that experimentation using a good experimental design trumps deductive logic.

Rick went and talked to a young pathologist friend named Mr. Shanks. Mr. Shanks is an inner city boy, and although he is well educated, particularly about numbers, he knows nothing about farming or what goes on behind closed abattoir doors. In fact, he is a vegetarian. When he heard about the dead rabbit theorem he laughed and laughed as he said you cannot conclude anything with a sample size of one. If you had treated about 40 rabbits and you found that on Monday 38 or more of them were dead, then you could say if you bled out a rabbit that after 3 days there is a strong likelihood that it would be dead. Rick asked Mr. Shanks if he knew about Tillet's presentation in 1752. Yes he did, but did Rick know that it was meaningless, because it was pseudoreplication, the study should have been repeated in southern France, and perhaps northern France as well.

As the forest pathology editor for CPS I once had a submission sent out for review. One reviewer scrawled across it "pseudoreplication". Now this paper had excellent methods, good sample sizes, statistically separated results and clear conclusions, however, the instructions to the authors clearly state that an experiment must be repeated in time and/or space. If this paper was submitted in 1975 it would easily have been published in any journal; however, in about the late 1980s we added this extra burden of proof before acceptable for publication. Does this mean that all our current research is standing on a house of cards? This repeating in time and/or space is often difficult to do with large forestry installations. It is particularly discouraging when a single large trial that was set up many years ago, and is now yielding interesting results, is not considered to meet today's standards for publication.

Also in recent years, statistical methods to analyze data have sometimes become very complex. Sometimes the statistical methods are published separately in an obscure journal by some of the same authors. Yet these papers seem to beg for simple statistical tests. I encourage reviewers to ask themselves do they really understand the statistics, and have the authors managed to get the reviewer to the same perspective as the authors, so that all can come to the same conclusion. If not, something is wrong.

Now, if you have been following what I have been saying you will know that I have been talking about the old Egyptian concept regarding the magic of threes (Harpur 2004). In conclusion, I want you to see the Three Graces from an old Greek mosaic (figure 1), because three graces grace the cover of Margaret Visser's (2008) text "The Gift of Thanks", which was my homework. I failed much of this work, but when it came to the important part I was ok. I know there are three key people to thank for spearheading me at receiving this great WIFDWC honour. Some of these people know I know who they are, but there are possibly more than three; hence, I can only truly thank the convocation as a whole. If I had received such an honour from my former employer it would not mean very much, because such honours are sometimes given for political reasons and also not given for political reasons. Yours is not one of these, your honour is truly real and I profusely thank you and Richard Hunt has even requested it be slipped into his casket at the appropriate time.

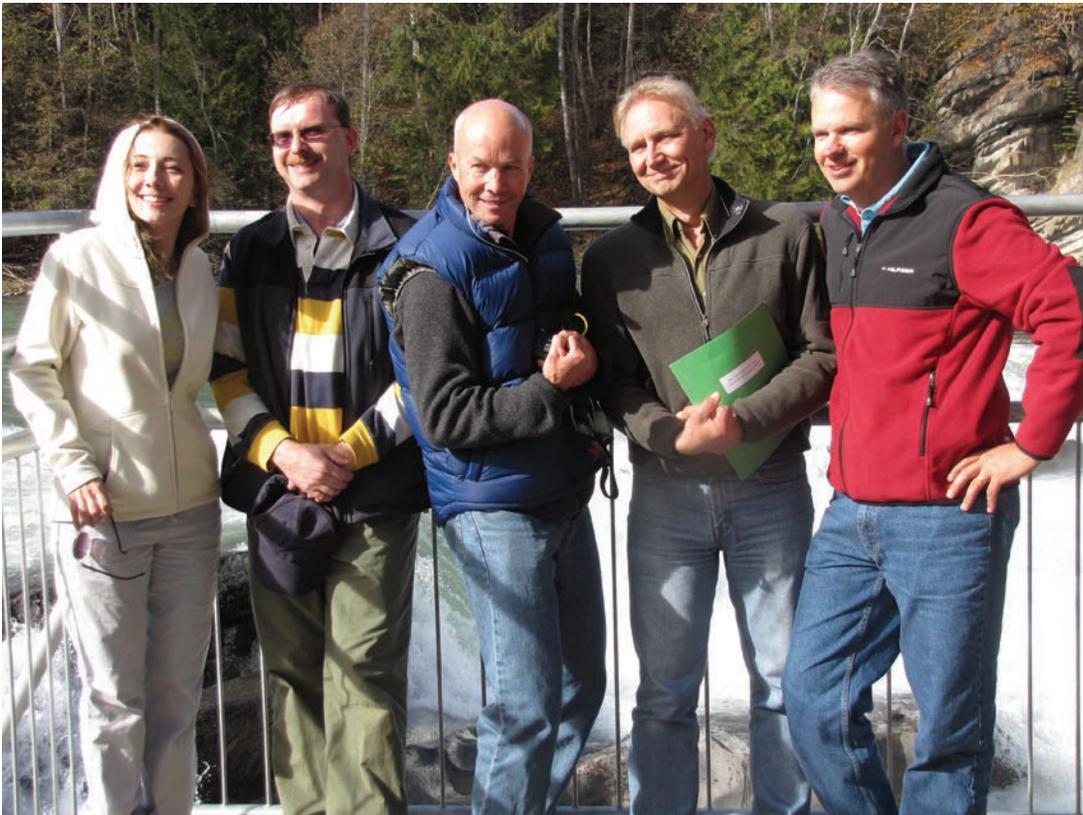
***Merci beaucoup.***



**Figure 1**—Greek Mosaic of Three Graces.

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## **2009 Outstanding Achievement Award Co-recipient Presentation- Bill Jacobi<sup>1</sup>**

### **Why I Have So Much Fun: It Is All About Family, Friends and the Excitement of Tree Pathology**

This award is not really for me but for all the people that I have been associated with in my private and professional life; my family, friends, fellow faculty and staff at Colorado State University, undergraduate students, graduate students, professional friends, and hundreds of cooperators in many agencies. Thank you for a wonderful life!

A little background on who I am: I was raised in western New York with great parents who liked camping and hiking which led to a BS degree at SUNY Environmental Science and Forestry at Syracuse, NY, a MS at West Virginia University, a PhD at North Carolina State University, a Post Doc at North Carolina State University and finally to a position as a professor of Forest and Shade Tree Pathology at Colorado State University from 1980 to present. I was lucky to have a sabbatical in Victoria, BC at the Pacific Forestry Center and to act as a student advisor at Lincoln University, New Zealand.

My academic family of Drs. Paul Manion, Bill MacDonald and Ellis Cowling have had a lifelong influence on me personally and helped shape my professional life. I started out as a Forest Biology major at Syracuse, and Paul Manion's pathology course was so exciting that I was infected with the pathology "disease" the rest of my career. I was introduced to research at Syracuse by working on *Phellinus tremulae* spore production, at West Virginia by studying Oak Wilt pathogenicity via histology, and at North Carolina State by exploring fusiform rust and pine callus interactions. The Colorado State University staff and faculty were a blast to work with over the last

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<sup>1</sup>Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins.

30 years and they have made coming to work every day worth getting up for. I may have worried about teaching and giving talks and such but I have always wanted to get up and go to work my entire career. I have had the fun and stress of teaching and advising undergraduate and graduate students for 30 plus years. The opportunity to advise students and conduct research and learn new information via graduate students has kept me glued to the University scene.

The many friends I have made at WIFDWC, WFIWC, NCERA 193, W-1187, Great Plains Tree Pest Council and APS have made up for any low points in my career. The new information, friendships, and field trips to see the country and ecosystems associated with meetings have made attending meetings a "religious" experience for me. I would like to thank all the undergraduate and graduate students and staff that worked so hard on the various projects and allowed us to learn new and exciting things about tree diseases and insects.

There are too many people to mention here, but I would like to acknowledge the graduate students and staff working on Dwarf mistletoe: Bruce Schaffer, Laura Merrill, Helen Maffei, Jennifer Klutsch, Russell Beam; Air pollution: Bill Aitken; Comandra Rust: Brian Geils, Ralph Zentz, Jane Taylor; Droopy Aspen: Valarie Scarpa; Aspen Diseases: Jeff Kepley, John Guyon, Valerie Scarpa, Susan Burks, Annette Ramaley, Meg Dudley; Root Diseases: Dan Omdal, Melanie Kallas, Holly Kearns, Sam Harrison; White pine blister rust: Holly Kearns, Anne Marie Casper, Jennifer Klutsch, Betsy Goodrich, Ronda Koski; Entomology projects: Luc Tran, Sheryl Costello, Joel Egan, Dan West; Firewood and pests: Christy Cleaver, Janet Hardin, Betsy Goodrich; Pathology research dissemination: Amanda Crump, Dust Control Salt: Betsy Goodrich. Finally, to the cooperators who have supported our students for 30 years! Thank you!



## 2009 Outstanding Achievement Award Co-recipient Presentation- Bob Edmonds<sup>1</sup>

### Notes

It was an unexpected surprise to be the co-winner of the 2009 WIFDWC Outstanding Achievement Award along with Bill Jacobi. It really is a great honor to be recognized by ones colleagues and friends. The plaque is very nice and it proudly occupies a conspicuous place in my office.

I have many people to thank for this honor, especially my wife Vickie who is here tonight. I would also like to thank Bill Heather, Professor of Forest Pathology at the Australian Forestry School in Canberra, who first got me interested in forest pathology, and Charlie Driver, who was my major professor at the University of Washington. The pathology graduate students I have had the pleasure of advising also deserve many thanks, including Will Littke, Dave Shaw, Tony Basbabe, John Browning, Marianne Elliott, Tom Hsiang, Kelly Leslie, Rosalind Hu, Roxanne Everett, Craig Cootsona, Amy Ramsey, Susan Frankel, Nick Brazee, Lydia Putnicki, Anna Leon, and my current student Nate Johnson, as well as the other 30 students who I advised in other areas of research. I only advise a few students now in contrast to the heady times in the 1980s and '90s when there were many graduate students (I think I had 11 in my lab at one time) and lots of funding. During those times we developed relationships with agency people like Ken Russell in the Washington Department of Natural Resources and Earl Nelson, Jim Hatfield, and Walt Thies in the USDA Forest Service. These were important contacts, and my relationship with Ken Russell goes back 44 years to 1966. Ken is now retired, but still gives guest lectures in my classes; talk about a long-term connection.

As one approaches the end of one's career you have an opportunity to think about the past and the track that got you to this point and ponder about the future.

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<sup>1</sup>University of Washington, bobed@u.washington.edu.

A little about my history may be of interest to you. I grew up in Sydney, Australia where I obtained a BS in Forestry from Sydney University and the Australian Forestry School in Canberra in 1964. I then worked for the Forest Research Institute in Canberra in the Seeds and Genetics section and was responsible for maintaining a number of arboreta in the mountains behind Canberra examining the performance of many northern hemisphere tree species and monitoring genetics trials of *Pinus radiata* (Radiata pine or Monterrey pine), the miracle tree widely used in plantation forestry in the southern hemisphere. It was interesting to finally visit a native stand of *Pinus radiata* near Pebble Beach, California in 2001 at the Carmel WIFWDC. I think that was the meeting. Time tends to blur over the years. Perhaps some of you can remember.

In 1965 I had a great job travelling extensively around Australia gathering Eucalyptus seeds, especially for use in reforestation India and Pakistan. Next I was employed as Teaching Assistant in the newly formed Department of Forestry at the Australian National University in Canberra and was first introduced to forest pathology by Professor Bill Heather examining nursery root diseases caused by *Pythium* and *Fusarium*. Interestingly, in cooperation with Will Littke, John Browning and Anna Leon I have gone back to working on root diseases in Douglas-fir nurseries 45 years later and my latest student Nate Johnson is working on this. Unfortunately, because of a clash with classes he could not be here.

Charlie Driver and I have advised 25 students in forest pathology at the UW and we are proud of this contribution. Many of them are very active in the Pacific Northwest and WIFDWC and that makes me feel really good.

In 1966 I decided to go to North America to go to graduate school to study forest pathology. At that time in Australia one could not get a graduate degree in natural resources in Australia so students went to the northern hemisphere. After receiving assistantships from Yale, Washington and UBC, I decided on the

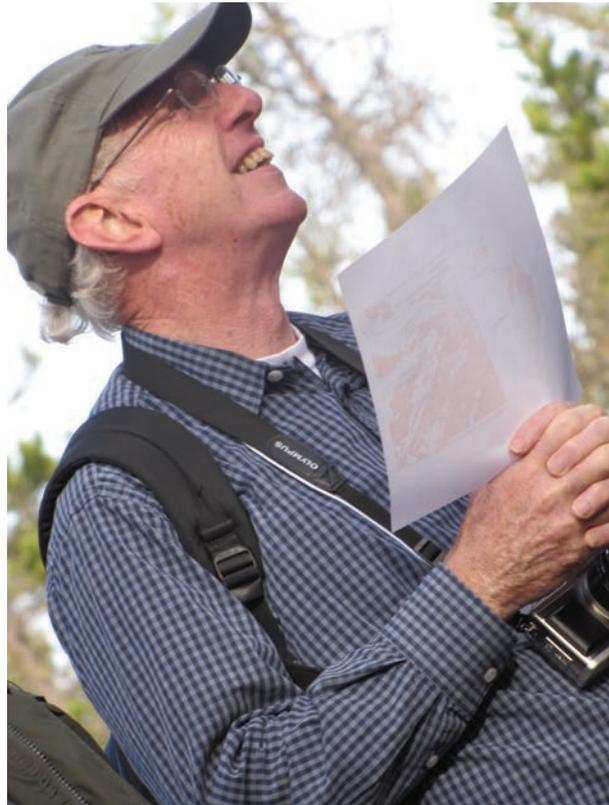
College of Forest Resources at the University of Washington to work with Charlie Driver on *Fomes annosus* as it was known then. I am probably the only person who worked on *F. annosus* for my MS and *Heterobasidion annosum* for my PhD. I had no idea what to expect when I got off the plane. However, when I arrived I unexpectedly found three students I knew from the Australia Forestry School. Australian students continued to come to Washington until the 1990s and 23 MS and PhD theses were produced. The last one was actually one of my students. Now there are several good natural resources graduate programs in Australia so we do not see any Australian grad students at Washington any more (pity!).

I married Vickie in 1969 (she is from Juneau, Alaska – about as far from Sydney as you can get). We had two children and now two grand children who live nearby. I couldn't get my kids interested in forest pathology, but I am working on my grandkids. My granddaughter (age 7) can identify Douglas-fir cones.

After completing my studies in forest pathology at the University of Washington, I took a position in 1971 in the Botany Department at the University of Michigan as the program coordinator for the US/International Biological Program (IBP) Aerobiology Program. Aerobiology is the interdisciplinary study of biological particles in the atmosphere. I organized scientific meetings, published a book on aerobiology and travelled extensively to Washington, D.C., South America and Europe fostering aerobiology research (what a great job).

I returned to the University of Washington in 1974 as a faculty member and Associate Director of the IBP Coniferous Forest Biome program directed by Stan Gessel. This program primarily involved scientists from the University of Washington and Oregon State University, but also others from most of the western states. Total funding for the program was \$10 million (a lot of money at that time) and I edited the biome synthesis volume which I calculated to be worth \$26,000 per page. I served several stints as a Department Chairman and as Associate Dean for Research from 2000 to 2009. Being associated with the Coniferous Forest Biome program gave me the perspective of looking at forest diseases from an ecosystem perspective.

Being a university professor is a great job. You can virtually work on anything you want as long as you can get funding and publish. You are definitely your own boss. The only danger is that you can spread yourself a little thin and it is hard to keep up with everything in a variety of fields, especially when your brain gets tired. In my 34 years on the faculty at the UW I developed a research program in forest pathology (especially on root diseases), and over time added new interests in soil microbiology (decomposition and nutrient cycling, soil invertebrates, and mycorrhizas), wood decay in structures, nutrient cycling, biofuels, air pollution effects, precipitation and stream chemistry, forest and riparian ecology and have even worked on salmon. It made life interesting. However, I have always maintained my roots and kept doing research in forest pathology.



One of my great loves is teaching and I have taught a wide variety of subjects including microclimatology, forest soil microbiology or soil ecology as we call it these days, forest ecology, introduction to forestry, forest pathology, forest protection, soil ecology, sustainable northwest ecosystems, and the water center seminar. In 1987 I ran a UW educational outreach tour to Australia. I wouldn't want to be a tour guide leader as a permanent job, however. For the

last 10 years I have produced the Denman Forestry Issues series that many of you may have seen on the research channel.

Teaching an integrated forest health class with Jim Agee and Bob Gara prompted us to write a Forest Health text now in its second edition. Hopefully, it is useful (one stop shopping for insects, diseases and fire). I have donated a copy for the auction.



In closing I would like to offer some thoughts about the future of WIFDWC and forest pathology. WIFDWC is a healthy organization with more than 50 years of history. There is strong attendance at meetings, which continue to have excellent programs and great field trips. There are many young people in the organization who have lots of energy to carry on the legacy. I do have a concern, however, about the training of forest pathologists in North America for the future. There are now fewer forest pathology professors and places to get degrees. If a forest pathology professor retires there is a tendency not to replace him or her. This is particularly true in these days of shrinking budgets. However, forest health issues are going to be important in the future, especially in the face of global warming. We need to your help to ensure that forest pathology continues to be taught at North American universities.

I would like to thank the WIFDWC selection committee and WIFDWC members for being nominated for the 2009 Outstanding Achievement Award. It is a great honor to accept it.



# Panel

## From the Big Picture to the Little Picture- All the Pieces of the Puzzle Are Needed



Rapids on the Fraser River above Rearguard Falls. Wikimedia Commons





## Landscape Level Armillaria Root Disease Aerial Sketch Mapping and Ground Surveys

Richard Reich<sup>1</sup>

Undeniably, the single most important risk factor to young stands in BC is Armillaria root disease, caused by *Armillaria ostoyae* (Romagn.) Herink (= *A. solidipes* Peck). However, we know relatively little about the spatial occurrence of Armillaria at the landscape level. In general, we know that it is common in the Interior Cedar Hemlock (ICH) biogeoclimatic zone (Pojar et al. 1987) based on a relatively low number of ground surveys. The limitation to getting better data has been that ground surveys are very expensive, and the reliability of aerial detection surveys has never been demonstrated. This paper describes a major breakthrough in the ability to collect low cost, yet highly reliable data. It presents the procedure used to aerially sketch map two large landscape units in BC, the results of the mapping, and the methods used to evaluate the reliability of the mapping.

The purpose of this Armillaria sketch mapping procedure is to provide a reliable operational map of the distribution of Armillaria root disease within managed stands (typically < 40 yrs old). This map is intended to provide a tool for operational foresters to use as a silviculture planning tool for making decisions regarding risk evaluation for treatments, and species selection for regeneration, and to more reliably estimate timber supply impact. However, this tool can also be used for modelling hazard and risk, climate change impacts, and carbon impacts. A detailed landscape level map provides a broader perspective of the relative importance of Armillaria and the ability to examine landscape level processes. Mapping at the northern limit of Armillaria provides a unique opportunity to gain insights regarding what, if anything, may be limiting its distribution.

The sketch mapping was conducted at the northern limit of the distribution of Armillaria root disease in BC.

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<sup>1</sup>BC Ministry of Forests and Range Northern Interior Forest Region.

The former Robson Valley Forest District (total area 1.457 M Ha) was the first district to be mapped between 1991 and 2008. It was mapped in piece meal fashion as funds became available. The former Clearwater Forest District (total area 1.455 M Ha) was mapped within a 3-week period in 2009.

### METHODS

The Armillaria sketch mapping procedure presented here was refined over a period of almost two decades within one district, in conjunction with considerable ground surveys. It was found that the key to effective and reliable mapping of Armillaria root disease is to fly very low (60-90 m) and slow (20-50 knots) using rotary wing aircraft, and to focus on second growth stands that are old enough to express the full spatial distribution of infection, but not too old that age related resistance in some species begins to mask the expression. Certain species of conifers are more likely to express infection than others; Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco), subalpine fir (*Abies lasiocarpa*, (Hook.) Nutt.), and interior spruce (*Picea glauca engelmannii* complex) stands are worth evaluating, whereas lodgepole pine (*Pinus contorta* Dougl.) is killed by so many other agents that it becomes problematic. Other factors such as moderate to heavy deciduous cover may also prevent reliable assessments and was typically evaluated on the fly.

The primary objective was to map, and delineate if possible, what appeared to be Armillaria root disease mortality centers based on stand level symptoms that matched the typical pattern of root disease. These symptoms include patches of dead and dying conifers that tend to be somewhat radially symmetrical, or more often amoeboid. These centers may occupy entire plantations or stratifiable portions thereof. Unaffected portions of these plantations typically had no observable mortality. Assessed stands were too young to contain regular mortality resulting from crown closure. Periodic ground checks were conducted to ensure that *A. ostoyae* was the causal agent, based on exposing the cambium of symptomatic and recently killed conifers. Although tree based disease incidence

was estimated for each plantation, this was of secondary importance. General landscape level patterns of occurrence were noted and attention was paid to flying elevation, since elevation (and aspect) has consistently figured as the most important predictor of occurrence.

In the late summer of 2009, all of the suitable openings (> 900 m) within the old Clearwater unit of the Kamloops FD were aerially sketch mapped. In addition, 3500 oblique digital aerial photographs were taken of representative conditions within 30 m of these young stands. These photos were GPS synchronized to GPS flight tracks to enable an independent verification using photo interpretation. Finally, over 20 silviculture openings were intensively ground surveyed using a transect method in the fall of 2009 to provide independent ground truth.

## RESULTS

### Trends and General Observations

Strong general trends were observed in both districts. The most significant trend was the inverse relationship between incidence and elevation. The highest incidence of Armillaria always occurred near valley bottoms, which range from 180-215 m. Incidence steadily declined to near zero at ~1200 m in the Robson Valley District and at ~1500 m in the Clearwater District. However, this trend is strongly influenced by both latitude and aspect. Armillaria occurred at higher elevations both as latitude decreased, and on warmer aspects. As a result, the highest elevation that Armillaria was found at in the Clearwater District was 2000 m compared to ~1500 m at the more northern Robson Valley District. A significant confounding factor for evaluating trends at the northern limit of the range is that the period of "occupation" by Armillaria within a watershed decreases with increasing latitude. Therefore, these trends must be examined within relatively small and logically delineated landscapes.

At its northern limit, the population of *A. ostoyae* appears to be moving northward in two main flanks, separated by the 2100 m Caribou Range, which is oriented on a north south axis. The eastern flank, situated in the base of the Rocky Mountain Trench, appears to extend approximately 100 km further north than the western flank, which is on the west side of the Caribou Range. The western flank may not have travelled as far north due to the more convoluted

pathway composed of complex topography forming barriers to spread, compared with the "open" north south axis of the Rocky Mountain Trench. At their northern edge, both flanks are characterized by an apparent density gradient of disease centers. Centers appear to become somewhat smaller and more distant from each other at the northern edge.

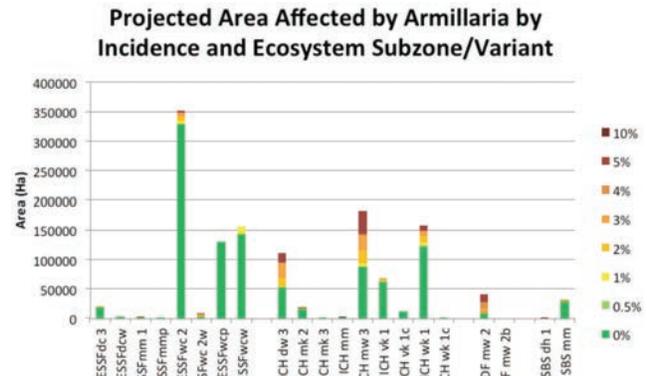


Figure 1—Projected amount of area affected by Armillaria by estimated tree based incidence and by ecosystem subzone.

The density gradient at the northern edge makes hazard rating challenging since insufficient time has elapsed for the culmination of infection and differentiation of hazard. Over time, differentiation occurs between ecosystems as a function of differences between climate, host composition, and site factors resulting in different rates of infection and spread of Armillaria. Figure 1 shows the projected percent area breakdown of area by incidence class by ecosystem subzone/variant. This figure was calculated by multiplying the percent area rated as infected (from aerial sketch mapping) by the total area within each subzone/variant. It should be noted that this breakdown should not be taken as a substitute for hazard since some of the areas are in close proximity to the edge of the distribution of Armillaria.

### Cost

Over 4000 plantations were aerially sketch mapped in total. Just over 1080 plantations were mapped within the Robson Valley District, and over 3000 in the Clearwater District. For the Clearwater District, the average cost of the helicopter time worked out to \$25 per plantation for an average plantation size of 40 Ha. This cost does not include in-house activities like the time spent preplanning the flight path on Google Earth and drawing flight lines onto the 1:50,000 scale maps (~20hrs), digitizing Armillaria polygons post flight (~35hrs), etc. For the Clearwater District, it should be

noted that the \$25/plantation cost does not include the approximately 1000-2000 additional plantations viewed aerially, but not rated for reasons such as insufficient age, too much deciduous vegetation, and/or non-target tree species.

### **Ecosystem Hazard and Risk**

Brief results covering two of the 7 larger ecosystems by area are described below. The ecosystem with the lowest percentage of *Armillaria* is the Engelmann Spruce Subalpine Fir, wet cold (ESSF wc2) biogeoclimatic subzone. It is also the largest subzone by area in the district at just over 350,000 Ha (27 percent). Approximately 93 percent of this subzone was rated as not having *Armillaria*. This was based on sketch mapping 44,950 hectares of plantations within the ESSF wc2 (13 percent of the total area within this subzone). Within this subzone, it was clear that the highest risk sites were low elevation, south facing slopes. In general, the incidence of disease was relatively low within the 7 percent of the area that was infected.

The ecosystem considered at highest risk overall is the Interior Cedar Hemlock moist warm (ICH mw3) biogeoclimatic subzone. It is the second largest subzone within the district at 14 percent of the total area (181,831 Ha). Approximately 48 percent of this subzone was rated as not having *Armillaria*. This was based on sketch mapping 11,584 hectares of plantations within the ICH mw3 (6.3 percent of the total area within this subzone). This subzone is very high risk to *Armillaria* and requires extra attention during surveys, treatment consideration, species selection, and calculation of timber supply impacts to the allowable annual cut.

### **Reliability of Sketch Mapping**

Establishing the reliability of the sketch mapping is considered one of the most crucial elements of this project. Considerable effort was invested in the design and implementation of a multiphase approach that would provide substantial rigor to the reliability assessment. To this end, three complementary, but independent methods were used to evaluate the reliability of sketch mapping. The primary method utilized was validation ground surveys. These consisted of high sampling intensity (~10 percent) transects designed to both detect low levels of *Armillaria* and help delineate distinct patches of root disease. After the initial survey was completed, disease centers were delineated by tracking along

their perimeter using GPS. Close to 100 stands were selected to capture the wide variability associated with sampling different species of conifer regeneration, incidence levels, ecosystems and site factors. The second method involved digital photo interpretation, which involved the use of large-scale colour photography of stands that were validation ground surveyed. This method was used to evaluate the reliability of the validation ground surveys themselves. The third method involved a DNA population structure study that utilized the results of the validation ground surveys. This method was used to more accurately interpret the results of the validation ground surveys, and to better understand the population dynamics of *Armillaria* at the northern limit of its range in BC.

### **Method 1 – Validation Ground Surveys**

The single most significant finding was that stands that were aerially rated as uninfected were almost always found to be uninfected based on ground surveys. These stands typically had almost no mortality visible by air, and this was confirmed on the ground. This finding demonstrated that a rating of uninfected was highly reliable. This is vitally important since fully 82 percent of the Clearwater District was rated as uninfected.

Stands rated as infected were almost always found to be infected by *Armillaria* root disease. The odd stand was found to be severely attacked by pests other than *Armillaria*, such as by black bears. In total, approximately 20/3000 stands within the Clearwater unit were noted as having significant bear damage based on aerial and ground surveys, and none within the Robson Valley unit.

Whereas the incidence of root disease estimated aerially for infected stands was correlated with that found in the ground surveys, the main goal was to demonstrate that the stratification (infected or not infected) was reliable. The observable tree based incidence is simply a snap shot in time, and only represents a small proportion of the cumulative mortality due to *Armillaria* root disease (Morrison et al. 2000).

The other main significant finding based on the validation ground surveys was that certain conifer species are far better indicators of *Armillaria* root disease than others. Not surprisingly, the most susceptible conifers in BC (Morrison et al. 1991), Douglas-fir, subalpine fir, and interior spruce, are very

reliable indicators of the presence of *Armillaria* root disease. Whereas, lodgepole pine generally speaking, is not a preferred indicator. Subalpine fir was consistently the best indicator because young subalpine fir is rarely affected by other mortality agents and retains its red foliage for several years. Subalpine fir also naturally regenerates prolifically and therefore may be relatively common. Douglas-fir, is rarely affected by other mortality agents, but doesn't retain its red foliage for more than a year. Interior spruce is affected by very few other mortality agents, but its main drawback is that it typically lacks a red foliage stage, typically losing its needles after turning chlorotic. Lodgepole pine is affected by other pests and requires ground truthing, which virtually precludes the benefit of aerial detection for this species.

### **Method 2 - Photo Interpretation**

Two types of photo interpretation were conducted in approximately 20-year-old spruce plantations containing discrete infection strata. The first type involved comparing validation ground surveys with digital stereo photo interpretation of 1:4,000 scale photos followed by ground truthing of all trees. Enlarged photos (1:200 scale) were used in the field for ground truthing all trees visible on the ground over a 14 Ha area. This provided a complete picture of the success of the photo interpretation, as well as a direct comparison with the validation ground survey. Williams and Leaphart (1978) employed a considerably different method than was used here, but of interest is their calculation of 92 percent accuracy using 1:4000 scale photography. The second use involved comparing validation ground surveys with digital stereo photo interpretation without detailed ground truth for two entire plantations (~ 40 Ha each).

With both methods it was clear that photo interpretation at this scale was very reliable and matched the results of the validation ground survey and disease center delineation very closely. This demonstrated that the ground survey and delineation process was able to accurately establish the perimeter of the disease centers in young stands.

### **Method 3 - DNA Population Structure Study**

The DNA population structure study dovetailed into the overall study through the use of the validation ground surveys. The purpose was to determine if the GPS delineated centers were unique clones of *Armillaria* root disease. If so, the survey results could be used to differentiate between apparently distinct *Armillaria* root disease mortality centers that were

really part of a single larger genet of fungus, from numerous small, but unique clones (Dettman and van der Kamp 2001a). Knowing the actual extent of individual disease centers, will in turn enable the approximate aging of clones (Ferguson et al. 2003).

Over 20 surveyed plantations were sampled for DNA by locating the GPS delineated disease centers and sampling two live infected trees at opposite sides of the center. The results were highly successful with 32 unique genotypes identified even though only 6 Random Amplified Polymorphic DNA (RAPD) markers were available at the time of the study (Brar unpublished data). Almost all of the delineated disease centers were in fact unique genotypes (clones). Larger disease stratum (often > 20 Ha) were more likely to be multiple genet of fungus that coalesced into a continuous mortality center. Small centers clearly separated by healthy forest by at least 20 to 50 meters were typically unique clones. Estimating whether a delineated mortality center is likely a unique genotype takes into account many factors, such as the relative density and spatial pattern of centers in a given area, ecosystem climate, site factors, long term species composition, etc (Dettman and van der Kamp 2001a, Dettman and van der Kamp 2001b).

Using a potentially conservative estimate of the annual rate of spread of 22 cm/year (van der Kamp 1993) it's estimated that some centers could be as old as 1600 years, whereas others maybe be only decades old. Clearly, *Armillaria* has been present for a relatively long time at this latitude, and it may be relatively active given the presence of numerous small centers found in some plantations.

## **DISCUSSION**

Demonstrating the reliability of aerial detection of *Armillaria* root disease is a complex and difficult challenge. This is primarily because ground truthing is highly specialized and very labor intensive. As a result, surveys are time consuming and expensive. This reliability study incorporated several complementary methods of testing. Validation ground surveys were conducted extensively within both districts. These surveys were supported by photo interpretation and detailed ground truthing. The DNA population structure study further supported the ground surveys and the aerial detection, by filling key knowledge gaps regarding interpretation of key spatial elements of disease stratification.

The main goal of aerial detection is to reliably and inexpensively stratify the landscape into infected and non-infected stratum. This can be a relatively simple task for age 10 – 30 year plantations of highly susceptible conifers, since the symptoms of *Armillaria* root disease infection are well expressed, and not typically confused with other causes. This method works best for Douglas-fir, subalpine fir, and interior spruce, but not at all well for lodgepole pine. Aerial detection is not recommended for older stands of any species, other than possibly Douglas-fir, since there are so many other causes of mortality, such as the bark beetles.

The three methods used to evaluate the reliability of aerial sketch mapping in plantations provided a rigorous test of the aerial mapping. They confirmed that the disease stratification of the validation ground surveys and the photo interpretation were typically delineating individual genotypes of the fungus. This provides a high degree of confidence that the sketch mapping is accurately reflecting the population structure both spatially and biologically.

Since the disease centers were mapped as close to their actual boundaries as possible, it is also possible to estimate relative age of these centers using an estimated lateral rate of spread (Ferguson et al. 2003). This may assist in modelling the spatial and temporal history of spore based infection. This model could provide an understanding of how long *Armillaria* root disease has been established within the district, and potentially be used to determine the rate *Armillaria* may colonize unaffected watersheds.

Although mature stands were not evaluated, the sketch mapped area is comprehensive enough to provide a relative risk rating for adjacent mature stands. If a mature stand of interest is located within a vast area of uninfected plantations, its risk of being infested is likely to be low. The converse also applies for mature stands located within areas of high infection.

## CONCLUSION

The detection and mapping of *Armillaria* root disease at the northern limit of its distribution in BC can be accomplished inexpensively and reliably using specialized aerial sketch mapping utilizing low, slow flights over highly susceptible conifer plantations using rotary wing aircraft. Mapping other districts at the northern limit of the range of *Armillaria* would provide comprehensive knowledge to aid silviculture planning of basic and incremental activities, and help ensure that sustainable harvest levels are set with a reliable estimate of the area affected by *Armillaria* root disease.

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## Field Studies Agree and Extend the Results of Greenhouse Trials for Host Resistance of Douglas-fir to Infection by *Armillaria ostoyae*

Mike Cruickshank<sup>1</sup> and Barry Jaquish<sup>2</sup>

In a previous 3-year greenhouse trial, potted Interior Douglas-fir (*Pseudotsuga menziesii* var. *glauca* (Biessn.) Franco) seedlings from 86 maternal half-sibling families were challenged with inoculum of *Armillaria ostoyae* (Romagn.) Herink (= *A. solidipes* Peck). The seed for the trees originated from four geographically distinct B.C. Interior Douglas-fir tree breeding zones, which represent physically and biologically different environments. Survival analysis revealed that most of the differences among families could be explained by the zone from which the family originated and to a lesser degree by the family itself; however, there was still significant variation among families within zones (Cruickshank et al. 2010). The objective of the current study was to compare rankings from the seedling study to the same families growing under field conditions, and to gain any information about how the family might achieve this.

### METHODS, RESULTS, AND DISCUSSION

Three top and two low ranked surviving families from one of the seed zones were chosen for field inoculation in a 22-year-old progeny trial in the same zone. In 2003 and 2004, fifteen trees per family were field inoculated with two 1.5 kg birch blocks that were colonized with an isolate of *A. ostoyae*. The soil was carefully excavated and two blocks were placed against each tree root collar on opposite sides. Five years after inoculum placement, the lesions caused by the inoculated blocks were measured for area and the degrees of horizontal spread (girdling) at the collar.

All lesions were callused after 5 years, and family survivability matched closely with the degrees of girdling at the root collar. Trees from less susceptible (i.e. high ranked) families had smaller lesion area and fewer degrees of horizontal lesion spread at the collar area for a given amount of inoculum (table 1).

Horizontal root collar lesion spread in low girdling families was typically elongated in the vertical direction, while in the more susceptible families lesion spread was horizontal. Since girdling at the root collar is the most important event associated with tree mortality, reduced lesion spread in the horizontal direction should result in greater probability of tree survival. The number of lesions per tree, tree size, or tree location on site had no effect on the lesion measures; however, the individual inoculum block that caused the lesion mattered. Inoculum block variation could be due to microsite conditions or the number and size of the rhizomorphs that contact the tree. The results suggest that selection of families based on seedling screening in a greenhouse should be stable under field conditions.



Comparison of growth rings between healthy and infected field trees within each family revealed that the family (422) with lowest girdling (best seedling survivor) had the most reduced growth after infection, indicating low host tolerance. The middle and lower ranked surviving families had the best and second best (421 and 423, respectively) host disease tolerance, but resulted in lower resistance. Although the study is based on a small number of families, results suggest that families with high resistance also have lower tolerance. Careful thought to the use of host tolerance/resistance for control strategies is needed. Cases could be made for the use of both host

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strategies where resistance limits pathogen damage or tolerance limits disease impact on yield and gives better competitors. Perhaps a mixture of both types of host response is needed to cover the range of possibilities needed by long-lived forest trees (Cruickshank et al. 2009).

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**Table 1**—Summary results of 22-year-old field trees inoculated with *A. ostoyae* and the survival results from the same families in greenhouse trials of 6-year-old seedlings.

Family	Year Inoculated	3-Year Seedling Survival (%)	Average Lesions Per Tree (S.D.)	Average DBH (cm) (S.D.)	# Trees Inoculate	# Trees With No Blocks Transfer	Average Degrees of Girdling at Root Collar	Average Lesion Area (cm <sup>2</sup> )
422	2003	47	2.78 (1.19)	15.8 (3.28)	15	6	17	15
620	2004	44	2.0 (1.8)	15.2 (3.46)	15	3	20	11
423	2003	35	2.4 (1.3)	15.0 (3.37)	15	0	31	69
421	2004	12	1.9 (1.1)	12.6 (1.77)	15	0	53	63
514	2004	7	1.2 (0.4)	16.8 (3.40)	15	3	53	62



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Kinbasket Lake south of Valemount BC. Wikimedia Commons





## New Fungal Associates of the Red Turpentine Beetle in China: A Phytosanitary Cautionary Tale

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### ABSTRACT

The red turpentine beetle, *Dendroctonus valens*, is a secondary pest of *Pinus* spp. in North America that was accidentally introduced into China where it has become a primary tree-killer, killing millions of Chinese red pines (*Pinus tabulaeformis*) as well as endangered Chinese lace-bark pines (*Pinus bungeana*). We initially speculated that different semiochemical responses in the founding population of *D. valens* might be responsible for its increased aggressiveness in China, but geographic variation in response to host volatiles was ruled out by a large series of tests across North American and China. We then began to examine differences in fungal associates of *D. valens* in North America and China, and found that it has acquired an almost completely new suite of fungal strains and species, including two strains of *Leptographium procerum* that appear to be much more virulent, at least to Chinese pines, than the known North American strains of *L. procerum*. Although the founding population of *D. valens* in China has been shown conclusively to originate from the Pacific Northwest, we have failed to isolate *L. procerum* from any western North American populations of *D. valens* despite intensive sampling. Source estimation studies continue in an effort to explain the origin of these more aggressive strains of *L. procerum*, and monitoring continues in Asia to document the expansion of *D. valens*' range there, where an almost unbroken corridor of naïve pine hosts extends westward across Eurasia. The existence of these two aggressive strains of *L. procerum*, as well as the large number of other ophiostomatoid species associated with *D. valens* in China, has given rise

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to concerns that exotic populations of *D. valens* might be re-introduced to their original habitat, carrying exotic fungal strains or species with which native North American pines have no co-evolutionary history.

### BACKGROUND

*Dendroctonus valens* is a secondary pest of pines in North America, and its native range in North America spans the continent (Furniss and Carolin 1977, Owen et al. 2010). It attacks almost all pines within its native range, so it could be expected to be a good invader in new pine ecosystems. It normally attacks only diseased or stressed pines in its native range, and the severity of damage attributable to it is usually associated with tree stress such as drought, injury, and disease (Eaton and Rodriguez Lara 1967; Owen et al. 2005; Owen et al. 2010). Unusual outbreaks have recently been noted in western North America, however, and it has been speculated that global warming may increase *D. valens* damage (Rappaport et al. 2001).

*D. valens* was accidentally introduced into China around 1985, apparently in mining timbers imported into the coal-mining region of Shanxi Province (Yan et al. 2005). Shanxi and neighboring provinces were the target of extensive afforestation efforts in China in the mid-1960s using primarily *Pinus tabulaeformis*, a diploxylon pine native to the region. The presence of *D. valens* went more or less unrecognized until the 1990s, when severe outbreaks focused attention on the species (Xiao, 1991); more than 6 million Chinese pines have been killed to date. An almost unbroken corridor of naïve pine hosts extends westward across Eurasia (Critchfield and Little 1966), so there is concern about expansion of the range of *D. valens* there.

In the early years of the Asian outbreak, Chinese and American entomologists met to brainstorm about possible approaches for reducing damage and developing a better understanding of the mechanisms behind the success of *D. valens* in China. Potential

explanations for its success as an invader included natural enemy escape, enhanced niche opportunity (escape from competition), climate differences, enhanced ability to locate hosts (semiochemical advantage), or differences in virulence of fungal associates. Early testing of semiochemicals in China and across the range of *D. valens* in the United States indicated that semiochemical response did not likely play a role because there were, globally, no substantial geographical differences in response (Sun et al. 2004, Erbilgin et al. 2007). Our principal finding was that across the ranges of *D. valens* in both North America and China, the key host attractant was (+)-3-carene and not (+)- $\alpha$ -pinene and (-)- $\beta$ -pinene, as had been thought previously (Hobson et al. 1993).

We therefore re-focused our attention on *D. valens*' fungal associates, both in North America and China, and began collaborations with researchers in the Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria, South Africa. The ensuing project, dubbed "The Boomerang Project" because of concerns that more virulent Asian fungal strains might be introduced into North America on *D. valens* (as a sort of Trojan Beetle), involved isolation of fungi from trapped beetles to document the new fungal associates that the insect has acquired in its newly invaded environment. Similar isolations were done in North America to expand our knowledge of native fungi. In the process, we developed a much-needed baseline phylogeography of existing beetle/fungal relationships in both the native and introduced ranges, so that we can identify new fungal introductions if they occur. In addition, several laboratory tests were conducted to elucidate beetle/fungal/host interactions in an effort to better understand the success of the *D. valens*/fungal symbiosis in China (Lu et al. 2010).

Previous to our sampling, the following were the known primary ophiostomatoid associates of *D. valens* in North America: *Leptographium terebrantis* (more virulent, dominant in western North America) (Harrington and Cobb, 1983; Owen et al., 2005), *L. procerum* (less virulent, found only in northeastern and north central populations of *D. valens*) (Wingfield, 1983, Klepzig et al. 1995, Jacobs et al. 2004, Six et al. 2003), *L. wingfieldii* (recently discovered in *D. valens* in New Jersey from contact with *Tomicus piniperda*) (Jacobs et al. 2004), *L. wagneri* (Goheen and Cobb, 1978), *Ophiostoma ips* (Owen et al. 2005, Klepzig et al. 1995), and *Grossmania clavigera* (Six et al. 2003).

Several new North American *D. valens* fungal associates were discovered in the course of this project, but are not reported here because of prior publication concerns.

### **Brief Description of Methods (see Lu, M. et al. 2010 for details)**

#### **Isolation of Fungal Associates**

Adult *D. valens* beetles were trapped in multiple funnel traps baited with (+)-3-carene, with no killing agent used in collection cups. To isolate associated fungi, living beetles were crushed using aseptic procedures and were plated onto malt extract agar. Pure cultures developed from these isolations are kept in the FABI archival collection in Pretoria. Identification to species was based on DNA sequence determinations at FABI.

#### **Pathogenicity Tests**

One-year old *Pinus tabulaeformis* seedlings were inoculated with seven isolates, two from North America and five from China, according to procedures described by Lu, M. et al. 2009. Seedling responses to inoculations were recorded as percent of seedlings remaining healthy one month after inoculation, and mean length of lesions formed in response to inoculation one month after inoculation.

#### **Testing For Mutual Suppression of Fungal Strains and Species**

Each isolate was inoculated onto MEA culture medium in Petri dish bottoms and cultured for 20 days. Then new cultures were initiated in MEA in Petri dish lids. Lids were then placed over Petri dish bottoms, in a complete factorial design such that all possible combinations of fungal strains were tested for mutual suppression. Growth of fungal colonies in the Petri dish lids was recorded every two days until the dish was fully colonized.

## **RESULTS AND DISCUSSION**

We isolated ten different species of fungi from *D. valens* trapped in China, and of those only two, *L. procerum* and *Ophiostoma ips*, had been reported from *D. valens* in North America. The newly associated Asian fungi included *Hyalorhinochlaediella pinicola*, *Leptographium pini-densiflorae*, *Leptographium truncatum*, *Ophiostoma abietinum*, *Ophiostoma floccosum*, *Ophiostoma minus* (European variety), *Ophiostoma piceae*, and *Ophiostoma rectangulosporium*-like (Lu, M. et al. 2009). Lu, Q. et

al. (2009) reported another five new associates from China: *Leptographium alethinum*, *Leptographium koreanum*, *Leptographium sino-procerum* (new species), *Pesotum aureum*, and *Pesotum pini*. Of the fifteen fungal associates acquired by *D. valens* since its introduction into China two decades earlier, then, the preponderance of species are new associations. Both reports indicate that *L. procerum* was the most frequently-isolated fungal species from *D. valens*, and both projects failed to isolate *L. terebrantis*, which is the most commonly-isolated fungal associate of *D. valens* in the Pacific Northwestern region of North America, the putative source of the introduction to China.

Of the seven fungal associates that were inoculated into Chinese pine seedlings, two Chinese strains of *L. procerum*, CMW25569 and CMW25614, were significantly more pathogenic to seedlings than any of the other strains, including an *L. procerum* strain from the eastern United States (table 1). Nearly all (95-100 percent) of seedlings inoculated with *L. procerum* and *L. terebrantis* (from the United States) and with *H. pinicola*, *L. pini-densiflorae*, and *Ophiostoma minus* (from China) were healthy one month following inoculation, whereas only 25 percent (for CMW25569) and 55 percent (for CMW25614) of seedlings inoculated with Chinese *L. procerum* strains were healthy at that point. Similarly, mean lesion length for the two Chinese *L. procerum* strains was three to five times longer than lesions produced by the other strains.

Mutual suppression assays showed that the two strains that were most pathogenic in seedling

inoculations were most adapted to suppressing other strains (table 2). These two strains, CMW 25569 and CMW25614, caused significant suppression of all the other strains, but were not themselves suppressed nor did they suppress one another. Lu et al. (2010) demonstrated that these two strains induce inoculated seedlings to produce much higher levels of 3-carene, and yet they were more tolerant of 3-carene vapors than all of the other fungal strains. There are multiple reports of toxicity of monoterpene vapors to beetle-vectored fungi, and this evidence suggests an intriguing potential mechanism of competitive advantage through selective adaptation to those vapors. Such competitive advantage might also benefit the beetle/fungal symbiosis as it colonizes the newly invaded ecosystem (Moran 2007), insofar as it results in greater attraction of the beetle vector of the fungus.

## CONCLUSIONS AND OBSERVATIONS

Taken together, this body of evidence indicates that *D. valens* has been remarkably adept at acquiring new fungal associates since its introduction into China, with only two of the 25 taxa reported from both the native and the introduced ranges of *D. valens*. The most frequently isolated strains from China were also the most pathogenic to Chinese hosts, but have not been reported to be strongly pathogenic in North American hosts (Wingfield 1986, Eckhardt et al. 2004); the exact origin of these two strains remains in question pending more intensive sampling in the northeastern United States and southeastern Canada.

Fungal Species	Source	Isolate Code	Healthy Seedlings (%)	Mean Lesion Length (cm)
<b><i>Leptographium procerum</i></b>	<b>China</b>	<b>CMW25569</b>	<b>25</b>	<b>2.32</b>
<i>Leptographium procerum</i>	US	CMW10217	95	0.57
<b><i>Leptographium procerum</i></b>	<b>China</b>	<b>CMW25614</b>	<b>15</b>	<b>2.36</b>
<i>Leptographium terebrantis</i>	US	CMW01764	95	0.76
<i>Hyalorhinocladiella pinicola</i>	China	CMW25613	100	0.46
<i>Leptographium pini-densiflorae</i>	China	CMW25600	100	0.44
<i>Ophiostoma minus</i>	China	CMW26254	100	0.41

**Table 1**—Response of seedlings to inoculations by ophiostomatoid cultures. Cultures in bold face caused significantly longer lesions than other cultures, and also resulted in significantly more dead and unhealthy seedlings at 30 days post-inoculation (redrawn with permission from Lu M. et al. 2010).

▼ / ▶	25569	10217	25614	01764	25613	25600	26254	Cont.
25569	—	—	—	—	—	—	—	—
10217	*	—	*	—	—	—	—	—
24514	—	—	—	—	—	—	—	—
01764	*	—	*	—	—	—	—	—
25613	*	—	*	—	—	—	—	—
25600	*	—	*	—	—	—	—	—
26254	*	—	*	—	—	—	—	—

**Table 2**—Analysis of mutual suppression by fungal strains. Numbers indicate CMW (Collection of Michael Wingfield) cultures assayed in complete factorial assay, including controls (“Cont.”). Arrow pointing right indicates culture in lid (responding culture); arrow pointing downward indicates culture in Petri dish bottom (suppressing culture). Asterisks indicate significant suppression of upper cultures by lower cultures; dashes indicate no significant effect (redrawn with permission from Lu, M. et al. 2010).

The founding population of *D. valens* in China has been shown conclusively to originate from the Pacific Northwest (Cognato et al. 2005), but surprisingly *Leptographium terebrantis*, the most commonly-isolated associate of *D. valens* in western North America, has not yet been isolated from any Chinese samples despite very intensive sampling (Lu, M. et al. 2009, Lu, Q. et al. 2009). We are continuing source estimation studies to resolve this conundrum. As might be expected, the two most frequently isolated strains in China were shown in laboratory tests to have traits that should provide competitive superiority (e.g. superior ability to survive toxic host metabolites combined with the ability to suppress other fungal associates).

New (or at least newly-discovered) beetle-fungal associations are becoming more prevalent, certainly partly because of increased global transport of goods but perhaps also because of our enhanced ability to detect and identify fungi phoretic on beetles (Zhou et al. 2006). Examples of the phenomenon of new and devastating beetle/fungal associations include laurel wilt (*Raffaelea lauricola*) vectored by *Xyleborus glabratus* (Fraedrich et al. 2008), thousand cankers disease (*Geosmithia* spp.) vectored by *Pityophthorus juglandis* (Seybold and Downing 2009), and possibly *Ophiostoma novo-ulmi* vectored by *Scolytus schevyrewi* (Seybold and Downing 2009). The ability of beetles that feed subcortically to inoculate fungal propagules into susceptible tissues of naïve hosts raises a whole new dimension in the field of invasion biology (Desprez-Loustau et al. 2007, Raffa et al.

2008; Klepzig et al. 2009) and represents a risk to native forests that merits greater attention from forestry researchers. Six and Wingfield (2011) encourage more intensive focus on competitive interactions among fungal symbionts, and our findings here would support that emphasis. The recent discovery of the role played by symbiotic bacteria in regulating fungal symbionts (Cardoza et al. 2008, Adams et al. 2008, 2009, and 2010) lends an additional layer of interaction to a symbiotic system that is already Byzantine in its complexity. These new associations, which will certainly play out in an environment of changing climate (Six and Bentz 2007) raise new challenges for forest land managers and underscore the urgent need for greater research emphasis in this area.

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Taxon	NA	CH	References
<i>Ceratocystis collifera</i>	X		Marmolejo and Butin, 1990
<i>Graphium</i> spp.	X		Owen et al. 2005
<i>Grossmania clavigera</i>	X		Six et al. 2003
<i>G. europhioides</i>	X		Wright and Cain, 1961
<i>G. piceaperda</i>	X		Rumbold, 1931
<i>Hyalorhinochlaediella pinicola</i>		X	Lu M. et al. 2009,
<i>Leptographium alethinum</i>		X	Lu Q. et al. 2009
<i>L. koreanum</i>		X	Lu Q. et al. 2009
<i>L. pini-densiflorae</i>		X	Lu M. et al. 2009,
<i>L. procerum</i>	X	X	Lu M. et al. 2009, Lu Q. et al. 2008, 2009, Wingfield, 1983, Klepzig et al. 1995, Jacobs et al. 2004, Six et al. 2003
<i>L. sino-procerum</i>		X	Lu Q. et al. 2008 and 2009
<i>L. terebrantis</i>	X		Harrington and Cobb, 1983; Owen et al., 2005
<i>L. truncatum</i> (sensu Jacobs et al. 2005)		X	Lu M. et al. 2009, Lu Q. et al. 2009
<i>L. wagneri</i>	X		Goheen and Cobb, 1978
<i>L. wagneri</i> var. <i>ponderosum</i>	X		Schweigkofler et al. 2005
<i>L. wingfieldii</i>	X		Jacobs et al. 2004
<i>O. abietinum</i>		X	Lu M. et al. 2009
<i>O. floccosum</i>		X	Lu M. et al. 2009
<i>O. ips</i>	X	X	Lu M. et al. 2009, Owen et al. 2005, Klepzig et al. 1995
<i>O. minus</i> , European variety		X	Lu M. et al. 2009
<i>O. piceae</i>		X	Lu M. et al. 2009
<i>O. piliferum</i>	X		Perry T.J. 1991
<i>O. rectangulosporium-like</i>		X	Lu M. et al. 2009
<i>Pesotum aureum</i>		X	Lu Q. et al. 2009
<i>P. pini</i>		X	Lu Q. et al. 2009

**Table 3**—Summary of reported fungal associations with *D. valens*; NA indicates those reported from the native range of *D. valens* in North America and CH indicates those reported from *D. valens* in China; the shaded portions show where the ranges of the nominal species overlap.

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## Rapid Threat Assessment of Aspen Health Data in Western U.S. - An Update

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Over the past decade, a noticeable loss of aspen (*Populus tremuloides* Michx.) acreage has incited ground level Forest Health Plot establishment by scientists throughout the west, including Arizona, California, Colorado, Wyoming, Utah, Nevada, Idaho and Montana. The recent onset of detailed, ground level stand condition data in aspen ecosystems both highlights the urgency to define the status of aspen health and offers unique opportunities for collaboration and information sharing among states and research groups. Colorado State University is cooperating with the Western Wildland Ecosystem Threat Center, Forest Health Management/Protection (FHM/P) from various USDA Forest Service Regions, and the Forest Health Technology Enterprise Team (FHTET) to develop the first phase of a Rapid Threat Assessment (RTA) of aspen health in the western United States. Phase one of the RTA is defined as the problem formulation and threat characterization phase. The major goal of this phase is to find and assemble all recent aspen health plot data available and determine the feasibility of combining datasets in order to have a larger scale examination of aspen health and damaging agents in the Western U.S.

Detailed ground-level aspen plot data has been compiled from FHM/P cooperators in Regions 1, 2, 3, 4 and 5 (n = 714 plots total). These data cover nine states and 33 National Forests, plus plots on tribal, Nature Conservancy and Bureau of Land Management owned land. Using the plot coordinates, we are compiling climatic and environmental data for each plot, including average precipitation and temperature variables, monthly/yearly precipitation and temperature anomalies from the 30 year support of FHTET, the PRISM Group (Oregon State University) and custom climate requests from the

Moscow Forest Sciences Laboratory. We will utilize the west-wide dataset to quantify the geographic distribution of biotic damages on aspen in the West and utilize the supplementary abiotic data to begin to test hypotheses from regional studies to determine if regional theories on aspen health scale up to a larger geographic areas.

Data from Regions 1, 4 and 5 have been successfully merged together at the plot level and aspen stand, overstory, sapling and seedling data are compatible. These three regions based aspen health survey methodology from 2006 surveys (R4 – J. Guyon) and utilized similar plot designs and methods. The strength of relationships between biotic damage agent incidence and aspen dieback varies by regions, and some important biotic damage agents occur less frequently in one region compared to others. We are continuing to define relationships between aspen stem, sapling and regeneration characteristics (stems/acre, health status, dieback, etc.) with climate data using models that account for biotic damage agent presence. The successful merging of datasets from three regions highlights the benefits of developing and following similar study protocols.

*Project Cooperators include Eric. L. Smith, Frank J. Krist, John Guyon, James Hoffman, Holly S.J. Kearns, Brytten E. Steed, Danny Cluck, MaryLou Fairweather, Brian W. Geils, James Worrall, James T. Blodgett and Charles G. Shaw.*



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## Restoration Planting Options for Limber Pines in Colorado and Wyoming

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Limber Pine (*Pinus flexilis*) populations in the southern Rocky Mountains are severely threatened by the combined impacts of mountain pine beetles and white pine blister rust. Limber pine's critical role in these high elevation ecosystems heightens the importance of mitigating these impacts. To develop forest-scale planting methods, six limber pine seedling planting trial sites were installed extending from the Medicine Bow National Forest in southern Wyoming to the Great Sand Dunes National Park and Preserve in southern Colorado. Seedlings came from the Colorado State Forest Service nursery and were 3 years old, container-grown, and originated from a Colorado seed source. A total of 2,160 seedlings were planted. Six plots within the sites were split between high and low density canopy conditions, with three in each canopy class.

In the canopy blocks, treatments were: 1) presence/absence of a nurse object and 2) presence/absence of Terra-Sorb Hydrogel. 1) We created nurse objects by burying 50 cm tall tree stem segments (20-40 cm dia) 10 cm in the ground. Trees were planted as close as possible to the object at the four cardinal directions to further test exposure stress. In the control treatment we planted seedlings in an east/west orientation 40 cm apart. 2) Terra-Sorb Hydrogel (Pittsburgh, PA) is a potassium-based co-polymer gel that absorbs up to 200 times its weight in

water. Hydrogels are commonly used in horticulture although scientific literature shows mixed results for tree survival. The hydrogel treatment consisted of dipping seedling roots in the hydrogel slurry before planting, per manufacturer directions. The roots of control seedlings were dipped in water before planting. The hydrogel treatment was omitted at two sites due to logistics. There were six replicates of each treatment combination, with 432 seedlings planted in each of the four sites with the Terra-Sorb Hydrogel treatment and 216 seedlings planted in each of the two sites without hydrogel treatments, for a total of 2,160 seedlings. We will continue monitoring health and survival of the outplanted seedlings in the future. Results from this project will be used to develop limber pine planting protocols for the southern Rocky Mountains.

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## Aspen Damage in the Colorado Rocky Mountains: Preliminary Results

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In recent years, stands of dying aspen (*Populus tremuloides*) were observed throughout Colorado. Worrall, et al. (2008), found rapid death of overstory of aspen in southwestern Colorado, and coined the phenomenon as Sudden Aspen Decline (SAD). Our objectives were three-fold: (1) conduct field surveys in damaged and healthy aspen stands on US Forest Service lands in Colorado not covered by Worrall's survey and based on the Forest Service's aerial survey data; (2) determine the overall health of aspen stands on U.S. Forest Service lands in Colorado through the establishment of extensive roadside survey plots; and (3) produce a state-wide hazard map for aspen damage using plot and spatial data. In 2006, the Forest Service began to map apparently declining aspen stands. Affected stands were mapped and categorized based on stand mortality and crown thickness. We placed approximately half of our plots in 'damaged' stands, and half in unaffected stands to sample these two aspen populations, though not as a paired-plot design. Five study areas were used to assess aspen stands for damage. Three study areas (including the Pike, San Isabel, and Medicine Bow National Forests) were east of the Front Range-Continental Divide and two were on the west side of the Divide (including the White River and Routt National Forests). The five study areas represented the drier front-range aspen types and the wetter western slope areas. In all, we established 97 plots (with three circular, fixed-area subplots positioned along a 100-meter transect) and over 600 extensive road side stand assessment plots from southern Colorado to southern Wyoming. Preliminary findings suggest: (1) *Cytospora* fungus is much more common among 'damaged' stands; (2) aspen bark beetles (*Procryphalus mucronatus* and *Trypophloeus populii*) were present in higher numbers on living trees in 'damaged' stands. Project summary is expected by mid 2011.

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## Distribution, Species, and Ecology of *Armillaria* in Wyoming

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*Armillaria* root disease is caused by a complex of *Armillaria* species associated with many conifer and hardwood hosts worldwide. *Armillaria* species differ in mating types, morphology, culture growth form, molecular sequence, chemical composition, aggressiveness, host range, and ecology. This root disease can play a role in tree mortality often attributed solely to insects or a different disease. In this study, a field survey was conducted to determine the geographic distribution of *Armillaria* spp. in various forest types throughout Wyoming, and to characterize their relationships in hosts and aggressiveness, soils, site conditions, and climate.

### METHODS

Previous to field visits, plot locations were systematically selected using a geographic information system. One plot was located in each cell of a three-by-three-mile grid across the state in accessible forested areas of public ownership (federal, state, and tribal lands). Stand selection was stratified by forest cover type. Variables recorded included: altitude; slope; aspect; forest cover type; organic matter thickness; frequency of rhizomorphs in the soil; number of stumps, snags, and logs; tree species and diameter at breast height for all live trees; and observations of other diseases, insects, and damage. Soil samples were analyzed for organic matter content, pH, and texture. For recent dead host-trees, root disease was confirmed if the root collar or a major root had resin and mycelial fans. For live hosts, root disease was confirmed if mycelial fans were observed in the root collar or a major root. Variables recorded for infected host trees include: species, DBH, host condition (living or dead), crown position, percentage live crown, and associated stress/mortality agents. *Armillaria* spp. were isolated from affected trees and identified in the laboratory by crossing them with haploid tester isolates of known *Armillaria* spp.

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### RESULTS AND CONCLUSIONS

Three hundred plots were assessed in 12 different forest cover types. *Armillaria* was found at 89 (= 30 percent) locations. A total of 133 isolates were collected. Four *Armillaria* species were identified. *A. solidipes* (= *A. ostoyae*) was the most common (40 plots; 63 isolates) followed by *A. sinapina* (32 plots; 43 isolates). *A. gallica* (11 plots; 15 isolates) and *A. cepistipes* (8 plots; 11 isolates) were rarely collected.

Results suggest that *A. solidipes* is the most common *Armillaria* species in Wyoming. This species was frequently associated with conifer hosts and cover types, which are far more common than hardwoods in Wyoming, and was found mainly in the south and northeast parts of the state. *A. sinapina* was associated with conifer and hardwood hosts and cover types, and was widely distributed, but not in the central area of the state. *A. gallica* was found mostly in oak and other hardwood hosts and cover types, and only in northeast Wyoming. *A. cepistipes* was found mostly in hardwoods, but also in conifers in hardwood cover types, and only in west-central Wyoming.

For all species combined, root disease was identified from 66 percent of the collections. *Armillaria* species differed in hosts, forest cover types, soils, and other site conditions. Differences in aggressiveness among *Armillaria* species were not evident with the combined data, but clear differences become apparent at the host level. Results here suggest that *A. solidipes* is the most aggressive species in conifers. This is in contrast to the often encountered statement that *A. solidipes* is the most aggressive species. Other *Armillaria* species were more aggressive in aspen and other hardwood species. *A. gallica* was the most aggressive species in oak.

*Armillaria* root disease is considered to be a major driver of stand structure and composition in many forest ecosystems. Determining the species of *Armillaria* in a stand by host can contribute significantly to understanding its disease dynamics.

# Panel

## WIFDWC Centennial Panel on White Pine Blister Rust



Yellowhead Lake, Mount Robson Provincial Park BC (Frank Kovalchek) Wikimedia Commons





## Blister Rust in North America: What We Have Not Learned in the Past 100 Years

Eugene P. Van Arsdel<sup>1</sup> and Edited by Brian W. Geils<sup>2</sup>

### ABSTRACT

Introduction of *Cronartium ribicola* (white pine blister rust) greatly motivated development of tree disease control and research in America. Although foresters and pathologists have learned much in the past 100 years, more remains to learn. The most important lesson is that fear of blister rust has reduced pine regeneration more than the disease itself. Based on six decades of study, I share what I've learned on five topics of personal interest—the evolution of pines and rust, history of blister rust, effectiveness of eradication, influences of climate and *Ribes*, and importance of sustaining research.

The rust fungi first arose on primitive plants and later evolved to alternate between angiosperms and gymnosperms. Early stems rusts were widely distributed on *Pityostrobus* before the modern pine subgenera of *Strobus* and *Pinus* emerged during the Triassic and Jurassic Periods. In the Cretaceous Period, blister rust fungi of the genus *Cronartium* became widespread on Laurasian pines. During the warm Paleocene and Eocene Epochs, pines retreated to cold refugia as angiosperm forests expanded. In North America, pine refugia were too cold for infection of *Ribes*; so, unchallenged by the rust, surviving pines lost their resistance. In Eurasia, a variety of environments allowed hosts and pathogens to coevolve. Pleistocene glaciations removed white pines from most of Europe; the pathogen and resistant white pines survived in Asia.

*Pinus strobus* was introduced from North America into Europe in 1553 but not widely planted until the 1700s. Before that, white pine blister rust was restricted to Asian white pines associated with *Ribes nigrum*.

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When European foresters and gardeners brought together highly susceptible *P. strobus* and *R. nigrum*, they set off a super-epidemic. Between 1907 and 1909, millions of white pines from European nurseries were imported to eastern North America. Infected seedlings went undetected, and another epidemic took off. Quarantine, inspection, and eradication of infected white pine failed. Control shifted to eradicating *Ribes*, especially *R. nigrum*.

Mortality early in the North American epidemic reduced white pine stocks by alarming rates. In the Northeast, blister rust was successfully controlled primarily by eradicating *R. nigrum* in well supported state programs. The long-term effectiveness of *Ribes* eradication in Maine was tested by comparing the percent incidence of infected trees in areas never treated with areas treated repeatedly over 70 years. Eradication produced a reduction in blister rust from 9.1 percent incidence without treatment to 3.8 percent with treatment. Since 91 percent of unprotected trees were not infected, did the amount of rust after the first wave and elimination of *R. nigrum* justify a continued program?

Differences in rust distribution across the Lake States suggested that climatic factors were important. Data for temperature and moisture requirements of rust development enabled me to draw regional hazard zones. In the lowest zone, infection was unlikely except in locally cool-moist sites. In the highest zone, locally cold climates in forest openings prevented *Ribes* infection but pine infection could occur in sites even with no *Ribes* because of long-distance dispersal.

On the cold, dry Yellowstone Plateau, *Ribes* were seldom infected; the few pine infections which did occur resulted from *Ribes* at lower elevations. A variety of conditions were found in the Sacramento Mountains of southern New Mexico. There are high-elevation sites too cold for *Ribes* infection, warm-dry low-elevation sites where susceptible hosts are sparse and blister rust is rare, and mid-elevation sites “just-right” for lots of rust.

Control and research on tree diseases requires an understanding of local conditions and a crucial, long-term commitment. Eastern ideas on Ribes control, spore dispersal, and climate factors had to be adjusted for the West. Unjustified embracement of antibiotics, followed by disappointment, lead to abandonment of the therapy strategy. Loss of white pine's commercial value disrupted research still needed to know where to grow white pine for wildlife and diversity.

## INTRODUCTION

Foresters, pathologists, and administrators have learned a great deal in hundred years of infestation by white pine blister rust in North America. Introduction of *Cronartium ribicola* J.C. Fisch. in Rabh., the pathogen of white pine blister rust, was a great motivator for development of tree disease control and research. But, there remain many things we have not yet learned or have repeatedly failed to apply. The most important lesson is that unjustified fear of blister rust has reduced pine regeneration more than damage from disease itself. White pines given a chance can produce enough seedlings to overcome destruction by blister rust. Blister rust is wiping out white pines on some sites, but there are also places where white pines are thriving. We need to learn from a better understanding of the rust where we can grow white pines.

A principal interest of my professional and volunteer work over six decades has been how climate and Ribes affect rust dispersal and infection. Other topics of personal and special interest have been the evolution of pines and rust, history of blister rust, effectiveness of Ribes eradication, and the importance of a commitment to research and white pine silviculture.

### Origins of the Rust

The first rust fungi probably arose on mosses, ferns, and other primitive vascular plants during the warm-moist carboniferous periods of the Paleozoic Era (Millar and Kinloch 1991). The early rusts were autoecious fungi with simple spores. The modern pine subgenera *Strobos* (white pines) and *Pinus* (yellow pines) diverged from *Pityostrobus* in the Triassic and Jurassic Periods (early Mesozoic) and diversified during the Cretaceous Period. Early *Cronartium* rusts evolved with pines during the middle Mesozoic Era after Laurasia and Gondwana separated from

Pangaea. *Cronartium ribicola* adopted a heteroecious lifecycle of alternating between *Strobos* pines as aecial hosts and first Ribes and later Pedicularis and Castilleja as telial hosts.

In the early Cretaceous, pines and Ribes were distributed across Laurasia. Blister rust spread in waves every few years on a species of Ribes ancestral to *R. nigrum* and *R. hudsonianum*. *Cronartium ribicola* became a Ribes rust that overwintered on pines tolerant or partially resistant to infection by frequent exposure. As Laurasia separated into North America and Eurasia, different populations of pines, Ribes, other telial hosts, and rusts coevolved on each continent

During the Eocene and Paleocene Epochs of the Tertiary Period, the earth was intermittently hot or cool (mostly hot) for 27 million years. Angiosperm boreotropical plants flourished up to 70° north; pines retreated to cool refugia at high elevations or latitudes (Basinger et al. 1994; Baez 2006). In North America, the white pines *P. monticola* and *P. lambertiana* crowded into the 5500 m Rocky Mountains; *P. ayacahuite*, *P. strobiformis*, and *P. flexilis* into the 5500 m Sierra Madre Oriental; and *P. strobus* along the Arctic shore (Millar 1993). Because these areas were too cold for *C. ribicola* to infect Ribes, surviving white pines were not exposed to infection and lost their resistance. In East Asia, a major refuge was along the Tethys Sea. Mountain ranges formed diverse environments as India pushed under Asia. Because both *R. nigrum* and *C. ribicola* persisted, selection for resistance remained in the Asian white pines (*P. koraiensis*, *P. pumila*, *P. sibirica*, and *P. wallichiana*).

The Pleistocene Epoch encompassed a series of glacial and interglacial periods. *Cronartium ribicola* resided on *P. pumila*, *P. sibirica*, and *R. nigrum* and tracked across Eurasia during the ice ages or persisted in unglaciated Siberia. Because of continuous association, Eurasian white pines retained rust resistance. In the Eem Interglacial, a pine forest extended across Eurasia and included pine infected by *Cronartium* (Flint 1971; Mirov 1967). This forest was largely wiped out during the Weichsell Glaciation when most of Europe was tundra. At the end of the Pleistocene, the only pines in Europe were *P. sylvestris* (a yellow pine, non-host for *C. ribicola*) and white pine species restricted to high-elevation sites—*P. cembra* in the Alps and Carpathian Mountains and *P. peuce* in the mountains of the Balkan Peninsula (Holzer 1972).

## Introduction of the Rust

*Pinus strobus* was introduced into Europe in 1553 but not widely planted in gardens until 1705 or forest plantations until 1750 (Spaulding 1929; Moir 1924). Although resistant *P. wallichiana* from Asia were planted, the only white pine throughout most of Europe was the susceptible *P. strobus* from North America (Holzer 1972). Long before 1750, *R. nigrum* was widespread across northern and mountainous Eurasia as a wild native (Spaulding 1929; Kakishima et al. 1995) and nearly ubiquitous in gardens as the European black current (Spaulding 1929). Tubeuf (1917) and Spaulding (1929) believed the *C. ribicola* which caused the European super-epidemic had originated in northern Asia. That rust first became widespread in Russia after *P. strobus* was introduced to gardens there and then spread to Europe in several waves on *R. nigrum*. Blister rust was discovered on Ribes in 1830 in Austria, 1846 in Crimea and 1854 in Estonia (Unger 1836, Spaulding 1929; Peterson 1973). The close association of susceptible *P. strobus* and *R. nigrum* in European gardens and nurseries allowed development of an epidemic Tubeuf (1927) suggested was more widespread across Europe than shown by available data.

White pine reforestation accelerated in the eastern States from 1900 to 1910. Initially, American nursery stock of *P. strobus* was too expensive and European stock was burdened with a high tariff. After the tariff was removed, millions of small trees—many infected with *C. ribicola*—were brought from Europe (principally Germany). From 1907 to 1909, these trees were established in plantations widely scattered across the northeastern States, Lake States and eastern Canada (Boyce 1961). Early introductions were at Kittery Point, Maine in 1897 on *R. nigrum* from Nottingham, England (Posey and Ford 1924) and Geneva, New York about 1900 on *P. strobus* (Stewart 1906). Separate and serial introductions in New England, Ontario, and Quebec resulted in widespread establishment of the rust on both white pines and Ribes (Detwiler 1918a, 1918b, 1920). Spaulding (1922) mapped changes in rust distribution 1909 to 1919.

Quarantine, inspection, and eradication of infected white pines proved ineffective. Tubeuf (1897) warned infected nursery stock was distributed throughout Germany. Although the J. Heinz nursery maintained that its stock was disease-free, Tubeuf (as cited in

Spaulding 1929) responded that disease absence could not be assured because symptoms were invisible for years after infection. Spaulding (1909) inspected trees from the J. Heinz Nursery and planted in New York. He confirmed symptoms could escape detection for the first three years and concluded nursery inspection could not detect every infected tree (Spaulding 1913). Infected Ribes and pine at the Geneva, New York introduction site were destroyed in 1906, but two 15-year-old pines sporulating in 1913 were found too late to prevent permanent establishment (Spaulding 1914).

Blister rust intensified and spread from northern and eastern nurseries (including Ontario) but did not spread from southern and western nurseries (Indiana, Illinois, and southern Pennsylvania). This difference was first attributed to eradication but later recognized as a result of a warm-dry climate unsuited for rust dissemination. I reported (Van Arsdel 1954) that rust did not spread where the mean July temperature was > 21°C. In addition to a warm climate, spread was barred by distance from wild Ribes (e.g., Gibson County, Indiana, 120 km, Van Arsdel 1949). Although infected trees shipped in 1908 from the J. Heinz nursery to an Illinois nursery failed to spread the rust in Illinois, re-movement of trees to Wisconsin, Minnesota, and western Ontario established infestations (Haddow 1969; Kroeber 1948; Pickler and Pierce 1919; Sampson 1918). Early efforts were made to control blister rust by destroying symptomatic pines and removing all hosts within an immediate introduction area, but the rust kept spreading. By 1919, the control strategy was changed to eradicating all Ribes within an infecting distance of pine stands (Kroeber 1948).

Mielke (1943) asserted the introduction of *C. ribicola* into western North America was the result of importation in 1910 of infected *P. strobus* from Ussy, France to Point Grey, Vancouver, British Columbia. Hunt (2003) and Geils et al. (2010) questioned whether this was the first and only western introduction. Other importations of *P. strobus* at the time into Cascade nurseries were from nurseries in Illinois and Ohio that were too warm for rust survival, so these imported trees could not have been infected. The early infestation on Mt. Hood, Oregon can be attributed to long-distance dispersal of aeciospores from infested sites to the northwest.

## Early Loss and Control

Soon after establishment in the East, there was a great deal of blister rust on both *Ribes* and pines; and it was spreading rapidly (Detwiler 1918b). Reports documented destruction of 90 percent of the best trees (Snell 1931) and loss of nearly 50 percent of the crop volume (Rusden 1952). Such losses lead to near panic. *Ribes nigrum* was banned and eradicated. Patriotic appeals were made, "The war against blister rust was just as important as the war against the Germans" (Detwiler 1918a).

In New England, early control was successful because *R. nigrum* was practically the only inoculum source; red currants (*R. rubrum*) and wild *Ribes* were not important (Snell 1941). Ostrofsky et al. (1988) evaluated the effectiveness of *Ribes* eradication in Maine by comparing disease incidence in areas never treated with areas where *Ribes* were eradicated for 70 years. On treated areas, 3.8 percent of trees were infected; on areas never treated, 9.1 percent were infected. These results showed an average, statewide reduction in rust coincident with eradication. But, did the small incidence in untreated areas—91 percent were not infected—justify eradication?

Success of the eradication program in the Northeast whereby *R. nigrum* was eliminated in the proximity of most pine stands means that blister rust is no longer a major disease there. Now, claims are made that blister rust never was a problem and *R. nigrum* can be grown again. Blister rust was a problem and would be again if *R. nigrum* were re-introduced. Two questions not resolved from work in the Northeast are: 1) the role of wild *Ribes* (including their susceptibility and distance to pine) and 2) how climate affects where and when blister rust would be a threat.

Blister rust control laws and programs were also established in the Lake States of Wisconsin, Minnesota, and Michigan. Under direction of Dr. E. E. Honey, a series of plots were established to determine the effects of *Ribes* eradication on pine infection, relative susceptibility of *Ribes* species, distance of rust spread, and *Ribes* reproduction. Information on these investigations was contained in unpublished reports by Honey and others from 1933 to 1947. I reviewed this work in Van Arsdale (1972) and briefly summarize various observations below.

On the Lake States plots, rust infection before eradication in 29 stands ranged from 0 to 118 cankers per 100 trees per year. Since *Ribes* were abundant in all these stands, this variation is attributed to

environmental differences. After eradication, infection on all plots was reduced to 5 to 20 cankers per 100 trees per year. Had *Ribes* not been removed, only several plots would have been severely damaged and most plots only slightly damaged (Van Arsdale 1968). The problem was to know beforehand which would benefit from treatment.

One environmental difference among the study plots was the mixture and behavior of *Ribes* species. *Ribes hirtellum* was susceptible enough to build an epidemic by itself. Although *R. cynosbati* was very susceptible, it defoliated before rust spread to pines; *R. americanum* was slightly susceptible. Where these latter two species were alone, few pines were infected. Where both species were present, infection went from pine to *R. cynosbati* to *R. americanum* to pine. Even with both *Ribes* present, 89 percent of trees on one plot remained infection-free. On another plot where *Ribes* were not removed, abundance, susceptibility, and per-leaf inoculum potential differed independently among the five species present and 70 percent of trees remained not infected.

Boyce et al. (1934) said that without control, *P. strobus* could not be perpetuated except where *Ribes* were few. Certainly, the loss of seedlings and damage to plantations had been great. But infection rates appeared to decline after the first waves—perhaps because there was less inoculum after blister rust defoliated the most susceptible *Ribes*. Where there is abundant natural reproduction, the regenerative power of white pines can mitigate the damage caused by the rust.

## Rust Hazard in the Lake States

My University of Wisconsin and Forest Service Research showed that frequent blister rust infection on pine required a favorable climate at either a regional level or in a locally cooled microclimate. Where the general climate was too warm for pine infection, the rust was limited to sites cooler and wetter than average. These included sites cooled by nocturnal, down-slope winds such as at the base of a slope or in a narrow valley and small forest openings cooled by net radiation heat loss. Using this information (and a wide pen), I mapped four rust hazard zones in the Lake States (Van Arsdale 1961, 1972; Van Arsdale et al. 2006). These are climatic hazard zones. Hazard at any particular site is further modified by the distribution and susceptibility of the *Ribes* species present and the landscape pattern of nocturnal winds transporting spores.

### **Climatic Hazard Zones**

Zone 0 is the southern 80 percent of the Midwestern states of Iowa, Illinois, Indiana, and Ohio. With a mean July temperature > 23°C, the area is so warm that rust infection or persistence on white pine is unlikely.

Zone 1 covers the northern 20 percent of the Midwestern states and lower southern portions of the Lake States. The general climate is too warm and dry for blister rust spread except in favorable sites where a combination of factors create a locally cool (and therefore mesic) microclimate. A favorable site would be where nocturnal air drains into a constantly shaded forest opening.

Zone 2 includes elevated regions across the lower half of the Lake States. Only a single microclimatic modification is required to cool a generally warm climate sufficiently for rust infection. A favorable site would be where a row of high trees shades an opening from morning sun long enough to prolong the dew period sufficiently to allow infection.

Zone 3 extends from northwestern Minnesota into a narrow band across central Wisconsin and Michigan. The general climate is cool and moist enough without local modification for pine infection to occur every few years. Cankers are scattered among many pines but are usually restricted to that portion of the crown within 2 m of the ground where dew and fog linger.

Zone 4 stretches across northeastern Minnesota, northern Wisconsin and Michigan. The general climate is so cool and wet that infection is possible on all sites and can extend to the tops of trees. The widespread distribution of infection and presence of cankers high in the crown indicate long distance spread from infected *Ribes*.

### **A Case History in a High Hazard Zone**

The experience of Tom Nicholls demonstrates that the outlook expressed by Boyce et al. (1934) was overly pessimistic. Blister rust has not been as serious in the Lake States as was initially feared. Tom manages a tree farm in Fifield, Wisconsin (Price County), a glaciated region now covered in a patchy mosaic of vegetation types. Logging the virgin white pine peaked in 1892 and ended in 1920 when the site was converted to dairy pasture. After dairy farming ended in the 1950s, a few remaining mature white pine seeded the pasture to a thick stand of saplings. The farm is now managed for timber and wildlife.

Typical of hazard zone 4, blister rust is scattered throughout the new stand. Although blister rust had killed many seedlings, enough regeneration remained to require additional thinning. Blister rust killed a few larger trees, but these snags are valuable wildlife habitat. Silviculture and pest control includes thinning, pruning, excising cankers, and periodic harvests. *Ribes cynosbati* is spread by birds along nearby fence rows and woods. Rather than eradicating the *Ribes* that attract birds, Tom minimizes pine infection by early pruning of the lower crown. Snowshoe hares assist with this pruning. To establish the next crop, Tom knows he will have to minimize damage from deer and white pine weevil. He protects white pines from weevils by shading young trees beneath overstory red pines. Tom has learned that even in northern Wisconsin blister rust is unlikely to wipe out the white pine. With good stewardship, he produces white pine timber and wildlife habitat on a site with a climate favorable for blister rust and susceptible *Ribes* nearby.

### **Extending the Rust Hazard Concept to the Interior West**

My observations in Wyoming and New Mexico have identified another climatic factor limiting blister rust infection. Because some high-elevation sites are too cold for *Ribes* infection, pine infections on these sites are dependent on spread by *Ribes* growing at lower, warmer elevations.

### **Yellowstone**

Yellowstone National Park is a high caldera (2450 m) surrounded by peaks ranging from 3000 to 3450 m. Vegetation includes *P. albicaulis* (whitebark pine, alpine-subalpine), *P. flexilis* (limber pine, subalpine and montane), and numerous *Ribes* species with their own distinct habitats (alpine, riparian, or forest edge). Because blister rust was considered a serious threat, early trials of *Ribes* eradication were established in the Park. Removal of *Ribes* on control plots, however, failed to reduce pine infection significantly from that on non-treated plots (Berg et al. 1975).

In an unpublished service report, Hendrickson (1970) discussed climatic escape to explain the low rate of pine infection and ineffectiveness of *Ribes* control. He reported temperature and humidity from weather stations representing Park elevations from a low at Mammoth (1950 m) to a high at Eagle Peak Summit (3450 m). My examination of these data confirmed that temperatures at night and during extended wet

periods were well below that required for aeciospore or urediniospore germination and therefore Ribes infection (Van Arsdel et al. 1956). For the most part, pine infections were not the result of spores from nearby Ribes (regardless of abundance and proximity) because it was too cold for them to be infected. Those few pine infections were from Ribes growing at much lower elevations located many kilometers distant.

### **New Mexico**

The idea that temperatures can be too cold for Ribes infection is demonstrated again in the Sacramento Mountains of New Mexico (Van Arsdel et al. 1998). *Pinus strobiformis* is common in montane, mid-elevation forests on a broad dissected plateau above extensive woodlands and below a preeminent volcanic peak (Sierra Blanco). The distribution of *R. pinetorum*, the most important telial host of *C. ribicola* in the region, is only a little smaller than that of the white pine. *Ribes pinetorum* abundance varies from site to site depending on disturbance history and canopy opening; it is rarely infected above 2750 m (too cold). White pines are rarely infected above 3000 m (as result of long-distance spread) or below 2450 m (where susceptible hosts are scarce). Between these limits, blister rust is common wherever susceptible hosts occur together.

### **Commitment**

Geils et al. (2010) presented a brief history of blister rust control and research in North America. Rather than repeat that story or expand with more details, I will only relate several important lessons to researchers, administrators, and foresters.

Ribes eradication methods and expectations were first developed in the Northeast. They did not translate well to the very different conditions in the West. There were more Ribes, bigger Ribes, more species of Ribes. Many of the species were more susceptible than most of the Ribes in the East. The climate was much more favorable for spread of the rust. Access to forest Ribes was extremely difficult; in these remote areas, eradication camps were established at high cost. One foreman observed, "We miss more Ribes per acre than are pulled by the crews of the East."

In spite of the difficulties, Western programs greatly reduced Ribes populations and lowered rust incidence to a very few trees within control zones. Stillinger (1944) thought the rust on the remaining infected trees was from Ribes outside the control zone. Swanson

and Walters (1953) thought these infections were from missed Ribes inside the control zone. Program administrators in Washington DC (Detwiler, Martin, and others) were sure that spread was limited to 300 m as it appeared to be in the East. They dismissed observations from British Columbia that spread could reach kilometers and that a single Ribes could produce much infection (Pennington 1925; Buchanan and Kimmey 1938). Both Stillinger and Swanson had a good understanding of rust epidemiology—Stillinger cited dilution formulae and Swanson recognized the importance of microclimate. My early work on microclimatic influences on rust spread in Wisconsin provided little insight to the extremely complex and highly favorable conditions in the Northwest. A very wet site, low diurnal temperature range, and sea breeze backflow allowed more spread and infection on some sites than considered at the time to be possible.

My work in Wisconsin and later studies in the West have helped to clarify matters of spread and climatic hazard. Spores dispersed from Ribes to pines are transported by diffusion and by air currents (Van Arsdel 1958, 1960, 1965, 1967; Van Arsdel et al. 2006). Ideas of sub-continental spread of aeciospores (Van Arsdel et al. 1998) were fostered by work on peanut rust (Van Arsdel 1973) and applied by Frank et al. (2008). The concept of blister rust hazard zones I developed for Wisconsin (Van Arsdel 1954) has been adopted for numerous regions by many other researchers. It has been revised to fit landscapes in Nevada (Van Arsdel and Krebill 1995) and New Mexico (Geils et al. 1999). Reports by others finding no relation between Ribes and pine infection are clearly wrong; they fail to understand the infection process. More work is still needed on many details of aerial dispersal, temperature control of Ribes infection, and the ultimate question of which Ribes bushes infect which white pine trees.

Chemical control of blister rust included not only use of herbicides to kill Ribes but also attempts to find an antibiotic that would act as a selective, systemic fungicide, killing the pathogen without damaging the pine host. Virgil Moss tested numerous candidates, including Actidione (cycloheximide produced by Upjohn). Although Actidione was not successful on cone rust, results of direct application to basal cankers were promising. But spraying each canker was little improvement over excising it, so a chemical cure remained more a dream than a practical reality.

In 1958, Phytoactin showed promise as a foliar spray delivered from aircraft. Encouraged by limited, short-term, field observations, the Forest Service quickly adopted Phytoactin as an operational tool (Benedict 1981) despite my urging, "Wait, let's test it first." I had noted in a greenhouse test that cankers treated with an antibiotic could resume growth and phytotoxic effects to pine could be severe (Van Arsdel 1962). Research field trials were started, but other testing reported systemic transport of the antibiotic throughout the tree, persistence for two years, and satisfactory performance (Moss 1961). Victory over blister rust was acclaimed. Then, research results from numerous regions and studies came in (Phelps and Weber 1968). Antibiotics had simply masked disease symptoms or temporally reduced spore production. The fuel oil carrier had an equal effect; treatment was not very practical since 87 percent of trees still died. What had gone wrong? The great desire for a chemical control and wishful thinking lead to large tests with more trees than could be carefully observed for a sufficient period. Alternative factors such as secondary fungi and natural inactivation were not considered (Benedict 1981). Although research disproved management's great hope, a good and solid background of information was developed. Rather than continue this work, however, the Forest Service decided to give up on blister rust control and research, and even on planting white pine (Ketcham et al. 1968).

Tom Nicholls showed that white pine in Wisconsin were able to produce abundant regeneration. With escape, old-age resistance, and silviculture, white pines can be grown there. Geneticists have found some white pines in western North America still carry resistance genes; breeders have produced lots of planting stock with these genes. We should have learned from Ribes eradication that disease results from an interaction of pathogen, host, and environment. We should have learned from antibiotic programs the importance of research before deployment. Nursery stock will be expensive; the fitness of resistance and other traits will vary with the environment. Genetic solutions do not relieve us the necessity of understanding epidemiology. I would like to see more research to answer the fundamental question: where can we grow white pines that escape or resist blister rust?

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## Durable Resistance to White Pine Blister Rust: What Are the Prospects?

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### ABSTRACT

Incorporation of partial resistance (PR) into white pines having major gene resistance (MGR) to white pine blister rust has promise to bring durable resistance to this group, and perhaps eventually stabilizing the epidemic.

Readily deployable genes for resistance are not identified easily in forest trees, yet four decades after the discovery of Cr1 (formerly, major gene resistance, or MGR), a dominant gene in sugar pine that confers virtual immunity to white pine blister rust, skepticism and doubt about its utility persist. In large part, this can be attributed to the 'boom and bust' legacy earned by major (R) genes in cereal and other crops earlier in the last century, but also because virulence to Cr1 in sugar pine did in fact emerge, as skeptics predicted, with some ugly effects. Did this mean that Cr1 should be relegated to the dustbin of useless genetic tools? How could one gene in a long-lived perennial withstand the selection pressure and enormous reproductive potential of a fungus? But before we risk throwing out the baby with the bath water, it may be worthwhile to examine how far the analogy to cereal-crop pathosystems applies to WPBR. Ecological and ontogenetic differences alone are, of course, very great between the two, but here we will limit discussion to genetic considerations.

Cr1 and Avcr1 are cognate genes in sugar pine and *Cronartium ribicola* that constitute a gene-for-gene system, albeit a very small one. In gene-for-gene interactions, an R gene for resistance in the host functions if (and only if) it is complemented by a matching gene for avirulence in the pathogen. Accordingly, an R gene is effective against all extant variability in the pathogen, except genotypes with the specific cognate allele for virulence. The match is very specific -- analogous to a lock and key. R genes

are thought to recognize a product (elicitor) of the avirulence gene, which triggers a reaction cascade that terminates in a hypersensitive reaction (HR), stopping the encounter. When avirulence mutates to virulence (at the same locus in the pathogen), recognition by the host R gene is suppressed and normal pathogenesis ensues. Subsequently, sporulation in the overcome host may increase the frequency of virulent inoculum in the local environment from rare to predominant, as neighboring MGR hosts become infected. This is what happened at the Happy Camp test site, where sugar pines with Cr1 came under intense selection pressure, and succumbed within a few years to vcr1.

Fatalistic notions of 'virulent races' lurking in the rust population, ready to destroy our hard gained efforts in selection and breeding, abound. But these notions are misleading, since, strictly speaking, there are no 'races' in the blister rust pathosystem analogous to those in cereal rust systems, which have drawn the most comparisons. For example, a single mutant urediniospore of wheat stem rust in Texas can propagate vegetatively (and exponentially) on compatible hosts, and be blown in clonal clouds of genetically identical spores of like virulence (i.e., a 'race') up the Puccinia path all the way to the prairie provinces of Canada. Many clones with different virulence profiles can exist in such a host/rust pathosystem. But unlike cereal rust pathosystems, blister rust spores infective on pine are basidiospores. These spores are products of meiosis, each genetically unique. So, it is conceptually inaccurate to speak of 'virulent races' of WPBR (often implying an indefinite number of them) out there in the metapopulation. Analogous races could of course exist on Ribes, but there is no evidence for corresponding virulence factors between the two alternate hosts. Of sole concern are virulent genotypes carrying the single, specific gene for virulence (vcr1). Without this gene, the rust, in all its other variability, is completely impotent against Cr1.

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But *vcr1* is rare in natural populations. This may signify reduced fitness in comparison to its wild type (avirulent) alternate allele. In any case, *vcr1* frequency may be no higher than the mutation rate, which, though unknown, might be expected to lie somewhere between  $10^{-4}$  (very high) to  $10^{-8}$  (very low) for virulence loci in plant pathogens. Nevertheless, its frequency can increase exponentially under selection by Cr1, and soon become predominant in a local population, such as a plantation. And this, of course, is the problem we're up against. The problem may be seen to become more tractable, though, by a change in perspective -- from an exclusive focus on finding resistance, to one of managing virulence. These are just different sides of the same coin, of course, but the shift in emphasis may bring a clearer focus and greater degree of optimism.

Since Cr1 is nearly totipotent, the protection this gene provides could be sustained in sugar pine populations if *vcr1* could be maintained at endemic levels in high value plantings with high proportions of Cr1. The key is reduction in inoculum potential; more specifically, reduction in the absolute frequency of *vcr1* (i.e., actual numbers of *vcr1* spores produced). Since *vcr1* originates by mutation of individual spores, any treatment that reduces the amount of inoculum produced lowers the probability of mutants arising in the local rust population. Both genetic and silvicultural techniques can be used in complementary ways to accomplish this.

Partial resistance (PR; aka slow rusting resistance) is very effective in reducing both rate and quantity of infection on families and individual trees of white pines. Recent (unpublished) data on 135 sugar pine progenies at the Happy Camp test site show up to a thirty-fold difference in numbers of infections among families. Since PR is non-specific -- no more vulnerable to *vcr1* than wild type inoculum -- it effectively sanitizes a significant proportion of

incoming inoculum, including *vcr1* (Cr1 takes care of all non-*vcr1* inoculum). Buffering Cr1 genotypes with PR can thus reduce the amount of effective *vcr1* infections on Cr1 hosts, as well as attenuate subsequent increase in *vcr1* frequency.

Eradication of *Ribes* in and immediately surrounding new plantations will greatly reduce overall inoculum potential. A strong association between pine infection and proximity to *Ribes* holds in all except wave years, when inoculum is widely distributed regionally.

Three other North American white pines (western white pine, *P. monticola*; southwestern white pine, *P. strobiformis*; and limber pine, *P. flexilis*) are each known to have a major gene for resistance to blister rust; these are different from each other and from Cr1. However, the existence and frequencies of cognate genes for virulence are as yet unknown, except for *P. monticola*. The distribution of the virulence gene (*vcr2*) that neutralizes the major gene in this species (Cr2) has an anomalous and patchy distribution -- relatively high in some areas and absent in others where it might be expected to occur. Although virulence management theoretically could apply to all of these species, it will only be feasible where *vcr* frequencies are naturally low, and useful frequencies of PR exist.

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## Cryptology 101: Lecture 2 - Application to WPBR

Rich S. Hunt<sup>1</sup>

I begin with a quick review of lecture 1 given during my Outstanding Achievement Award presentation. First, we saw that during the twilight of the dark ages that it was unnecessary for learned men to have data in order to discuss and conclude about various topics. Even these perspectives were probably secondary to that of the king and pope. This was followed by anecdotal evidence and causal observations that the learned men debated; their perspectives and conclusions were broad and various. The year 1752 was a turning point in phytopathology, because for the first time all the learned men were forced to the same perspective by a powerful set of replicated experiments and this method of doing the science is considered to have trumped deductive logic. More recently, it has become necessary to repeat experiments in time and/or space. (By the way, although Tillet had all the Bordeaux learned men on the same page in 1752, he repeated his 1751 experiments in 1752 and 1753 and published in 1754 – [en.wikipedia.org/wiki/Mathieu\\_Tillet](http://en.wikipedia.org/wiki/Mathieu_Tillet)).

Likewise, here in lecture 2 there will be some hyperbole. Also here, to emphasize that it is criticism of the science that I am making and not of the scientists themselves I will be giving individuals pseudonyms. Some may think this is unnecessary because the works I will be referencing are old, and the researchers have been retired many many years, so that few would know them by name. Nevertheless, I am going to hide the identities of the characters in my story by pseudonyms.

So, upon a time there was a loud thud in the land of Idaho as a mass of data landed on the desks of Ronald and Mc Hoof. This data was the putative resistance responses of western white pine seedlings derived from the Moscow arboretum. These fellows applied some pre-1752 technology to the data

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and concluded the following: 40 percent were resistant because the infected needles fell off before *C. ribicola* reached the stem, or “early needle shed” (eNS), and this happened about 1 year post-inoculation, or just prior to September 21; 10 percent had “spots only” (SO); i.e., no cankers, but spotted; 6 percent had “bark reactions”; 15 percent had “no spots and no cankers” (NO); 29 percent were normally cankered (Hoff et al. 1973; 1980). Again pre-1752 technology deemed the eNS and SO to each be caused by a pair of recessive genes (McDonald and Hoff 1971). At WIFDWC 2002 I told you that we have a plantation established with Moscow arboretum seed that fits the 71 percent resistant 29 percent susceptible pattern and we did everything we could to try and observe eNS in inoculated seedlings. No matter how hard we tried using different inoculum sources and different incubation locations (Hunt 2004), examination of inoculated seedlings 13 months post-inoculation; i.e., in October, spotted needles always remained on inoculated seedlings. What we needed was to apply some post-1752 technology and answer the question: “when does the stem become cankered”. We considered pulling off infected needles at different incubation times, but in a previous experiment where spotted needles were removed within a week of appearing, it appeared that if the needle was spotted then the stem was already infected. But, perhaps we may have missed a spot; it certainly didn’t seem like a foolproof experiment. If we pulled all the needles off at different incubation times the seedlings would die.

What we needed was a bean plant and follow what Yarwood would have done; i.e., have a treatment on one leaf and use its paired partner as a control. We made our pine trees into Yarwood bean plants by covering the top during inoculation, so that only the bottom of the seedling was inoculated. We would then have a compliment of healthy needles to keep the seedling alive while we could then pull off all the inoculated needles at various times to determine when the stem became infected. We repeated this in about 4 different years and at various locations ranging from Cowichan Lake on Vancouver Island to Cranbrook in the Rocky Mountain trench. This data indicates that

most seedlings are cankered in the stem within 1 month of spot counting; i.e., June at the coast and July in the interior (Hunt et al. 2007). So if eNS occurs in September, it is occurring too late to be the cause of resistance. However, one could argue that it is the consequence of resistance.

Another approach to the eNS problem is to cross two eNS trees, as such a cross should yield 100 percent of the offspring with shedding needles and subsequently they would not develop any cankers. The BCFS obtained some selected Idaho stock for a seed orchard several years ago and being mature stock, cones were produced within a few years. Some of these trees were classified as eNS trees. We made crosses among these trees and inoculated the full sib families.

At this point I would like to tell you about the religious zealot. He claims to be a Unitarian. Unitarians have the ability to change their religious belief when new data comes in, contrary to most religions that have fixed dogma that is inflexible when new data comes in. I would like to introduce a new religion – eNS (figure 1). The worshippers of this religion ignore the data from the Yarwood bean experiment, because it does not fit with their dogma. When told that crossing eNS X eNS produced nothing they were glum, but eventually they came back saying that the original designations of the trees they supplied for the seed orchard were probably incorrect, so it was unlikely that we had made any eNS X eNS crosses. This seems like a fairly dogmatic religion.

I would like to introduce a new player, whom I will call Mr. Braw. In his Phytopath paper documenting the Cr2 gene, he introduced me to the concept of the “pollen cloud” (Kinloch et al. 1999). We can use this concept in the Vlem plantation as there is virtually only Moscow arboretum sources trees left in the plantation. Since the susceptible trees are dead the relative proportions of the resistant trees increase. For eNS this is 57 percent. So by following Mr. Braws example: if we can collect the cones from a eNS tree within the plantation, 57 percent of the seedlings should be resistant from eNS pollen X with the sampled parent. No families approached 57 percent, but our sample size was only three families. The BC Forest Service resampled the plantation, but by then the number of rusted trees had increased slightly, but fewer of them were contributing to the pollen cloud because many rusted trees died. The percentage of eNS parents contributing to the pollen cloud ranges from 49 to 67 percent, depending on how the Armillaria killed trees are removed from the sample and whether one accepts NO parents. Similarly for SO, the percentage

would vary from about 9 to 14 percent. Using 100 coloured beads (whose colours represent the proportions of the putative resistant traits) in a bag and making 100 withdrawals, we can find the sample size that is confident 95 percent of the time is 4 or less families for eNS, and similarly for SO it is 16 families. The sample size was 17 families, therefore we should see four families losing needles so that >48 percent of the siblings are canker free, and one family with SO resulting in 9 to 14 percent of the siblings canker free. The data I am presenting is preliminary data belonging to the BC Forest Service. About 100 seedlings/17 families were inoculated in 2008. All of the approximately 1700 seedlings were cankered in October 2009 and April 2010. By July 2010, 13 families were 100 percent cankered. The four remaining families had 4 percent or less canker-free siblings, far below the expected for eNS or SO.

## Saving white pine



• eNS

**Figure 1**—Innocent looking goddess of early-needle-shed resistance.

Hopefully everyone is on the same page and can not only conclude that eNS does not exist, but that NO or SO also do not exist, or are exceedingly rare. Inoculations of *P. monticola*'s sibling species, *P. strobus* L. by neither Riker et al. (1943), Patton (1967), Heimburger (1972), Zufa, (1981), Smith et al. (2006), nor Jacobs et al. (2009), has ever reported any eNS, NO, or SO. Yet the zealots of the eNS religions see the NO, eNS and SO traits in – *P. albicaulis* Engelm. a species parsecs removed from *P. monticola* or any of its sibling species! Also at Placerville, these “traits” have not been observed in either inoculated sugar pine (*P. lambertiana* Doug.) or Sierra white pine (*P. monticola* D. Don). In fact, 20 years ago, at a “Rust Busters” meeting in Dorena, Mr.

Braw challenged the supporters of eNS to re-test one of their eNS “resistant” seedlings and when the phenotype was reconfirmed, to forward an example to him for testing at Placerville. To date he has not received a single example from any program routinely testing for blister rust resistance in western white pine. Evangelists of the eNS religion often cross the border into Canada preaching their beliefs, unfortunately, with considerable success. Recently, I hear that my Canadian colleagues are willing to send their precious white bark pine seed to a USA puppy mill for processing. I say give your head a shake! How would you use seedlings classified as eNS, NO, or SO; or for that matter, any classification system derived from one of these puppy mills? I implore people with power and influence within the US Forest Service, such as Terry and GERALD, to pick up a wooden stake and drive it through the heart of the beast.

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## After 100 Years, Is Coevolution Relevant?

Geral I. McDonald<sup>1</sup>

“This potential independence between DNA and epigenetic genotype for a gene even raises the potential for intragenic epistasis whereby the DNA and epigenetic genotypes for a gene interact to determine the phenotypic outcome. The simplest example would be where the DNA polymorphism toggles between a functional and nonfunctional allele and the epigenetic polymorphism toggles a silent and expressed gene.” (p 483 Kliebenstein 2010)

On the 100<sup>th</sup> anniversary of the introduction of *Cronartium ribicola* into western North America, it is fitting to assess the philosophical foundation of plant pathology and forest ecology. We should ask whether this foundation provides sufficient understanding of blister rust, other diseases of North American forests, and general forest ecology to insure the application of biologically appropriate and sustainable management scenarios. Perhaps the most significant advances in understanding how host-pest interactions fit into the scope of biology have occurred in the last 10 years. This review focuses on an introduction to four recent developments that are fundamental to our understanding of how life originated, evolves, and functions. First, the almost universally accepted model of life, the Modern Synthesis (Huxley 1942), has provided biologists with a solid philosophical foundation for 70 years. In particular, this model has provided the theoretical basis for population genetics (Stern and Orgogozo 2009). Knowledge gleaned from complete genome (DNA) sequencing (see Mattick 2009) and the discovery of short, non-coding RNA transcripts (see Siomi and Siomi 2009) has eroded principal aspects of this venerable model and forced significant restructuring, which is currently in progress (Pigliucci 2010). Second, relevant new

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concepts (ecological immunity, plant immunity, and ecological speciation) born under the framework of the Modern Synthesis need updating. Integration of the genomic revelation into these concepts stands ready to revolutionize our understanding of forest ecology in general and plant disease in particular. Finally, this new lens will be focused on white pine blister rust in an attempt to uncover some new understanding.

### Expanding the Modern Synthesis

Essentially all biological investigations of white pine blister rust were conducted from the philosophical perspective of the Modern Synthesis. A recent summary of the Modern Synthesis is quoted below to emphasize how a new model will contribute essential insight into forest biology.

“1. Heredity occurs through the transmission of germ-line genes. Genes are discrete units that consist of DNA and are located on chromosomes.

2. Hereditary variation is equated with variation in DNA base sequence. Cases in which acquired variations appear to be inherited can all be explained in terms of variation in DNA.

3. Hereditary variation is the consequence of (i) the many random combinations of pre-existing alleles that are generated by the sexual processes; and (ii) new variations (mutations) that are the result of accidental changes in DNA. Hereditary variation is not affected by the developmental history of the individual. There is no “soft inheritance” (in which heritable variations are the result of environmental effects, use and disuse, and other factors).

4. Selection occurs among individuals that are, at almost all times, well-defined entities. The target of selection is almost always the individual, which may co-evolve with its symbionts and parasites. Although some role for group selection has been acknowledged, this form of selection is assumed to be of marginal significance in evolution. The community is rarely considered as a target of selection.

5. Heritable variations have small effects, and evolution is typically gradual. Through the selection of individuals with phenotypes that make them slightly more adapted to their environment than other individuals are, some alleles become more numerous in the population. Mutation pressure is not an important factor in evolution. With a few exceptions, macroevolution is continuous with microevolution, and does not require any extra molecular mechanisms beyond those operating during microevolution.

6. Evolution occurs through modifications from a common ancestor, and is based on vertical descent. Horizontal gene transfer has minor significance – it does not alter the basic branching structure of phylogenetic divergence. The main pattern of evolutionary divergence is therefore tree-like, not web-like.” (pp 389-390 in Jablonka and Lamb 2008)

### **Expansion of the Modern Synthesis Focuses on the Following Challenges to its Dogma.**

“1. Heredity involves more than DNA. There are heritable variations that are independent of variations in DNA sequence, and they have a degree of autonomy from DNA variations. These non-DNA variations can form an additional substrate for evolutionary change and guide genetic evolution.

2. Soft inheritance, the inheritance of developmentally induced and regulated variations, exists and is important. Soft inheritance includes both non-DNA variations and developmentally induced variations in DNA sequence.

3. Since many organisms (including humans) contain symbionts and parasites that are transferred from one generation to the next, it may be necessary to consider such communities as targets of selection.

4. Saltational changes leading to evolution beyond the species level are common, and the mechanisms underlying them are begging to be understood. Macroevolution may be the result of specific, stress-induced mechanisms that lead to a re-patterning of the genome- to systemic mutations.

5. The Tree Of Life pattern of divergence, which was supposed to be universal, fails to explain all the sources of similarities and differences between taxa. Sharing whole genomes (through hybridization, symbiosis, and parasitism) and partial exchange of genomes (through various types of horizontal gene transfer) lead to web-like patterns of relations. These web-like patterns are particularly evident in some taxa (e.g. plants, bacteria) and for some periods of evolution (e.g. the initial stages following genome sharing or exchange).” (p 390 in Jablonka and Lamb 2008)

New challenges will continue to arise. In the meantime, these known challenges require additional discussion to examine why a reformulated Modern Synthesis carries important consequences for forest biologists.

### **Challenge Number 1– Genome and Epigenome**

An accumulation of molecular and other data demonstrate that cells contain two sources of inheritance. The genome, home of DNA, and the epigenome, a non-DNA cellular memory that can pass through somatic cell lines, germ cell lines, and across generations. This heritable information is carried by two epigenetic signals (Bonasio et al. 2010). Cis epigenetic signals are physically associated with chromosomes (e.g. DNA and histone methylation marks) and trans signals are composed of various molecules partitioned by cytoplasm transfer. The cis signals are collectively known as the methylome and each cell type in an organism carries a unique signal that guides development and physiological function through gene regulation (see Rival et al. 2010). The methylome features sufficient conserved characteristics to permit construction of phylogenies (Zemach et al. 2010).

Quantitative and molecular genetics investigations of complex traits were based on the assumption of stable transmission of causative alleles encoded in the DNA genome (Johannes et al. 2008). This assumption was recently falsified in an experiment designed to control for DNA variation while maintaining variation in the epigenome. Heritability, arising from the epigenome, of 0.3 for height growth and 0.27 for flowering time was demonstrated in *Arabidopsis thaliana* (Johannes et al. 2009). The last few years have witnessed an explosion of papers investigating epigenomes, epigenomics, epialleles, and methylomes (see Baker 2010, Rival et al. 2010).

## **Challenge Number 2 - the “Ghost of Lamarck”**

It is clear that short (18 to 200 nucleotides long) non-coding RNA transcripts, the genome, and the epigenome (see Ghildiyal and Zamore 2009; Lelandais-Briere et al. 2010), participate in sensing and “recording” information gleaned from abiotic and biotic environments to facilitate non-DNA inheritance of “acquired” traits (Hollick 2008; Bonduriansky and Day 2009). This phenomenon, also known as soft inheritance (Jablonka and Lamb 2008), is not well understood in plants. However, soft inheritance is currently known to influence regulation of all life-cycle phase transitions in plants, from seed germination to seed production, floral development, shoot apical meristem development, leaf development, vascular development, root development, abiotic and biotic (pests and competition) stress responses, and growth hormone signaling (Jung et al. 2009). In most of these cases, environmental induction is believed to be the source of variable expression (see Angers et al. 2010). In addition to the above participation in soft inheritance, small RNA transcripts travel throughout plants in the sap stream (Dunoyer et al. 2010; Zhang et al. 2009). Some specific examples of soft inheritance are: (1) juvenile growth rate in springtails (*Orchesella cincta*) (Eilers et al. 2008), (2) seed production (Whittle et al. 2009), and (3) flower production and height growth, as mentioned above, in *Arabidopsis thaliana*. Other examples are: (2) timing of bud phenology in Norway spruce (Yakovlev et al. 2010), (2) antibiotic resistance in bacteria is inherited via an epigenetic pathway (Adam et al. 2008), and (3) regulation of plant immunity responses to viral, bacterial, insect, and fungal pests of plants (Padmanabhan et al. 2009; Pandey and Somssich 2009). Finally, small RNA transcripts are upregulated by *Cronartium quercuum* f. sp. *fusiforme* infection in *Pinus taeda* (Lu et al. 2007) but the epigenomic connection has not been investigated in this couplet. Symbiotic associations in plants also involve these regulatory pathways and epigenetic/RNA connections (Lelandais-Briere et al. 2010).

## **Challenge Number 3 – Transgenerational Transmission of Symbionts and Parasites**

It may be necessary to consider transgenerational communities as targets of selection. These situations may be of particular interest to the study of resistance as discussed below.

## **Challenge Number 4 – Rapid Evolution**

Ecological speciation, also known as divergence-with-gene-flow, is currently a hot topic all its own (Via 2009). Studies illustrating detailed function of the epigenetic/RNA system in ecological speciation were not found. However, there is considerable support for the idea that environmentally driven genome-epigenome rearrangements could be responsible for divergence-out-gene-flow (Angers et al. 2010; Turner 2009; Aubin-Horth et al. 2009). Dramatic changes in the small non-coding RNA transcriptome are a known feature of artificial wheat hybrids (Lui et al. 2009) but details are lacking. An examination of the “long” transcriptome (transcripts longer than 100 nucleotides) of a species known to be expressing ecological speciation, the apple maggot, revealed a catalogue of potentially useful genes (Schwartz et al. 2009). Unfortunately these authors did not report on the “short” transcriptome (transcripts 18 to 30 nucleotides long). Study of the short transcriptome currently requires specific targeting through separation from the total transcriptome before sequencing (Zhang et al. 2010).

## **Challenge Number 5 - Web-of-Life or Tree-of-Life**

Phylogenetic and phylogeographic theory will be significantly impacted if the Tree-of-Life morphs into the Web-of-Life, since these theories are built on the assumption of a last universal common ancestor (see Doolittle 2009; Koonin 2009).

## **Ecological Immunology**

A field of study that weds ecology and immunology was initiated in the early 1990’s from attempts to understand resistance to parasites and immune responses in birds and other vertebrates (Sadd and Schmid-Hempel 2009). Recent realization that ecological immunology applies to all life has codified important concepts (Sadd and Schmid-Hempel 2009) and led to the application of these concepts to all of biology (Schulenburg et al. 2009). Inclusion of plants was accelerated by the realization that plants also possess a fully functional immune system (Jones and Dangl 2006).

## **Plant Immunity**

Upon attack, plants display an initial array of structural barriers and preformed antimicrobial metabolites. How these barriers work is not well understood, but it is clear that many pathogens can routinely penetrate these preformed defenses and activate systems of systemic immunity. The following short sketch of how plant immunity is currently understood to function is based on a recent review (Pieterse et al. 2009). Small

molecules known as phytohormones are essential signaling components that regulate plant growth, development, reproduction and survival. The primary defense phytohormones are salicylic acid (SA), jasmonates (JA), and ethylene (ET). However, abscisic acid (ABA), auxins, gibberellins, cytokinins and brassinosteroids may also play a role. The specific signal signature of the phytohormonal blend produced by a particular combination of host, trigger (i.e., pathogen, endophyte, symbiont, insect, parasitic plant, or wound), and environment activates a specific set of defense-related genes. Plant pathogens are classified as biotrophs (derive nutrients from living host tissue), necrotrophs (kill host tissue, then feed), and hemibiotroph (function both ways at different life cycle stages) and each triggers its own signal signature.

### **Basal or Quantitative Resistance**

After initial penetration by a biotroph, necrotroph, or hemibiotroph, one of three types of recognition events occurs. First, pathogen-associated molecular pattern triggered immunity (PTI) initiates a cascade of basal defense gene activity that typically leads to (1) synthesis of callose and lignin to fortify the cell wall, (2) production of secondary metabolites such as phytoalexins, (3) accumulation of pathogenesis-related proteins, some of which can degrade fungal cell walls (Pieterse et al. 2009), and (4) stomata closure (Hou et al. 2009). Various pest infections, symbiotic associations, and wounding can prime basal resistance thereby increasing its effectiveness (Ahmad et al. 2010). Primed (Ahmad et al. 2010) and unprimed (Chen et al. 2010) basal defense appears to be the cellular mechanism leading to systemic induction of well known broad spectrum, durable, horizontal, or quantitative resistance mechanisms. Evidence from microarray experiments is beginning to show connections among basal resistance, priming and the epigenetic/small RNA gene regulatory network through regulation (positive and negative) of genes known to be associated with PTI in the soybean/*Phytophthora sojae* (Wang et al. 2010) and barley/*Puccinia hordei* (Chen et al. 2010) couplets. These studies also show that large numbers of genes (637 in soybean and 802 in barley) change state upon inoculation in resistant and susceptible comparisons. In general, plant hormones seem to be important in disease and defense against all microbial attacks (Bari and Jones 2009). In the barley/rust couplet, infection induced significant changes in a transcription factor (HvERF4) known to be active in defense

pathways relating to ethylene, jasmonic acid, and abscisic acid (Chen et al. 2010). Finally, degree of basal resistance expression is influenced by temperature through the regulation of a defense-related proteins and/or impacts on SA signaling (Zhu et al. 2010). Expression of quantitative disease resistance is often associated with various aspects of growth and development such as flowering time, stomata development, and ability to repel water (Poland et al. 2008).

### **Major Gene or Qualitative Resistance**

If a specific host-pathogen couplet has a coevolutionary history, the pathogen may acquire effector molecules that promote virulence to initiate effector-triggered susceptibility (ETS). At some point in their coevolutionary history, the host acquires resistance proteins that sense the pathogen effector. This action initiates a secondary response termed effector-triggered immunity (ETI). ETI produces a burst of reactive oxygen species that initiate the well-known programmed hypersensitive cell death, a well known indicator of major gene resistance.

### **Endophytes and Symbionts**

Beneficial microbes also communicate with the host, but trigger a phenotypically similar yet distinct kind of systemic immunity called induced systemic resistance (ISR). With ISR, signal molecules are JA and ET; the induced condition primes for defense, not outright activation of defense. Pathogens, insects, and wounding can trigger a JA- and ET-mediated response. However, crosstalk among the phytohormones is common and many details are lacking. The above model of host-pest interaction has been developed through the use of Arabidopsis and a few other model systems. Many aspects of the system have been observed in various species of conifers and angiosperm trees (Eyles et al. 2010).

### **Common Machinery**

The epigenetic/small RNA gene regulatory network appears to modulate ETI as well PTI (Padmanabhan et al. 2009). The major players (microRNA and small interfering RNA) also participate in epigenomic interactions leading to soft inheritance. Multiple specific miRNA transcripts all functioning in different manners can be produced by a single micro RNA gene (Zhang et al. 2010), and there may be thousands of these micro RNA genes in the "junk" DNA. Biogenesis of small interfering RNA (siRNA) transcripts is even more complex. In this case, sense

and antisense transcripts are derived from overlapping regions between adjacent genes, pseudogenes, and distant compatible genes (Ghildiyal and Zmore 2009; Muro and Andrade-Navarro 2010; Rival et al. 2010). Once PTI or ETI is activated at the site of infection, SA often spreads systemically to produce systemic acquired resistance (SAR). This long-lasting and broad-spectrum disease resistance is generally triggered by biotrophs. SAR initiated by necrotrophs and wounding is most often triggered by JA or ET.

### **Tolerance**

Tolerance (see Raberg et al. 2009) is part of the ecological immunity paradigm with its own physiology and genetics. This concept, which applies to plants and animals, is difficult to understand because quantification requires assessment of reaction norms for host fitness across a range of pest burdens. Degree of tolerance is indicated by differences in slopes of the obtained reaction norms and equal reaction norm slopes are a measure of host general vigor. No examples of functioning epigenetic/small RNA gene regulation networks were found for tolerance reactions to include in this review.

### **Ecological Speciation**

Conventional wisdom states that gene flow will homogenize adjacent populations so strongly that divergence in the absence of geographic separation will not occur. But, the sheer number of examples of phenotypic divergence between adjacent populations (suture zones) raised the need for an explanation. The following sketch is based on a recent review of “divergence-with-gene-flow” (Via 2009). Current understanding of speciation genetics is almost entirely based on long-range retrospective studies conducted from the viewpoint of “good species”. This so-called spyglass model has culminated in the dominance of the idea that reproductive isolation (no gene flow) is essential for speciation to happen and that geographic isolation is a necessary condition to stop gene flow. A new approach, called the magnifying glass model, is based on the study of how genetic x environment interactions can lead to incipient reproductive isolation among ecotypes or races in the absence of geographic isolation. The obvious result of having two evolutionary avenues is that ecological speciation can happen rapidly with the development of “ecological barriers” or classic speciation can happen slowly with geographic isolation. Divergence-with-gene-flow can occur only if reproductive barriers developed within the genome are strong enough to maintain phenotypic

differentiation. This condition means strong selection and rapid divergence can happen when selection is directed at multiple traits bearing on resource or habitat use. In this case, selection is also strong against migrants and hybrids. Studied examples indicate that conditions suitable for development of divergence-with-gene-flow are common in host-pest interactions but are not limited to these interactions. Additional important aspects not recognized by Via (2008) are the roles gene expression and epigenetics can play in ecological speciation. However, others (Wolf et al. 2010) suggested that integration of gene network thinking into speciation genetics may have an impact similar to that of Mendelian genetics on Darwin’s original framework.

### **Ecological Immunology x Ecological Speciation**

Finally, a brief examination will show how critically important the environmental context of host-pest interactions is for determining specific outcomes. This brief synopsis is largely based on a recent review of immunity in a variable world (Lazzaro and Little 2009). Given that activation of immunity incurs physiological costs, the whole organism is involved when a part is attacked. Further, the strength and duration of the immune response is heavily influenced by the overall condition of the potential host. Important variables are abiotic environment, genotype x environment interactions of host and pest, host genotype x pathogen or symbiont genotype interaction (biotic environment), and pleiotropic constraints. The potential importance of these interactions is illustrated by the results from a study reported in Lazzaro and Little (2009). A three way host genotype x pest genotype x environment (presence or absence of rhizobacteria) interaction showed that as much as 42 percent of the barley performance and 32 percent of the aphid performance was explained by the 3-way interaction.

To understand why outcomes of such associations are so difficult to predict, consider a hypothetical host x symbiont x pathogen expression of basal resistance (all eukaryotes) in two environments given that each of the living participants were cloned to ensure the same interacting sets of DNA in both environments. To examine how these interactions might function, we need a little more background. We know from our discussion above that about 600 host genes respond to infection. Let us assume 200 additional host genes respond to the symbiont, that 100 pathogen and 100 symbiont genes also respond. Thus, 1000 genes are

interacting. Not all these responders encode proteins. So assume that 0.8 (800 genes) represent non-coding elements associated with both genome and epigenome which are differentially expressed by cell type (Baker 2010). There are eight types of non-coding elements (Alexander et al. 2010), of which transcription factors, small interfering RNA, micro RNA, and pseudogenes are the most well known. In addition, expression varies by cell type and number of cell types (ca. 4 for fungi and 20 for conifers) varies by complexity of the organism (Alexander et al. 2010). Alternative splicing of messengerRNA from coding genes leads to multiple expressions of single genes in eukaryotes (Nilsen and Graveley 2010); therefore 200 (coding genes) is multiplied to about 600 expressed states (assume 3 alternative forms per gene). Next, consider that each organism has its own genotype x environment, genotype x development, and genotype x ontogeny interaction (Kliebenstein 2010). Also individual microRNA genes can generate multiple functional transcripts (Zang et al. 2010), and pseudogenes can participate in the production of natural antisense transcript gene regulators (Muro and Andrade-Navarro 2010). Even with this simplified level of complexity, it is evident that we are a long way from understanding basal resistance in conifers!

### **Potential Blister Rust Answers and Pitfalls**

Perspectives provided by an expanded Modern Synthesis (i.e. evolution, development, and function rise from gene regulatory networks that are formulated via the interaction of genomes, epigenomes, and environment) can further inform our understanding of white pine blister rust (WPBR).

### **Why do Plantations Exhibit so Many Pest Problems?**

As discussed above, the realization is growing that gene regulatory networks sense information from the environment and store gene regulation profiles for the current as well as future generations. We expect profile construction to start at embryogenesis and continue to develop until at least reproductive maturity. In long-lived organisms, the gene regulation profile may change year by year to ensure an adequate response to accumulated lifelong stressors – abiotic as well as biotic. Over a few generations, these gene regulation profiles may become incorporated into the genome so as to facilitate rapid “adaptation” to environmental change (e.g., ecological speciation). Populations of long-lived tree species may be especially fine-tuned to specific populations of

endophytes, symbionts, pests, and competitors as well as multiple factors in the abiotic environment (see Bossdorf et al. 2008). We have already examined how multiple levels of genetic and environmental interaction can produce an incredible range of outcomes. Artificial reforestation methods could cause much greater problems than currently realized because many sources of interaction are ignored under current practice. Disruption of local gene regulation profiles in natural hybrids could be the driving force in ecological speciation (Wolf et al. 2010) and artificial (breeding) and natural (offsite) hybrids likely contribute to plantation problems in the same fashion. Since forest trees have extended development periods, it is also possible that many aspects of nursery (e.g., growing seedlings in mismatched biotic and abiotic environments) and planting practice might impart negative influences for the life of a plantation. Thus, one would expect intermittent expression of significant pest and/or environmental problems at a local scale for off-site (i.e., planted) plantations. The application of the expanded Modern Synthesis paradigm also leads to two additional conclusions: (1) global climate change would likely cause more disruption in plantations than in natural stands, and (2) development and interpretation of experimental plantations requires much caution. Next up is a case in point.

### **Why did Blister Rust Resistance Fail at the Merry Creek and Hold Firm at Gletty Creek?**

Large bulk-seed lots of northern Rocky Mountain western white pine (WWP) representing: (1) open-pollinated controls, (2) full-sib, 1<sup>st</sup> generation crosses (phenotypically resistant parents), 2<sup>nd</sup> generation full-sibs (1<sup>st</sup> generation crosses), and full-sib back crosses (1<sup>st</sup> generation x original parents) were divided into two groups and planted at two sites (Merry Creek 1970, Gletty Creek 1972) (Bingham et al. 1973). After 26 years of exposure, all sources planted at Gletty Creek were still below expected infection threshold; whereas, all sources planted at Merry Creek dramatically exceeded expected infection thresholds (McDonald and Decker –Robertson 1998; McDonald et al. 2004). Was this failure attributable to a resistance gene collapse or some other cause? A major gene for resistance to WPBR is known to be present in some WWP subpopulations (Kinloch et al. 2003), but neither the host resistance gene nor the pathogen virulent gene are known to exist within the range of the WWP subpopulation used in these plantations (Kinloch et al. 2003; Kinloch et al. 2004).

Several lines of evidence indicate that the population tested at Merry Creek and Gletty Creek is expressing quantitative (basal) resistance (McDonald and Decker–Robertson 1998; McDonald et al. 2004).

#### **Basal Resistance in WWP:**

Investigation of quantitative resistance expression in WPBR has deposited several clues about its nature. The updated Modern Synthesis implies that understanding the failure at Merry Creek requires consideration of host developmental stages, cell types, influence of the physical environment, and associated symbionts. Since stomata are the infection court and white pines produce 3 kinds of needles or organs (i.e., cotyledons, primary needles, and secondary needles), which each supports at least 4 cell types, as many as 12 methylomes could be involved, in addition to host and rust genetics. Cotyledons (i.e., embryonic leaves) have been used to assess qualitative resistance to WPBR (Kinloch et al. 2004), but not basal resistance.

We will compare results from inoculation tests at Moscow, ID in 1964, 1966 and 1967, 1968, and 1970. Primary needles (i.e., first-year growth) were inoculated and inspected in several tests due to the tendency for delayed germination. Most often, seedlings were inoculated after bud set in their 2<sup>nd</sup> year when only secondary needles were present. Some combinations of years, sites, and/or families led to a second flush of leader growth that also supported primary needles; however, these are customarily removed before inoculation. Resistance observed in the Merry Creek families and other sources of WWP (McDonald and Hoff 1970b, 1971; Hoff and McDonald 1971; McDonald et al. 2004; Hoff et al. 1980) are likely basal. The six resistance phenotypes defined in these studies, listed in order of occurrence along the plant-development/cell-type pathway, are: (1) needle-spots-only in primary needles (NOSP), (2) rust-free secondary needles (RF), (3) reduced-needle-lesion-frequency in secondary needles (RNLF), (4) needle-spots-only in secondary needles (NSOS) composed of premature-needle-shed of secondary needles (PNS) and fungicidal-short-shoot (FSS), and (5) partial-girdle resistance (PG) expressed in stems and branches.

#### **Needle-Spots-Only Primary (NSOP) vs. Secondary Needles (NSOS)**

Influence of foliar type at infection on canker presence 3 years after inoculation was assessed in the 1966 test (data on file Moscow FSL) and the 1970 test (Hoff

et al. 1980). In the 1970 test (6 years in greenhouse and lath house), 23 percent of 35 F<sub>1</sub> seedlings supporting needle spots on primary foliage were clean (no rust) after 3 years and 36 percent of 1,108 seedlings with needle spots on secondary needles (mixture of 2- and 3-year-old plants) were clean (Hoff et al. 1980). In the 1966 inoculation (outside Moscow ID), susceptible (open-pollinated infected parents) and resistant (F<sub>1</sub> and F<sub>2</sub>) seedlings were compared (Moscow FSL data on file). About 60 seedlings in each primary and secondary class for each seed lot (i.e., F<sub>2</sub>-primary-full sib family) supported needle infections at 12 months. Percent clean in each class was determined 3 years after inoculation. Results, in percent clean for the three stocks (about 250 seedlings/cell), were control (7 percent), F<sub>1</sub> (10 percent), and F<sub>2</sub> (18 percent) for first year seedlings, and 22, 34, and 65 percent, respectively, for second year seedlings. Individual F<sub>1</sub> and F<sub>2</sub> families, 60 individuals per cell, exhibited dramatic differences in the primary vs. secondary comparison. Family 242 x 224 (F<sub>1</sub>) showed no difference (15 percent vs. 13 percent); 129 x 224 showed a large difference (0 percent vs. 38 percent); and 208 x 241 showed another large difference (8 percent vs. 39 percent). Family 58x25-9 X 18x17-9 (F<sub>2</sub>) changed from 7 percent to 83 percent, while 58 x 25-9 X 22x1-4 changed from 24 percent to 28 percent. Relatively small differences between the tests for F<sub>1</sub> stock (NSOP 23 percent vs. 10 percent and NSOS 36 percent vs. 34 percent) may be noteworthy because of the dramatic difference in growth regimes. The 1970 test was conducted under controlled conditions for the entire 6-year duration, in which the first 3 years alternated between greenhouse and lath house and the last 3 years were entirely in the lath house. In the 1966 test, seedlings were outside for the entire duration of the test. Does this indicate some development x environment interaction for NSOP and none for NSOS? On the other hand, the individual family results indicate a highly significant genetic (genome and epigenome?) x development interaction.

#### **Reduced Needle Lesion Frequency (RNLF) Resistance**

This mechanism was reported for 80 full-sib F<sub>1</sub> families inoculated in their 2<sup>nd</sup> growing season in 1966 (Hoff and McDonald 1971) and 120 different F<sub>1</sub> families inoculated in 1970 (McDonald et al. 1991). Both tests were conducted on seedlings grown outside at Moscow, ID. The average spots/meter of needle length in the 4 lowest and 4 highest families for

the 1966 inoculation (spores cast not reported) was 1.75 and 14.25, respectively, or an 8.1x difference and a mean of 7.5. The same data for the 1970 inoculation (2,500 spores/cm<sup>2</sup>) was 2.15 and 18.4 or 8.6x differences and a mean of 8.4. The species difference reported for secondary needles in the 1970 greenhouse growth and inoculation test (4,900 spores/cm<sup>2</sup>) was 0.1 (*P. peuce*) and 28.0 (*P. ayachuite*) or a 280x difference. The mean spotting frequency of WWP (F<sub>1</sub>) in the greenhouse test was 5 spots/meter of needle. In another experiment conducted in the greenhouse/lath house at Moscow, 11 two-year-old WWP and *P. lambertiana* families obtained from the Dorena Program located in Oregon were quantitatively inoculated in large settling towers featuring rotating basidiospore delivery beds (McDonald et al. 1991). Inoculation efficiency was calculated on the basis of stomatal area exposed to spore cast as determined by multiple spore traps in a small area. Spores were delivered, by design, at 8 levels varying from 600 to 18,000 spores/cm<sup>2</sup>. Yet, infection efficiency was relatively stable and the families varied from 0.1 to 1 relative to the highest spotting family, again a 10x difference. Others demonstrated that stomatal area and contact angle of water drops formed on secondary needles differed significantly in comparison of susceptible and resistant materials (Woo et al. 2001). Another study reported that stomata/row, stomatal shape, stomatal density, mean stomatal area, wax degradation, water-contact angle with wax and contact angle without wax all vary significantly in the same F<sub>2</sub> seed lot grown at three different nurseries (Woo et al. 2002). The surface water vs. basal resistance connection has been made by others (Poland et al. 2009). In summary, RNLf would seem to be a relatively stable trait that could influence amount of rust infection in various WWP families and perhaps in differing white pine species. However, the needle-trait studies and the large differences in level of rust-free seedlings in F<sub>1</sub> WWP grown and inoculated under varying conditions raise significant cautionary flags.

### **Needle Spots Only (NSO) and Partial Girdle (PG) Resistance**

Tests inoculated in 1964, 1968, and 1970 also investigated a resistance mechanism termed needle-spots-only, hypothesized to be a composite of two mechanisms called (1) premature-needle-shed (PNS), where infected needles were shed before the rust penetrated the short shoot (McDonald and Hoff 1971), and (2) fungicidal-short-shoot (FSS) where infection

failed to penetrate the short shoot (Hoff and McDonald 1971). In the 1964 test, 99.5 percent of the seedlings exhibited needle infections and only the composite mechanism was delineated on F<sub>1</sub> and susceptible stocks 2 years post infection. Of 2,878 F<sub>1</sub> seedlings, 19 percent expressed resistance, and of 345 open-pollinated control seedlings, 10 percent were classified resistant. Results for the 1968 inoculation were based on 546 open-pollinated controls, 2,876 F<sub>1</sub>s, and 3,061 F<sub>2</sub>s. Two new categories were added, rust-free (RF), seedlings without symptoms, and a bark-reaction labeled partial girdle (PG) wherein the cankers appeared to be cleared from stems and/or branches. Rust-Free seedlings were removed from the totals to calculate the remaining percentages in both tests. PNS equaled 15 percent (control), 20 (F<sub>1</sub>), and 48 percent (F<sub>2</sub>); FSS equaled 2 percent (control), 5 percent (F<sub>1</sub>), and 12 percent (F<sub>2</sub>); and PG equaled 2 percent (control), 8 percent (F<sub>1</sub>), and 7 percent (F<sub>2</sub>) (Hoff et al. 1973). The 1970 test (Hoff et al. 1980) included 18 species of white pine, but only F<sub>1</sub> WWP stock is included in this discussion. Results were 21 percent (PNS), 4 percent (FSS), and 11 percent (PG). In regard to PNS, FSS, and PG in the F<sub>1</sub> families, the three tests compare very favorably. We conclude that NSO was stable under the range of abiotic (and maybe biotic) conditions experienced under experimental conditions up to about 6 years of age, and that infection in F<sub>1</sub> stock should plateau at 65 percent (e.g. 1-PNS (20 percent) + FSS (5 percent) + PG (10 percent) = 65 percent). From these results, open-pollinated controls should plateau at about 80 percent and F<sub>2</sub> at 35 percent. Predictions (1968 test) were control = 81 percent, F<sub>1</sub> = 67 percent, and F<sub>2</sub> = 34 percent infection (Hoff et al. 1973).

### **Rust-Free Resistance (RF)**

Occasionally rust-free seedlings, which showed up in the large-scale inoculation tests, were treated as escapes at Moscow. On the other hand, given the possibility that induced stomatal closure can enhance basal resistance (Hou et al. 2009), the issue should be revisited. No information was found in the materials supporting this review pertaining to RF primary needles. In the 1968 test, RF equaled 4 percent (control), 1 percent (F<sub>1</sub>), and 13 percent (F<sub>2</sub>). In the 1970 test, RF equaled (24 percent) for F<sub>1</sub> stocks. Thus, RF in F<sub>1</sub> families increased dramatically (i.e., 1 percent to 24 percent), when the inoculation was conducted within the bounds of a greenhouse and lath house, while expression of other mechanisms changed little (see above).

## NSO in the Real World

Stocks developed from the northern Rocky Mountain WWP breeding program were placed in test plantations in addition to Merry Creek and Gletty Creek. Early stock representing  $F_1$  full-sib families from phenotypically resistant parents were planted with open-pollinated controls at Priest River and Deception Creek Experimental Forests in north Idaho (McDonald et al. 2004). After about 45 years of repeated assessments, disease progress curves showed that infection in  $F_1$  families reached a plateau of 40 percent at 25 years and remained stable for another 17 years at both sites. This performance was better than expected. The controls planted at the Experimental Forests also presented surprises. At 45 years, disease progress curves for both were still trending upward, but the expected infection plateaus (estimated by curve fitting) hint at a rapid increase in resistance phenotypes in the susceptible population. Open-pollinated seed was collected from the same cankered trees in 1953 and 1955 and used as control stock for the 1957 and 1959 plantings. Estimates of infection plateaus generated by curve fitting were 72 percent and 83 percent at Priest River and 77 percent and 89 percent at Deception Creek for the two collection years respectively. Both sites indicate a 10 percent gain in resistance from susceptible parents in two years and both plantings fit with nursery test expectations. Was this gain caused by changes in gene frequency (Modern Synthesis) or soft inheritance? After 26 years, at Gletty Creek (sister planting of Merry Creek), control,  $F_1$ , and  $F_2$  stocks, supported 94, 46, and 20 percent infection, respectively (Moscow FSL data on file). Since these trends appear to be following those at Priest River and Deception Creek, I conclude that Gletty Creek is meeting or exceeding expectation of resistance.

The northern Rockies stocks were also planted in California and British Columbia (BC). At the Happy Camp site in northern California, control,  $F_1$ , and  $F_2$  families were planted in the early 1970s and inspected periodically (Kinloch et al. 2008). Since none of these WWP materials express the major gene, this aspect of WPBR at Happy Camp will be ignored in this discussion. First, we must address the matter of expected resistance. As discussed above, individual control lots,  $F_1$  full-sib families, and  $F_2$  full-sib families can vary widely in expected levels of resistance as judged from inoculation tests of seedlings supporting only secondary needles. Ranges of variation in expected percent infected (i.e., percent infected = 1-

tested percent clean) observed in a limited data set from the 1966 test are: control (14 to 34 percent),  $F_1$  (5 to 49 percent), and  $F_2$  (58 to 83 percent) (Moscow FSL data on file). Since the Happy Camp data are presented as simple scatter plots with uneven time intervals, comparison to disease progress curves is complex. Thus, I will focus on the apparent infection asymptote demonstrated in the published scatter plots (Kinloch et al. 2008 p72). Control-lot asymptotes of 100 percent and 90 percent meet nursery expectation. Three  $F_1$  families show 70, 60, and 90 percent asymptotes and the expected range is 51 to 95 percent. Thus, the  $F_1$  families meet the nursery test expectation. Two  $F_2$  families reached asymptotes of 35 percent and 40 percent, which is within the expected range of 17 to 42 percent.

Idaho  $F_2$  stock and local controls were planted at 2 coastal British Columbia sites, one low elevation and one high elevation (Hunt and Meagher 1989). After 12 to 13 years exposure to WPBR, infection levels were, low-elevation control 52 percent, low-elevation  $F_2$  75 percent, high-elevation control 21 percent, and high-elevation  $F_2$  10 percent. After 20 years of exposure at an interior BC site,  $F_2$  stock was 35 percent infected while controls were 100 percent infected (Hunt 2005). Test plantings of  $F_1$  and  $F_2$  stock in California (1 site), Idaho (3 sites), interior BC (1 site), and high elevation coastal BC (1 site) have performed to expectation or better. Two sites, low-elevation coastal BC and north Idaho, failed to meet expectation. Some production plantations of  $F_2$  stock have also exhibited higher than expected levels of infection (Schwandt and Ferguson 2003). In light of the expanded Modern Synthesis we can hypothesize these failures are triggered by the environment. Also, Hunt (2004) discusses the effects of environment on expression of blister rust resistance and, although numbers are small, indicates that environmental factors may be implicated of the failure of  $F_2$  NSO resistance. Hunt (2005) compared WWP seedlings and grafts and low and high elevation coastal sites and concluded that genetic x environment interactions influenced expression of resistance.

Direct influence of environment on RNLF expression comes from a couple of "accidental experiments" reported by Woo et al. (2004). Two seed lots grown at two different nurseries inoculated in a common inoculation facility and then returned for 3 years of development to their original nurseries. One lot was expected to express a relatively high level of

resistance and the other a low level. Mortality in the resistant stock was 48 percent 3 years post infection, while the susceptible stock exhibited 30 percent mortality. This prompted an experiment designed to control for genetics and environment. Bulk  $F_2$  seedlings were grown at two different nurseries prior to infection and were scheduled to be inoculated in September of 1999 at a common facility. Seedlings from one nursery were placed in cold storage in December of 1998. However, due to an oversight, the seedlings were not removed from cold storage until early August of 1999. Meanwhile, the other lot was subjected to a normal cycle. At 5 months post infection, seedlings exposed to extended cold storage, (immature secondary needles) exhibited 100x the infection efficiency of the mature needles – a level indicating low RNLF in  $F_2$  stocks. The overall conclusion is that the failure of resistance at Merry Creek was probably due to an environmentally triggered collapse of basal resistance. It seems WPBR basal resistance is a classic example of a “plastic immunity response” as described for the concept of ecological immunity (Sadd and Schmid-Hempel 2008).

#### **White Pine Blister Rust Phytohormone Interaction**

Materials from the 1966 progeny test were also used to investigate the interaction of WPBR infection, rooting medium, and hormone application on rooting of WWP (McDonald and Hoff 1970a). Single-needle fascicles were harvested from each of 12 individual outdoor-grown seedlings from each of 104 WPBR-resistant families in early March at 5 months post inoculation. Needle infections were not visible at harvest; however, 99.5 percent of the seedlings exhibited needle spots by early June, indicating that the uninfected needles were most likely derived from infected seedlings. WPBR infection depressed rooting by 40 percent in half-sib family 17. Tester 17 was later shown to segregate in selfed families (McDonald and Hoff 1971) as a trait controlled by two recessive genes. Meanwhile, the remaining half-sib families (19, 22, and 58) exhibited depressed rooting by 11, 9, and 19 percent, respectively. When selfed, these families segregated in a single recessive pattern. From the perspective of an expanded Modern Synthesis, these results probably indicate a connection between WPBR infection and hormone metabolism. On the other hand, this genetic hypothesis has been questioned (Kinloch et al. 2008; Hunt 2004). In fact, given current understanding of transcriptome behavior associated with basal resistance, I also question this genetic hypothesis. Further, it must be said that disproving a

genetic hypothesis about a trait does not negate the reality of the phenotype. So, what does depression of rooting by WPBR needle lesions tell us about how ecological immunology functions in WPBR? Since the rooting depression seems not to be expressed in healthy needles obtained from plants supporting a needle infection, systemic signaling may not occur until penetration of cell types located in the stem. But, how would this observation fit with the possibility of needle endophytes priming basal resistance as discussed above. Significant interactions among the WPBR fungus and naturally occurring endophytes appears to be an important aspect of blister rust biology (Ganley et al. 2008). Many root zone and stem pathogens and symbionts could also be present and functioning with the aid of a systemic signaling system.

#### **Some Additional Questions**

The demonstrated existence and importance of the epigenome allows us to re-examine persistent questions that are associated with WPBR studies. These questions include the following: Is the common occurrence of overdispersion (maximum infection incidence often fails to reach unity) in WPBR epidemics (McDonald et al. 2005) related to the abiotic environment, induced immunity, endophytes, soil conditions, or some combination of these factors? Why did a high-severity burn during site-preparation at Merry Creek result in equal infection rate increases in control,  $F_1$ , and  $F_2$  test populations relative to a lower severity burn (McDonald and Decker-Robertson 1998)? Can burn treatments influence host resistance in a manner similar to biochar, which has been shown to induce resistance to both biotrophic and necrotrophic pathogens on both tomato and peppers (Elad et al. 2010)? In a general sense, is an appropriate local natural control population required to insure reliable results from any forest management experiment? Given our current understanding of plant immunity, does the existence of major-gene resistance in the pine host imply a significant coevolutionary history between white pines and *Cronartium ribicola*? Would a reexamination of historic data from a new perspective reveal new awareness about WPBR interactions? Regarding ongoing screening and breeding programs of white pines, how confident can we be that subpopulation structures of pine have not “evolved” rapidly enough by way of ecological speciation to ultimately cause significant host genotype x pathogen genotype x environment interactions? Does awareness of many new alternate

hosts (Mulvey and Hansen 2011, Kattera and Hiltunen 2010) indicate pine genotype x Ribes genotype x Pedicularis genotype x Castilleja genotype x rust genotype interactions should be expected? Do such complex host-pathogen-environment interactions reflect a mature pathosystem? Do implications arising from soft inheritance indicate that more emphasis should be placed on natural regeneration of conifer forests? Are there serious negative consequences to developing resistant host populations through creation of artificial hybrids screened for resistance under artificial conditions that do not reflect the conditions where the resulting populations are deployed? Results from an initial range-wide study of AFLP molecular markers in WWP (Kim et al. 2011) and analysis of host growth and blister rust expression in a common garden experiment (McDonald ms in progress) together illustrate a potentially complex and interactive WWP population substructure.

### **Summary - A Management Dilemma**

The expanded Modern Synthesis poses management dilemmas, such as (1) selection and deployment of natural quantitative or forced qualitative resistance, (2) deployment of potentially disruptive stock from a geographically broad breeding program vs. prudent local management of natural populations, and (3) utilization of large economically efficient screening facilities vs. sophisticated screening designed to yield stock that fits subpopulation boundaries. Basal resistance, while sometimes subject to environmentally triggered breakdown, might still be the best choice as indicated by its major role in dampening pest activity in natural forests. This observation fits with “optimal immune defense”, an important concept of ecological immunity. Qualitative resistance, notoriously susceptible to virulence variation, may be a poor option for forest trees judging from its relatively minor role in forest systems and, according to the “usage costs of defense” principle of ecological immunity, this option would likely incur higher fitness costs than basal resistance. Increasing knowledge about ecological speciation and the complexities of genome x epigenome x environment interactions indicate local management of local populations may result in more stable forest ecosystems. Further, the biological paradigm encompassed in a new Synthesis argues that large screening facilities will most likely produce maladapted populations due to the sheer complexity of controlling all the important sources of variation. Approaches discussed will also apply to

understanding and managing the heightened pest problems expected with climate change (Grulke 2011).

Will the new Synthesis apply to our target populations? A good foundation for molecular investigations has been developed and was recently reviewed (Richardson et al. 2010) from the perspective of classic Modern Synthesis. Some important factors associated with the new paradigm that have been observed are: microRNAs in lodgepole pine (Morin et al. 2008); small RNAs in Norway spruce, white spruce, eastern white pine, and Douglas- fir (Dolgosheina et al. 2008); and microRNAs and epigenetic inheritance were associated with climate adaptation of Norway spruce (Yakovlev et al. 2010). As already mentioned, microRNAs were shown to be regulated by fusiform rust infection in loblolly pine (Lu et al. 2007). The CC-NBS-LRR subfamily of proteins, known to be associated with plant immunity, was shown to be active in WWP with possible links to qualitative resistance (Liu and Ekramoddoullah 2007). Eighty-three members of the WRKY family of transcription factors, associated with plant immunity, were found in WWP and one member was linked to a major resistance gene (Liu and Ekramoddoullah 2009). Another examination of signaling and pathogenesis-related proteins in WWP revealed that levels of a pathogen-related protein were increased by WPBR infection, wounding, and methyl jasmonate application (Ekramoddoullah et al. 2006).

Limitations imposed by the gene-centric Modern Synthesis have significantly constrained depth of analysis of WPBR evolution and ecology in particular and forest biology in general. A major weakness is lack of awareness regarding the impact of interaction between genotype, development, and soft inheritance. A theory incorporating epigenomes, gene regulatory networks, soft inheritance, ecological immunity, and ecological speciation should be embraced. As suggested above, an expanded Modern Synthesis can reveal management implications of large effect. I propose using the new paradigm to: (1) conduct a thorough review of current breeding and forest management programs (e.g., Schoettle and Sniezko 2007; King et al. 2010); (2) review existing literature using a brighter light; (3) re-evaluate remaining data archives to achieve improved insight; (4) develop new study designs; and then (5) initiate pilot studies in conifers, while awaiting sequencing of a conifer

genome, directed at transcriptome discovery and function using 3<sup>rd</sup> generation single-strand sequencing (see Morozova et al. 2009; Mamanova et al. 2010). In conclusion, new theory, a 100-year legacy of experimentation and observation, powerful new tools, and a substantial need for new knowledge all argue for rapid advance toward a more comprehensive understanding of WPBR coevolution. The goal, of course, is sustainable management of conifer ecosystems worldwide.

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# Whitebark Pine - Threatened and Endangered Species Status

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## ABSTRACT

This report briefly describes the current status of listing whitebark pine as a threatened and endangered species in the United States and Canada. This includes brief summaries of the procedures being followed by various agencies in both countries. Even though the timelines specified in various regulations are not being met, the process is continuing and we may have some decisions by the fall of 2011.

## INTRODUCTION

Whitebark pine is a keystone species of high elevation ecosystems throughout western North America. Most of the whitebark pine occurs in remote roadless areas, often in wilderness, or national parks where 300- to 500-year-old gnarled relics define high elevation vistas and provide much of the character of the alpine experience (Tomback and others, 2001). Recent surveys across its range have found dramatic declines in whitebark pine due to a combination of insect, disease, species competition, fire, and climate change (Schwandt 2006). The inadvertent introduction of white pine blister rust from Europe in 1910 has now spread to these fragile whitebark pine ecosystems where it has dramatically disrupted natural regeneration (Tomback and Achuff 2010). Many areas that once were dominated by whitebark pine now only have remnant individuals, and populations on isolated mountain tops are winking out and not likely to be restored without human intervention. The urgency of the situation has recently increased due to outbreaks of mountain pine beetle which may kill trees that harbor natural resistance to blister rust (Kegley and others, in press).

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In: Fairweather, M. Comp. 2011. Proceedings of the 58th Annual Western International Forest Disease Work Conference; 2010 October 4-8; Valemount, BC. US Forest Service, AZ Zone Forest Health, Flagstaff, AZ.

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As a result of these concerns about whitebark pine, it has been the subject of considerable interest in listing it as an endangered species by a number of groups and agencies. This includes a global listing by the International Union for Conservation of Nature as vulnerable (at high risk of extinction in the wild in the medium-term future). The following is a brief summary of the efforts to list whitebark pine as an endangered species in both Canada and the United States.

### Current Status in the United States

The Endangered Species Act (ESA) of 1973 provides protection for species at risk of extinction or those that may become endangered in the near future. In 2007, the Western Washington Fish and Wildlife Office of the U.S. Fish and Wildlife Service (FWS) listed whitebark pine as a "species of concern" (WWFWO 2007). On December 8, 2008 the Resource Defense Council (RDC), officially petitioned FWS to provide this species with threatened and endangered (TandE) status (NRDC 2008).

The ESA guidelines for these requests give the FWS 90 days to determine if the request is supported by scientific evidence and that a listing request has merit for further investigation. However, there are no consequences for missing this 90 day response time and the FWS felt it did not have the resources necessary to make this determination so only limited action was taken. Finally, the RDC filed a lawsuit in the spring of 2010 to force the FWS into action. On July 20, 2010 the FWS announced that the petition did have merit and that the listing of whitebark pine "may be warranted".

This announcement is the next step in the process which gives the FWS one year to collect and analyze scientific data in order to make the next decision which has three possible responses:

It can decide that the data does not support the listing, and this petition process would be finished.

It can decide that the data supports listing but other species are a higher priority for listing (termed “Warranted but precluded”). In this case the species is considered as TandE but no rules are proposed, and an annual review is required until it can be formally listed.

It can decide that listing the species is warranted and develop proposed rules for its management that are published in the Federal Register. Once this is published, there is a 60 day period for public comments and peer review including input regarding the potential economic and social impacts of listing.

At the end of the 60 day public review period, the FWS makes a final decision regarding its listing. This may result in a decision not to list whitebark pine as a TandE species, or if the final decision is made to list whitebark pine, it must then publish a final ruling in the Federal Register and the species would be added to the TandE species list (effective 30 days after the rule is published). The total estimated time frame could be 4-6 years from petition to recovery plan finalization.

Even without the TandE status, there are critical administrative issues that still need to be resolved. For example, over 90 per cent of the whitebark pine population in the US is on federally managed lands (USFS, National Parks, BLM, BIA), and much of this is in designated wilderness areas where management activities are under very close scrutiny. Planting might only be considered if it could be with locally collected seed, but caging of cones may not be permitted since cages would be “alien” to a wilderness.

### **Current Status in Canada**

Listing of whitebark pine in Canada is further along than in the United States. In Alberta, whitebark pine has been listed by the Minister of Sustainable Resource Development as Endangered under the Alberta Wildlife Act (COSEWIC 2010). This currently provides no legal protection but measures have been taken to ensure that, outside of protected areas, it is not inadvertently harvested and that planning for harvesting, fire management, and mountain pine beetle management takes it into account.

In British Columbia in 2008 the Ministry of Environment’s Conservation Data Center adjusted the conservation ranking of whitebark pine, adding it to the blue-list, a list of species of special conservation concern in British Columbia (Campbell 2008). Whitebark pine was uplisted due to a “severe negative long-term trend expected from mountain pine beetle infections, the white pine blister rust epidemics, climatic warming trends, and successional

replacement”. This designation provides no legal protection and whitebark pine has been harvested in some areas, although the extent is not clear. However, British Columbia government agencies have suggested voluntary conservation measures. About 26 percent of whitebark pine range in British Columbia occurs in protected areas.

Canada’s scientific advisory group on endangered species, COSEWIC, (Committee on the Status of Endangered Wildlife in Canada) makes recommendations to Canada’s government regarding which species should be added to the Country’s Species At Risk Act (SARA) – the Canadian equivalent of the Endangered Species Act in the United States.

In April 2010, (COSEWIC) recommended whitebark pine be added to Canada’s Species At Risk Act. Their finding included the following statement:

“This long-lived, five-needled pine is restricted in Canada to high elevations in the mountains of British Columbia and Alberta. White Pine Blister Rust alone is projected to cause a decline of more than 50 percent over a 100 year time period. The effects of Mountain Pine Beetle, climate change, and fire exclusion will increase the decline rate further. Likely, none of the causes of decline can be reversed. The lack of potential for rescue effect, life history traits such as delayed age at maturity, low dispersal rate, and reliance on dispersal agents all contribute to placing this species at high risk of extirpation in Canada.”

The COSEWIC decision goes to Minister of Environment recommending the listing of whitebark pine. The minister then prepares a response after taking several months for review, consultations, and analysis of socio-economic impacts of listing. In the spring of 2011, the review and consultations will end and the Minister is expected to make his recommendation to the Governor in council (GIC) which is the Canadian federal cabinet. Next fall it is expected that the GIC will take one of the following actions:

- Accept COSEWIC assessment and add whitebark pine to the TandE list
- Decide not to list whitebark pine as a TandE species
- Refer the issue back to COSEWIC for more information or clarification
- If no action is taken for 9 months, the species is added to the TandE list

## Implications of Listing

If listed, whitebark pine would be the plant with the largest range by far to be listed. There would certainly be additional attention to this species, but whether that would translate into additional resources for restoration activities is not a certainty. If listed, critical habitat would be designated, and a recovery plan would be required. A recovery plan could be based largely on existing restoration plans and hopefully additional resources could be found to conduct needed research into different approaches to dealing with the threats of blister rust and mountain pine beetle, which could help slow the pines' decline and protect refuge areas that have yet to be affected.

There are major unresolved issues about the impacts the listing might have on current restoration activities. For example, it is not clear if all restoration activities would be halted until a recovery plan is developed. There may be a mandatory FWS review (consultation) on all restoration activities which could slow down the implementation of restoration activities. In addition, actions like prescribed fire to reduce competing vegetation and enhance natural regeneration might not be allowed (or might create lengthy litigation) if some existing whitebark were burned in the process.

Even though the final decisions regarding listing in both the US and Canada have not been made, with mortality outpacing the ability of whitebark pine forests to self-replenish, the loss of these distinctive timberline communities will continue. As the keystone whitebark pine dwindles, we expect the web of dependent living organisms to suffer.

Although restoration plans and projects are being implemented in both countries, addressing this range-wide problem will take a concerted effort for a long time regardless of its listing. However, if we can develop, disseminate, and maintain blister rust resistant whitebark pine populations across its natural range, natural restoration processes may eventually be resumed. Whether listing WBP as a TandE species helps or hinders this process remains to be seen.

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## Special Papers: Session II







## Influence of Nursery and Stocktype on Incidence of White Pine Blister Rust

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### ABSTRACT

White pine blister rust (*Cronartium ribicola*) is such a frequent mortality agent of five-needle pines that the threat of this disease has drastically reduced the planting of western white pine in coastal British Columbia (BC). To determine if the choice of seed source or cultural factors could be used to lower the risk of infection, we designed a trial to examine four variables – seed source (seedlot), stock type, nursery source and the application of an early low-branch pruning treatment. Five seed sources were used, three from putatively resistant parent trees (one canker-free and two slow canker growths) from Texada Island, BC, and the other two wild-collected control lots. Seedlings from these five seedlots were raised as either of two stock types - one-year plug stock transplanted for a second year into bare soil or as two-year plug stock - and all were raised at two different nurseries, one in a high-hazard rust area and the other in a low hazard rust area. Five years after planting half of the subject trees were pruned to reduce the risk of developing rust infections. This design provided 20 different treatment combinations of the four variables that could be tested. The trial was halted prematurely after nine years due to sale and destruction of the test site. Analysis of the data revealed that pruned trees had less infection but that the advantage was small (5 percent) four years post-treatment. Interactive effects complicated interpretation of the impact of the remaining three variables but it was clear that the canker-free seedlot was superior to both control seedlots. Another result suggests that bare root stock grown at the interior nursery may be less prone to rust than the other combinations but the evidence is not definitive.

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### INTRODUCTION

For decades researchers have searched for attributes of tree physiology, culture and tending that may provide clues as to why some families of western white pine are more susceptible to white pine blister rust (WPBR) than others. Attributes such as the amount and distribution of foliage, internode length, proximity of first internode to soil surface, and number of stem needles have all been measured in speculation that differences may explain infection rates, especially in young trees. Some of these differences can be created culturally in the way planting stock is raised. For example, nursery-grown Styroblock (plug) raised trees are usually of larger diameter and denser of lush foliage due to fertilization in their greenhouse environment than field-grown, bare root seedlings.

Various mechanisms of natural resistance have been documented in the western white pine population and the presence of these putatively resistant traits have formed the basis of tree selection and breeding efforts for a generation (Hunt 1997, King et al. 2010). Texada Island, near Powell River, BC, is an area with abundant *Ribes* spp. (the alternate host for *C. ribicola*) and, subsequently, a very high rust infection pressure. Over the years numerous individual western white pine on the island that exhibit resistance responses like slow canker growth, bark reaction, or a complete lack of stem infections have been identified and used as sources of seed for reforestation. This seed, while considered better than open-pollinated, wild-collected seed, still requires some operational field testing to quantify how much better it is than wild seed in lowering the infection and mortality rates caused by WPBR.

Additionally, the pruning of lower branches of young white pines has long been considered of benefit in reducing the incidence of WPBR regardless of any inherent resistance that the tree may or may not have (Hungerford et al. 1982, Hagle and Grasham 1988, Hunt 1991, 1998).

This trial was conducted to test if genetic factors like putative resistance mechanisms to WPBR or cultural factors like the type of stock used or the nursery that produced the seedlings had any bearing on the incidence of WPBR on trees from various commonly used wild and putatively resistant seed sources.

## **MATERIALS AND METHODS**

The trial was designed as a test of five seedlots raised as two different stock types at two separate nurseries for a total of 20 different treatment combinations. The five seedlots consisted of open-pollinated, bulk collected seed from individual parents that exhibited desirable characteristics. Three of the seedlots (31751, 31755 and 31756) were from parent trees located on Texada Island, BC where a high percentage of putatively resistant trees are found. Seedlot 31751 consisted of trees raised from canker-free parents while the other two seedlots were from trees that were stem cankered. These cankers though are so-called “slow cankers” because they develop gradually and the tree usually keeps pace or expands in diameter faster than the canker, thus staying alive much longer than normal (Hunt 1997). The two remaining seedlots were commercial bulk collections from natural stands near Honeymoon Bay on Vancouver Island (13881) and from the interior (3727) of BC near Lillooet and were used as controls.

The two stock types used were PBR 313B 1+1 (stock raised one year in a greenhouse in 160 hole Styrobloc containers and then transplanted into a bare field for an additional year) and PSB 415B 2+0 (stock raised for two years in greenhouse 112 hole Styroblocs with plug length of 15 cm). The two nurseries selected were a Ministry nursery located on the coastal mainland at Surrey, BC and the privately-run KandC Nursery located in Oliver, BC in the Okanagan Valley. The Surrey Nursery is in a rust-prone area with *Ribes* present nearby while the KandC Nursery is in an area with no known rust present for at least 30 km.

For our trial, the two-year old trees were planted in March 1995 in a bare, non-irrigated field at the Surrey Nursery. Each of the 20 treatment combinations was represented in a plot by one randomly ordered row of 75 trees for a total of 1500 trees per plot. A second identical plot was installed immediately adjacent to the first with a different random order of the 20 rows (treatments). Starting in the spring of 1996, the trees

were assessed annually for the occurrence of WPBR stem and branch cankers. Mortality due to WPBR or other factors was recorded as well as the occurrence of any abiotic or other pest damage. Tree height and diameter were to be measured every 10 years. In 1999, after five growing seasons, all the live trees in one plot were pruned using the guideline of the day for low branch pruning for prevention of WPBR (BC Ministry of Forests 1996). The trial was ended prematurely after nine years when the nursery site was sold and the trees destroyed.

Statistical analysis on how the four categorical variables (seedlot, stock type, nursery and pruning) affect the proportion of WPBR infected trees per row was done mainly with logistic regression analysis using the GENMOD procedure of SAS (SAS Institute Inc. 2004). The significance of variables was evaluated and compared using likelihood ratio (chi-square) tests and least square means (i.e., estimated probabilities of infection) for each level of variable. Analysis was slightly complicated by tree mortality that occurred outside of that caused by WPBR. Our initial balanced design was upset by tree losses to lawn mowing, the improper application of herbicide during bud flush, and the antics of a drunk driver who mowed down several saplings one evening while joy-riding through the nursery grounds. These events combined to exclude just over 10 percent of our study trees.

## **RESULTS AND DISCUSSION**

Regression testing of the variables seedlot, stock type, nursery and pruning, and the 40 permutations of those variables, led to all but six comparisons being deemed statistically insignificant (table 1). All of the main effects are significant plus the interactive effects of stock type\*nursery and seedlot\*stock type\*nursery. While all are significant, the presence of interactive effects involving all the variables, except for pruning, indicates that statistically we may only draw definitive conclusions from analysis using the pruning variable.

Examining the pruning results more closely, we found that even with only four growing seasons for the treatment to take effect that there is a significant difference in the incidence of WPBR between the pruned and unpruned trees. Only 24.9 percent of the pruned trees were infected versus 29.7 percent of those not pruned. The statistical significance of this relatively small difference illustrates the benefit of having a large sample from which to draw and the

power that it provides in being able to validate small, but still statistically meaningful, differences in our data. This is a recurrent theme in our analysis.

However, in practice, a difference of about 5 percent incidence is likely not a compelling argument for expending funds to conduct pruning as a preventive treatment. At this point in the trees life, the costs would still likely outweigh the potential benefits. The short post-treatment time frame also limits a meaningful comparison of what pruning means in terms of lower tree mortality, not just infection incidence. Our result does agree with the findings of numerous other studies that examined the efficacy of pruning as a treatment for WPBR (Hungerford et al. 1982, Hagle and Grasham 1988, Hunt 1991, 1998).

The presence of significant interactive effects limits our ability to make definitive statements about the relative merit of seedlot, stock type or nursery but there were some interesting differences within these variables. There were significant differences in the infection rates between the seedlots. Quite clearly, the interior seedlot from Lillooet (3727) had the highest rust incidence after nine years with almost a third of all trees infected (figure 1). The least infected trees were from the canker-free Texada Island seedlot (31751), where fewer than 20 percent of the trees were infected, and from one of the slow canker growth

seedlots (31755) also from Texada Island. The incidence of WPBR for these two seedlots was significantly less than for the Lillooet seedlot. Over time one would expect that the putatively resistant trees would have less WPBR but it is uncertain why one slow canker growth seedlot shows lower incidence than the other, since they were both collected from similar trees in the same area.

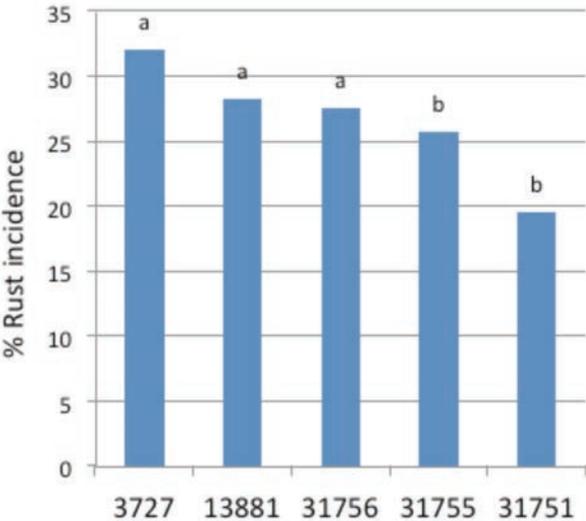
It should be noted that tree mortality due to WPBR does not follow the same pattern as infection incidence although the canker-free Texada seedlot has experienced the least mortality to date (2.5 percent) and the Lillooet seedlot the most (4.1 percent). However, since mortality is just starting to accelerate at this age, it is premature to base any conclusions on this evidence.

There was a significant difference in WPBR incidence between the two stock types with the bare root stock (PBR 313B) having 3.7 percent lower rust incidence than the plug (PSB 415B) stock. Similarly, the trees raised at the interior KandC nursery had 3.2 percent less rust than those raised at the coastal Surrey Nursery. In both cases these differences were statistically significant but, with the influence of the interactive effects, the differences may not be meaningful enough to advocate for a change in the current choice of either stock type or nursery.

**Table 1**—Comparison testing of all categorical variables using logistic regression. Tests of non-significant interactions are not shown.

Source	Df	Chi-Square	Pr > ChiSq
Seedlot	4	27.57	<0.0001
Stock type	1	8.26	0.0041
Nursery	1	6.58	0.0103
Pruned	1	8.97	0.0027
Stock type*Nursery	1	5.27	0.0217
Seedlot*Stock type*Nursery	4	18.77	0.0009

In order to see if any definitive recommendations could be extracted from our results, despite the premature conclusion of the trial and obscurity caused by the interactive effects, we created a table to explore what combination of variables might be most favorable even with the uncertainty in our data. The individual seedlots were compared with the stock type and source nurseries and tested to see which variables, or combinations of variables, was significant. The results are presented in Table 2.



**Figure 1**—Percent incidence of white pine blister rust on five seedlots of western white pine after nine years. Bars with the same letters are not statistically different.

**Table 2**—Comparison of interactive effects on the categorical variables seedlot, stocktype and nursery using least square means estimates on the logit scale, so a value of zero represents an estimated probability (of infection) of 0.5, negative values represent small (< 0.5) estimated probabilities and positive values represent large (> 0.5) estimated probabilities. The best result (most negative value) for each seedlot is bolded while the worst is italicised. The shaded seedlots produced no statistically significant results so they make no contribution to our conclusions although they are provided for completeness.

Seedlot	PBR 313B KandC	PBR 313B Surrey	PSB 415B KandC	PSB 415B Surrey	Significant?
3727	-1.3591	-0.6614	-0.6174	-0.4193	Stock type, Nursery
13881	-1.5315	-0.7386	-0.7591	-0.8085	Stock type*Nursery
31756	-0.8675	-1.0704	-1.165	-0.7783	No
31755	-1.0598	-1.3055	-0.9217	-0.9704	No
31751	-2.2142	-0.9262	-1.2104	-1.5851	Nursery, Stock type*Nursery

Illustrated in this manner, it appears as if bare root stock raised at the KandC Nursery produced, in three of five cases, the least WPBR-infected trees after nine years of testing. If the two shaded rows are excluded, as they did not produce a statistically significant result for any permutation tested, this result is even more dramatic. Interestingly, the significance of the superior stock type and nursery holds true for the two poorest performing, in terms of WPBR incidence, seedlots (the controls) as well as the best (the canker-free) seedlot.

Conversely, the poorest results seem to be from the Surrey Nursery where, in two of three cases the bare root stock performed the worst and in the remaining case it was the plug stock. Although it cannot be stated with certainty, it appears as if stock raised in the relatively WPBR-free southern interior of BC may perform better even if it is to be used on the coast.

With the exception of the positive result that pruning gave in lowering the incidence of WPBR in treated trees, we fully acknowledge that any conclusions involving the variables of seedlot, stock type and nursery are very preliminary and not definitive. We wonder though if they might point to the promise of what the results could have been had the trial been allowed to reach its natural conclusion. We hope that others may pursue this line of study to a more fruitful end.

## ACKNOWLEDGEMENTS

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## Genetic Characterization of *Phellinus sulphurascens* in Western North America and Eurasia

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*Phellinus sulphurascens* Pilát (syn. = *P. weirii*) is a facultative parasite that causes laminated root rot (LRR) of several commercially important coniferous species. LRR spreads in infected stands primarily by way of vegetative mycelia transferring at points of contact between healthy and infected roots. Sporophores of *P. sulphurascens* form rarely in nature and spores appear to be unimportant in initiating new infections. To better manage LRR we need to better understand the population structure of *P. sulphurascens*. We carried out a comparative genetic diversity study among *P. sulphurascens* isolates from Western North America (Canada and USA) and Eurasia (Siberia and Japan) to investigate the population genetic structure relative to the origin of this species (i.e., introduced vs. native).

By analyzing the small and large mitochondrial ribosomal RNA subunit genes (mtSSU rRNA and mtLSU rRNA) and the following six nuclear loci, internal transcribed spacer region (ITS), actin (ACT2), RNA polymerase II largest subunit (RPB1), RNA polymerase II second largest subunit (RPB2), putative laccase (LAC-like) and translation elongation factor 1-alpha (TEF1), we observed that none of the alleles was shared between Western North American (WNA) and Eurasian *P. sulphurascens* populations. In total, 55 multilocus genotypes (MLGs) were retrieved in these two continental regions. Of these, 41 MLGs were observed among 56 isolates sampled at widespread locations in British Columbia

(Canada) and North-Western USA, and 14 MLGs were observed among 16 isolates sampled in Siberia and Japan. We compared molecular diversity parameters at nuclear loci and found that there were more segregating sites, higher nucleotide diversity, and either equal or higher number of haplotypes among isolates from Eurasia compared to WNA, despite the lower number of Eurasian isolates analysed. Haplotype networks showed that for RPB1, RPB2 and LAC-like loci, alleles associated with WNA isolates were always grouped. Phylogenetic relationship among the *P. sulphurascens* isolates was estimated from the allelic variation found in the mtSSU rRNA, mtLSU rRNA, ACT2, RPB1, RPB2, LAC-like and TEF1 loci. As expected, the combination of these seven loci for reconstructing the phylogenetic network showed that WNA isolates clustered together.

In summary, all these results showed that *P. sulphurascens* in Eurasia is highly diverse (which may indicate that this region could be the centre of origin of this fungus), and that gene flow appears to be restricted between these two continental regions.



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## Some Historical Perspectives on Pest Damage to Sugar Pine in Southwest Oregon

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Sugar pine (*Pinus lambertiana*) has very important scenic, wildlife, and watershed values and is also a high quality timber species. It possesses a number of virtues including frost hardiness, great growth potential, ability to reach substantial ages, capacity to survive and grow on infertile soils, a high level of resistance to root disease pathogens, and superior wind firmness. In southwest Oregon, sugar pines are widely distributed in suitable mixed conifer stands and currently comprise about 5 percent of the stocking in many mid-elevation forests. Older sugar pines are frequently the largest trees in these forests.

While sugar pine is a highly desirable species for management in southwest Oregon, it faces some serious health threats. As with other five-needle pines throughout the West, sugar pines are affected by the extremely virulent, non-native fungal pathogen, *Cronartium ribicola*, cause of white pine blister rust. They also may experience high levels of infestation by bark beetles, especially mountain pine beetle (*Dendroctonus ponderosae*). This is particularly true in dense stands where stress due to competition with many other trees can greatly decrease sugar pine vitality.

Recent evaluations based on single time surveys show disturbingly high insect- and disease-caused mortality levels for sugar pines in southwest Oregon (Goheen and Goheen unpublished). Have sugar pines been suffering similar high levels of mortality for many years in southwest Oregon? Were sugar pines more widely distributed and substantially more plentiful in the past? We don't really have answers for these questions. Quantitative data on the long term impacts of diseases and insects on sugar pines in southwest Oregon or on the historic demographics of the species in the area are mostly lacking. In this paper, we report some intriguing observations concerning the

magnitude of declines in the occurrence and numbers of sugar pines in two individual southwest Oregon sites for which some historical information was available.

### Panther Mountain

Panther Mountain near Port Orford in Curry County, Oregon was among the first locations where white pine blister rust was reported on sugar pine (Bedwell 1936). In 1937, a survey was done of this site and results were published (Mielke 1938). A 30 acre area on the east slope of Panther Mountain just below the summit was examined. The area was described as steep, rocky, brushy, and rather open with a moderate stocking of sugar pine and a few young Douglas-firs (*Pseudotsuga menziesii*). One hundred and twelve sugar pines with 6 to 30 inches dbh were examined and all were found to show *C. ribicola* infections. The oldest infection was back-dated to 1926. *Ribes cruentum* was found and reported to have a limited distribution near the top of the mountain.

In 2008, we revisited the Panther Mountain site. We did a systematic 19 point stand examination on what we judged was the same 30 acre area on the east slope of the mountain as had been surveyed 71 years previously. We agreed with the original surveyors that the area was indeed steep, rocky, and brushy, but found the following changes:

1. The stand on the site is now dense (average basal area is 250 ft<sup>2</sup>/acre) and dominated by Douglas-firs ranging from 24 to 36 inches dbh. There is also a significant understory of hardwoods, especially tanoaks (*Lithocarpus densiflorus*) and canyon live oaks (*Quercus chrysolepis*).
2. No living sugar pines whatsoever were encountered in our survey, and we only found one large snag of the species. We also examined surrounding stands to a distance of two miles and found no other living or dead sugar pines.

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3. *Ribes* species still occur in and around the Panther Mountain site and some exhibit *C. ribicola* infection. We identified *R. cruentum* and *R. sanguineum*, the latter with *C. ribicola*.

### Mill Creek Plot

In 1952, just after the surrounding area had received a major *Ribes* eradication treatment, a one acre monitoring plot was established at Mill Creek near Prospect in Jackson County, Oregon. At the time of establishment, it contained 460 live sugar pines that were “well distributed in size and age classes” and made up most of the stocking in the stand. The plot was revisited and surveyed in 1954 (Showalter, Fullmer, Watsom, and Miller unpublished). At that time it was found that: 1) There were no *Ribes* plants in the plot; 2) There were 422 live sugar pines, 78 of which had identifiable white pine blister rust infections; 3) The 38 sugar pines that had died between 1952 and 1954 died of “natural causes” (specifically mentioned were mountain pine beetle infestation and *Armillaria* root disease).

We relocated and did a 100 percent resurvey of the Mill Creek plot in 2009, 55 years after the previous examination. We found no *Ribes* in the plot. Average stand basal area was 240 ft<sup>2</sup>/acre, it was now a mixed stand that contained considerable numbers of Douglas-firs, incense cedars (*Calocedrus decurrens*), and ponderosa pines (*Pinus ponderosae*), and the plot contained 130 live sugar pines, of which one was 52 inches dbh, five were in the 10 to 30 inch dbh classes, four were saplings and 119 were seedlings. Most of the small trees appeared to be in poor condition due to overtopping by Douglas-firs and incense cedars. Only three living sugar pines in the plot exhibited identifiable white pine blister rust infections. Fourteen dead sugar pines between 6 and 20 inches dbh had obviously been infested by mountain pine beetle based on the occurrence of characteristic galleries (one also exhibited a probable blister rust canker and one showed evidence of *Armillaria* root disease as well as beetle galleries). An additional 10 dead trees though still identifiable as sugar pines were too deteriorated to accurately determine the causes of death. Evidence of any other sugar pines that died since the original survey was non-existent.

## DISCUSSION

We provide current data on two southwest Oregon stands whose sugar pine components were reevaluated after many years. Both exhibited dramatic declines in numbers of live sugar pines since the time of original survey. The Panther Mountain site showed essentially 100 percent elimination of the species in a 71 year period and the Mill Creek site showed a 70 percent reduction in number of live sugar pines and a shift to mostly seedlings in its sugar pine component over a 55 year period. We found it particularly interesting that many of the sugar pine trees that must have died in the periods between surveys at both sites were no longer detectable in current surveys, being so thoroughly decayed that they were either no longer identifiable as sugar pines or essentially gone.

We make no claim that the two examples we evaluated are typical or representative of large numbers of sites in southwest Oregon. However, they do indicate that substantial sugar pine declines are possible, at least on some sites, as a result of the combined effects of white pine blister rust, mountain pine beetle infestation, fire exclusion, and burgeoning stand densities. High grade logging, often cited as a major contributor to sugar pine decline, was not a factor in either of the cases that we examined.

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## Comandra Blister Rust Resistance: A Needle in the Haystack?

Richard Reich<sup>1</sup>

### ABSTRACT

The objective of this project is to model the influence of multiple factors in predicting risk of infection of lodgepole pine, (*Pinus contorta* Dougl.), in a comandra blister rust (CBR) (*Cronartium comandrae* Peck) resistance trial. Factors include: site, climate, ecology, host resistance, and alternate host abundance and susceptibility. Infection by CBR was very high on two of three sites and very low on the third. Stalactiform blister rust (SBR), caused by *Cronartium coleosporioides* J.C. Arthur, incidence was very high on one of three sites. Western gall rust, (WGR) caused by *Endocronartium harknessii* (J.P. Moore) Y. Hiratsuka, incidence is relatively low on all 3 sites. After 5 years of annual remeasurements the family rankings for comandra resistance appears to be stabilizing somewhat. Possibly three out of 130 families show a reduced level of CBR infection, indicating that a low level of moderate resistance exists within this population. However considerable variation exists between rankings on different sites. Two factors that may account for some of this variability are presented in detail. A spatial analysis of the percent incidence and the intensity of CBR infection revealed that the cumulative risk of infection after 5 years varies significantly, based on the distance from the alternate host bastard toad flax (*Geocaulon lividum* (Richardson) Fern). Incidence after 5 years is very high (85 – 90 percent) when lodgepole pine seedlings are in very close proximity (1m) to *G. lividum*. The incidence decreases dramatically over the first several meters away, and then more gradually, to approximately 45 percent at 15 m away. The second factor, which is potentially influencing ranking on one site only, is infection by SBR, whose relationship to CBR is examined through a family level correlation over the five year period assessment period.

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### INTRODUCTION

Comandra blister rust is one of the most damaging diseases of young lodgepole pine in British Columbia (van der Kamp and Spence 1987, Woods et al. 2000). It occurs throughout the province, but is most common in the Sub Boreal Spruce (SBS) biogeoclimatic zone. The disease alternates between comandra, *Geocaulon lividum*, and lodgepole pine (Ziller 1974).

Management options include increased planting densities of lodgepole pine, to account for anticipated mortality, and mixing non susceptible host species (British Columbia Ministry of Forests 1996). However, mortality from CBR is patchy, and most non susceptible species are typically not ecologically suitable on cold dry sites. They tend to develop very slowly and are often severely damaged and killed by repeated severe growing season frost damage (Reich and van der Kamp 1993). Hazard rating conducted for CBR by Jacobi et al (1993) identified several key variables. They found that rust incidence was significantly negatively correlated with distance to comandra. Numerous authors previously reported that infection is typically found in close proximity to the alternate host (Andrews 1959, Applegate 1971, Cordel et al. 1969).

Apparently, resistance to CBR has not been previously investigated. The general perception that resistance may not be common likely arises from reported occurrences of CBR incidence topping 80 to 90 percent in operational plantations. However, in 2000, a formal disease assessment of a Ministry of Forests progeny (F1) test south of Fraser Lake showed significant and large differences in family infection rates. The family infection levels ranged from 0 to 69 percent in the 309 families assessed, though the average incidence for the site was only 16 percent at age 16 years. Based on this glimmer of hope, a field trial was designed specifically to test comandra resistance of genotypes established in the Bulkley Valley low elevation seed orchard (Orchard 219).

## METHODS

Three sites were selected on the basis of high levels of CBR incidence, within established pine plantations, and abundance of the alternate host, *G. lividum*. Each two hectare plot (100 m by 200 m) was prepared by mechanically mulching the existing pine plantation trees. One hundred and thirty seedlots were included in the trial. Fifty seedlings per family were established, on a 1.5 m grid, in single tree plots. All three sites occur within the Sub Boreal Spruce dry cool (SBSdk) biogeoclimatic zone.

Two sites, Endako and Thompson, were assessed in 2006, 24 months after planting, and the third site, Holy Cross, was assessed in 2007. In 2006, 2007 and 2008, the assessment consisted of counting individual branch and stem infections caused by CBR, SBR and WGR. After 2006, new infections were painted with a dab of tree paint: blue in 2007, pink for infections that were new in 2008. In 2009 and 2010, only new occurrences of a rust species on a tree were recorded for all 3 rusts.

Upon determining from the 2006 assessment, that the spatial pattern of comandra infection was clustered, alternate host mapping was conducted in 2007 on all 3 sites centered on the 1.5 m tree grid. The number of individual *G. lividum* stems were counted within each 1.5 m by 1.5 m tree cell. Using GIS, 1 m concentric buffers were created around each cell containing Geocaulon in order to determine the distance that each tree was from the closest Geocaulon occupied tree cell.

Presence/absence (rather than individual stem counts) of common red paintbrush, *Castilleja miniata*, Dougl., was also assessed on the 1.5 m grid, on all 3 sites, in order to evaluate its role in local spread of SBR. Occurrence of cow-wheat, *Melampyrum lineare* Dres., was very common only on the Holy Cross site, but was not mapped due to the very low incidence of SBR on this site. *M. lineare* was not found on the two other sites.

Presence/absence of yellow rattlebox, *Rhinanthus minor* L., was only mapped on a small portion of the Endako site to investigate whether it played a role in the distribution of SBR. Yellow rattlebox was found in 2010 at a low level on the Holy Cross site where it has been detailed mapped on one half of the site.

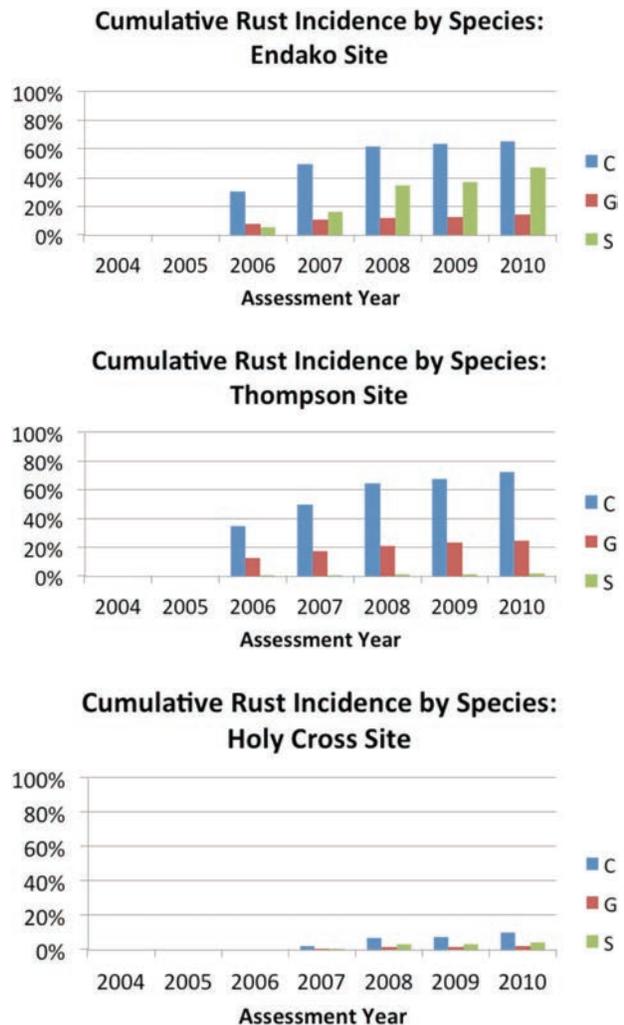
Additional data collected included a decimeter accuracy differential GPS survey for terrain in order to evaluate the effect of slope, aspect, and mesoslope position on patterns of rust occurrence and spread. A

standard ecoclassification of site series boundaries throughout each site was conducted in 2007.

## PRELIMINARY RESULTS

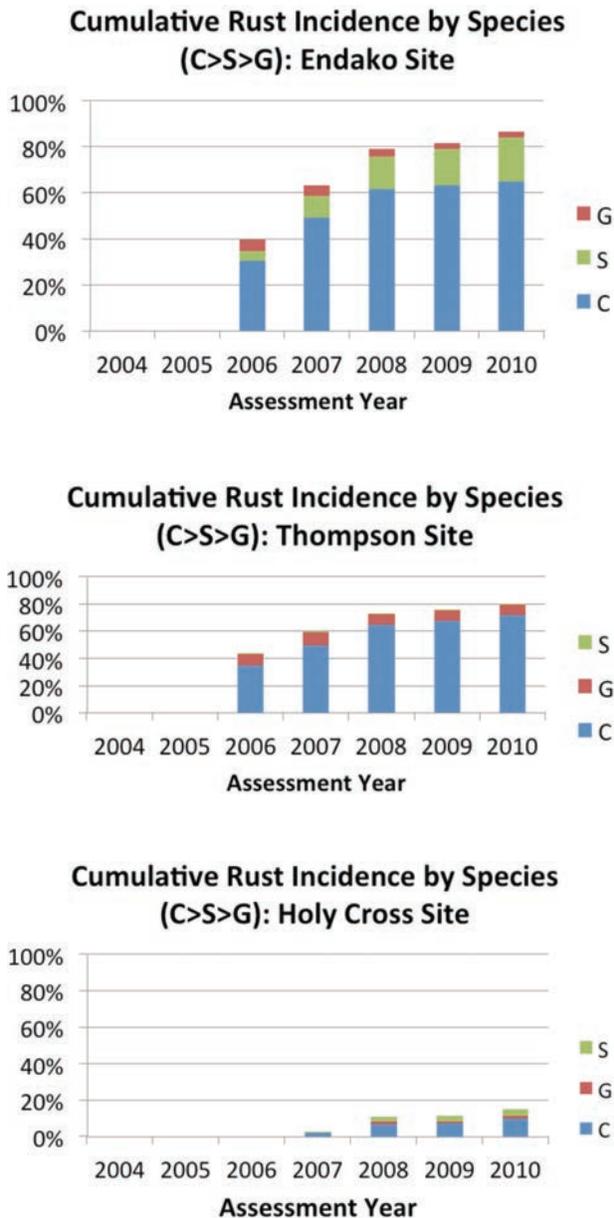
### Rust Occurrence by Species

Infection by CBR was very high on the Endako and Thompson sites, but very low on the Holy Cross site over the 5 year monitoring period. On the Endako and Thompson sites, moderate to high infection by CBR occurred in 2004, 2005, 2006, 2007, and 2008. These two sites are 12 km apart and likely share very similar weather conditions, whereas the Holy Cross site is 102 km to the southeast of the other sites and appears to be in a lower risk area. All three sites have relatively similar levels of the alternate host *G. lividum*. Figure 1a-c shows the cumulative rust incidence over time, for each hard pine rust species, at each site.



**Figure 1a-c**—Cumulative incidence of all three rusts over time at the three sites. The legend symbols are as follows: C - CBR; G - WGR; S - SBR.

Figure 2 shows the cumulative rust incidence over time, but infection is shown for the most lethal rust species. The order of severity is comandra first, and then stalactiform, then gall rust. As such, incidence of CBR includes trees infected by CBR or WGR, SBR incidence includes trees with WGR, whereas gall rust incidence is WGR only. Of note is the Endako plot where both CBR and SBR occur at a high level (figure 1a), but SBR “appears” underrepresented (figure 2a).



**Figure 2 a-c**—Cumulative incidence of all three rusts over time at the three sites. The legend symbols are as follows: C - CBR; G - WGR; S - SBR.

### Comandra Resistance Ranking

Figure 3 shows the cumulative incidence of CBR by family sorted by the 2007 assessment. The purpose of this sort is to demonstrate the high degree of variability in the results for the subsequent years even though the targets of 50 percent mean CBR incidence for the trial was achieved in 2007. By 2010, many families that were at the lowest end of the incidence range in 2007 became far more infected than families ranked as highly susceptible. Therefore, selecting the top 20 families in 2007 for a CBR resistance orchard would have been premature, resulting in numerous susceptible families being erroneously classified as “resistant”.

Since the vast majority of families have a high level of infection, it is clear that the level of even low or moderate resistance in improved seed source is very low at best.

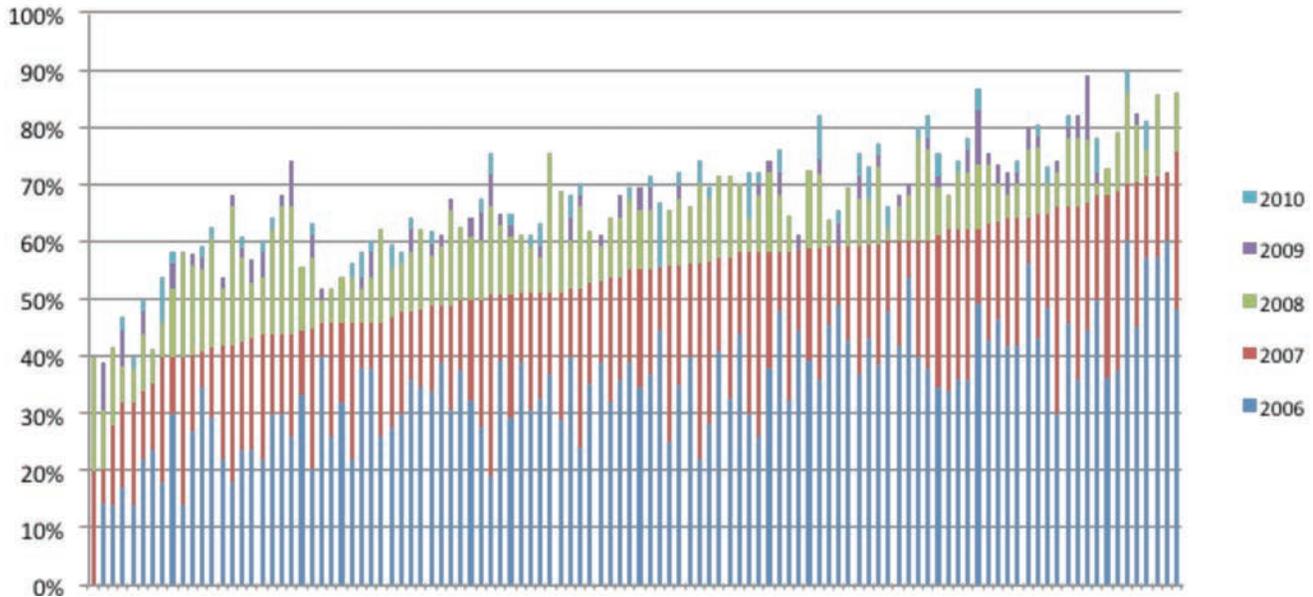
### Sources of Variability of Ranking

The relatively high degree of variability in ranking between families over time, and between sites, has several potential sources. This variability is likely due in part to the relatively low number of trees per family in relation to spatially variable alternate host distribution, and possibly the presence of other pests which may interact at the family level on some sites. A third factor which is being addressed in another study is the variation in susceptibility of the alternate host and will not be reported on here. However, based on the clustered distribution of alternate host (which supports a putative clonal designation for many clusters) and the relative severity of infection on the lodgepole pine associated with these putative clones, it would appear that there may be considerable variation in resistance between alternate host putative clones.

### Effect of Proximity to Alternate Host

Figure 4 shows the cumulative intensity of infection, over time, by one meter concentric zones centred around the alternate host.

## Endako: Family Comandra Incidence Sorted by Year 2007



**Figure 3**—Family comandra incidence sorted by year 2007.

The intensity of infection is the mean count of infections for trees within each one meter concentric zone. This assessment was conducted for 3 years on the Endako and Thompson sites. The risk is several times higher for trees in immediate proximity to the alternate host (1 m zone on graph) as compared to trees even a few metres farther away. The risk more gradually decreases for trees planted from 5 m to 17 m distant. The Endako site features very similar annual trends (but is not shown here).

This same pattern of decreasing risk with increased distance from the alternate host is exhibited with incidence of infection in Figure 5.

However, the risk is typically only two times higher in immediate proximity to the alternate host, than for trees planted several meters away, but decreases more gradually for trees planted approximately 6 m away from the alternate host.

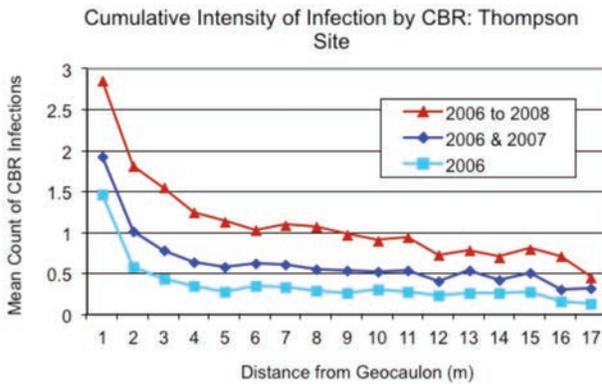
The difference in proximity based risk for incidence is less than half of that for intensity, indicating that incidence is not as sensitive a measure of the decrease in risk based on proximity to alternate host. In addition, the number of infections per tree will likely also be a more sensitive indicator of the likelihood of mortality, than incidence alone, since not all branch infections are lethal.

The graph for the Endako site shows a very similar pattern, but is not shown here. Note that the slight peak at 15 m is likely an artifact related to decreasing sample size with increasing distance from alternate host (minimum sample size was 30 trees). If the minimum was set at 100 trees, data points from 12 m to 17 m would not be shown.

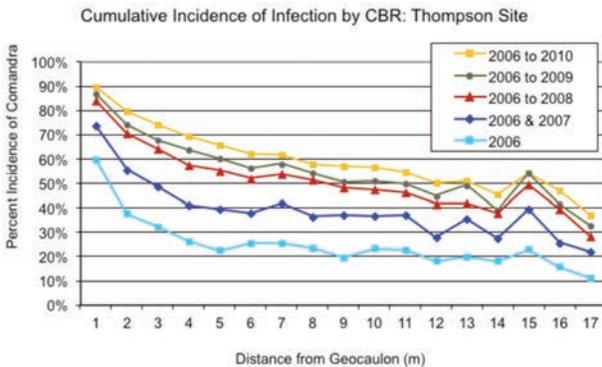
### Interaction with Other Factors

How other factors affect the ranking for CBR is unclear. However, the Endako site developed a high level of SBR which allowed a comparison of susceptibility at the family level between CBR and SBR. Although the CBR incidence peaked much sooner than the SBR incidence, both incidences increased steadily over time (see figure 1). The family level correlation  $R^2$  between the two rusts decreased steadily as the incidence of both rusts increased (figure 6). Note, that the 2009 data (and  $R^2 = 0.015$  label) is omitted from the Figure 6 to reduce clutter.

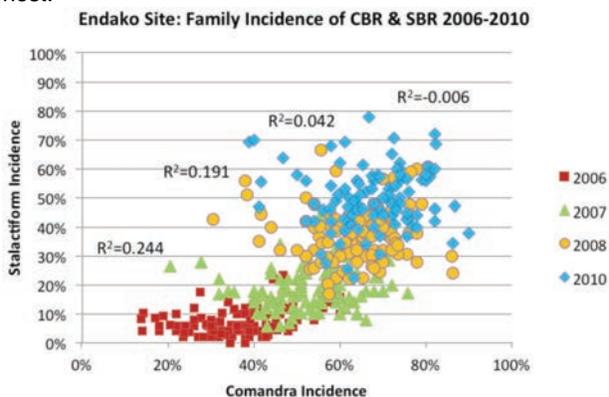
In 2006, when the SBR incidence was very low, but CBR incidence was high, there was a weak positive correlation  $R^2$  of 0.244. However, by 2010, the CBR was very high, and the SBR was very high as well (47 percent), and the correlation  $R^2$  dropped to -0.006. It therefore appears that resistance to these two rusts may be inherited independently. In addition, it would also appear that under insufficient levels of infection by one or more rust, the appearance of weak correlation could lead to an erroneous conclusion that two variables are related when they are not.



**Figure 4**—Cumulative intensity of infection, over time, by one meter concentric zones centered around the alternate host.



**Figure 5**—Cumulative incidence of infection, over time, by one meter concentric zones centered around the alternate host.



**Figure 6**—Correlation of comandra and stalactiform family level incidence for the Endako site. 2009 data was omitted to reduce clutter.

## DISCUSSION

### Infection Dynamics

Infection by CBR occurred sooner than expected, and at higher levels than expected, on two of the three sites. The timing of the infection is relatively simple to determine since assessments and infection marking were conducted annually. Although visible infections

were counted annually, the actual infection year would have normally occurred 2 to 3 years sooner. The visible infections observed in 2006 occurred only on the 2004 internodes. Fully 50 percent of those infections were sporulating, and the other 50 percent consisted of swollen non-sporulating cankers in the summer of 2006. In subsequent years, it was evident through classifying the stage of canker development in relation to internode age, that CBR and SBR infection occurred on all ages of foliage, though primarily on the current years foliage. For the most part, new infections counted in any assessment year corresponded to infection occurring 2 (to 3) years prior.

Although moderate to high infection by CBR can be attributed to 2004, 2005, 2006, 2007 and 2008 on the Thompson and Endako sites, this is not evident from Figures 1 and 2 since only cumulative infection is shown. In these figures, only newly infected trees result in an increase in infection from year to year. New infections on previously infected trees are not shown. This is important to note in terms of ranking families for resistance. Trees grown in high risk micro-sites were under high infection pressure for numerous years. Therefore if they remain uninfected, while their immediate neighbors are often reinfected annually with numerous new infections, these uninfected trees are much more likely to have some form of resistance.

### Variability Between Sites

The very low infection by all 3 rusts at the Holy Cross site demonstrates the range of variability that occurs within a single subzone/variant/site series classification across a large landscape. This variation is most likely due to weather events that occur more frequently in one geographic location than in another. The weather conditions at the Holy Cross site over the past 5 years apparently were unsuitable for all three rusts. It underscores the challenge of hazard rating based on any single set of variables such as ecosystem classification alone.

Even between the two highly infected sites, the relative proportion of each rust varied somewhat. Both sites had a similar level of *G. lividum*, yet even the CBR levels varied from year to year, presumably due to weather patterns.

### Evaluating the Stalactiform Distance Relationship

There was a similarly moderate amount of well separated, highly clustered *Castilleja* plants on all 3 sites. This distribution remained fairly static over the

study period. However, it seemed peculiar that SBR only reached a high level of infection on the Endako site, and the other two sites had negligible levels. Doubly perplexing was the fact that the weather was highly conducive for blister rust infection at the Thompson site for 5 years running, yet SBR was absent. It was somewhat surprising that the highly clustered spatial pattern of SBR at Endako in 2006 (6 percent SBR) and 2007 (16 percent SBR) didn't correspond spatially to the clustered *Castilleja* distribution. A subsequent search in the fall of 2007 for a previously unnoticed figwort alternate host was conducted near some clustered SBR infection. The only putative source that could be found was yellow rattlebox, a seldom mentioned, but confirmed alternate host for SBR blister rust (Ziller 1974). The only problem was that it was widespread across large portions of the Endako site in 2007, and was summarily dismissed due to the lack of spatial correspondence. Ironically, by the 2010 assessment (3 years later), yellow rattlebox had virtually disappeared from the site, but now the SBR was widespread across the entire site. Given that it takes 2-3 years for infections to become evident, it follows that the 2010 SBR spatial pattern likely reflects the 2007 rattlebox distribution. Since rattlebox was not fully mapped in any one year, the distance relationship can now only be inferred by examining the SBR spatial pattern over time. Discussion with a local range ecologist confirmed that yellow rattlebox is known to rapidly invade disturbed sites. This study shows that it can also rapidly disappear. Westbury (2004) confirms that *Rhinanthus* populations are often observed as transient patches.

The initial highly clustered nature of SBR in 2006 and 2007, likely indicates point source infection involving a short distance proximity relationship from small clusters of rattlebox, not unlike the CBR/*G. lividum* relationship described in this study (figures 4 and 5). The apparent rapid spread of the alternate host presumably resulted in a corresponding widespread distribution of SBR by 2010. Although the evidence is largely circumstantial, it is highly plausible that yellow rattlebox was in fact the main alternate host for SBR during this outbreak. It also follows that since *Castilleja* was highly clustered, but was not spatially related to the initial clustered SBR infection, that it may not be as important of an alternate host as was previously believed. The relative importance of the alternate hosts of SBR may still be an important knowledge gap.

### **Quantifying CBR Resistance**

Quantifying resistance to CBR is challenging due to significant variation in family ranking over time, between sites, and within sites. Significant effort was spent quantifying these risk factors. However, there remain a few families that consistently demonstrate a low incidence and intensity of infection, even when located in the highest risk locations. The vast majority of families tested appear to be very susceptible.

Within site variation was potentially very high due to proximity to the alternate host. Risk of incidence of infection was twice as high for trees located in immediate proximity to the alternate host. The intensity of infection was several times higher for trees located in immediate proximity to the alternate host. Therefore stratifying the sites based on alternate host proximity could ensure that families are ranked on a more uniform level of risk. Some families clearly were at higher risk than others based on the relative frequency of trees in close proximity to the alternate host.

The overall results indicate that only under what would be considered a very high level of risk do family rankings separate reliably. After 5 years of repeated infection pressure by CBR, and a CBR incidence of approximately 70 percent, several families still consistently remain in the top ranked families.

Since the range of SBR infection on the Endako site was very high this may affect CBR ranking on that site, even though there was no relationship between CBR and SBR. Families highly infected by SBR, may have developed less CBR due to interactive effects.

### **CONCLUSION**

Low to moderate resistance to CBR has been detected in a few of the 130 improved-seed, lodgepole pine families tested. This is clearly well below the required number of families that is needed to form a resistance orchard. If this test is representative of the other lodgepole pine orchards, the returns are likely insufficient to warrant their screening due to cost. It was concluded that reliable screening for resistance to CBR requires a very high mean site incidence of rust, in the order of 70 percent – 80 percent, based on sequential rankings conducted over time under conditions of steadily increasing CBR incidence.

Fine scale mapping of alternate host distribution enabled stratification of high risk locations with sites. High risk locations (immediate proximity to alternate host) have two to several times higher incidence and intensity, respectively, than the low risk locations. Stratification by proximity to alternate host permits greater separation between family rankings due to increased uniformity and certainty of rust pressure.

Other variables such as infection by other rust species may also confound ranking for CBR resistance, but this is unclear. Finally, it is speculated that *Rhinanthus minor* may be a much more effective alternate host for SBR spread than *Castilleja miniata*, based on strong circumstantial tempo-spatial evidence.

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# Poster Papers and Abstracts



BC's Two Jack Lake and Mt. Rundle. Wikimedia Commons





## Variation in *Heterobasidion annosum* on Different Hosts in Western Washington

Robert Edmonds<sup>1</sup>, Marianne Elliott<sup>2</sup>, and Kathryn Coats<sup>2</sup>

*Heterobasidion annosum* is common in forests of western Washington on a variety of conifer and hardwood hosts. Hsiang and Edmonds (1989) demonstrated physiological specialization of *H. annosum* on conifer hosts in Washington, but there has been little study of the genetic variation in *H. annosum* populations in Washington forests, although variability has been studied in Christmas tree plantations (Dart et al. 2007). They found that isolates from different trees had distinct genotypes, but root systems of individual trees were dominated by 1 or 2 genotypes. Garbelotto et al. (1999) also studied the genetic structure of *H. annosum* in white fir mortality centers in California and found that each mortality center was characterized by several fungal genotypes.

### OBJECTIVE

Our objective was to study the variability in *H. annosum* populations from different hosts in western Washington; Douglas-fir, western hemlock, red alder and cottonwood.

### METHODS

Fruiting bodies of *H. annosum* were obtained from 2 downed hemlock trees, 2 Douglas-fir stumps, and dead red alder and cottonwood trees near Seattle in western Washington.

The following DNA oligonucleotide primers were used for the arbitrary-primed polymerase chain reaction analysis (Garbelotto et al. 1999) NS3-NS1.5R; NS3-Ctb6; ITS4b-Mb2KimQ-KJ2; CNS3.6-Mb2NS6-ITS2; ML5-ML6.

PCR analyses were conducted at the Puyallup Research and Extension Center, Washington State University, Puyallup, WA.

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<sup>2</sup>Washington State University, Puyallup.

### RESULTS

There was considerable variation in *H. annosum* obtained from different tree hosts in western Washington using the 7 primers. There appeared to be no clustering by tree species, however. This variation is similar to that found in *H. annosum* (1) from different Christmas tree hosts in western Washington (Dart et al. 2007), and (2) white fir in California (Garbelotto et al. 1999). All isolates appear to be S strain *H. annosum*. Ostrosina and Garbelotto (2010) have proposed that the S strain in North America be known as *H. occidentale* and the P strain as *H. irregulare*.

We will continue to examine the variability in *H. annosum* in both western Washington and eastern Washington on different hosts.

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## Aspen In Montana and Northern Idaho: Third Year of Monitoring Aspen Condition in the Northern and Intermountain Regions

Holly S.J. Kearns<sup>1</sup>, Brytten Steed<sup>2</sup>, John C. Guyon II<sup>3</sup>, and James T. Hoffman<sup>4</sup>

Forest, range, and wildlife managers in the Interior West have documented a decline in total forest acreage and health within aspen forests since the 1970's. Two primary forces are most commonly cited as contributing to this decline: 1) changes in fire regimes since European settlement; and 2) heavy ungulate browsing. Forest insects and diseases are often notable, but play a largely undefined role in aspen decline.

Over the first two years (2006 – 2007), 126 permanent plots were established in aspen stands throughout Utah, Nevada, southern Idaho, and western Wyoming to provide data on forest damage/decline agents. In 2008, 76 permanent plots were established in Montana and northern Idaho. Plots were randomly established in stands that met the minimum criteria of having at least seven live stems  $\geq 5$ " dbh within a 1/20<sup>th</sup> acre plot. Site, stand, tree, and damage data were collected on each 1/20<sup>th</sup> acre plot for all live and recently dead stems  $\geq 5$ " dbh. Small trees ( $\geq 2$ " but  $< 5$ " dbh) and sprouts ( $< 2$ " dbh) were sampled on three 1/300<sup>th</sup> acre sub-plots nested within each plot.

The 76 plots in northern Idaho and Montana ranged in elevation from 2,265 to 8,040 ft. Conifer competition was severe on 27 percent and low to moderate on 61 percent of plots. Only 22 percent of surveyed aspen clones were considered expanding, while 49 percent were stable and 30 percent were retreating.

Over the course of this survey, 1,423 aspen 2.0 – 24.2 inches dbh were evaluated for crown dieback and damage agents. Approximately 91 percent of the aspen were alive; 82 percent of these had minor crown dieback (1-33 percent), 15 percent had moderate crown

dieback (34-66 percent), and 2 percent had greater than 66 percent of their crowns dead. The most commonly recorded damage agents on aspen  $\geq 2$ " dbh were wood boring insects, which were noted on 44 percent of surveyed stems. Other damages included: mechanical and animal damage on 29 percent, defoliating insects on 28 percent, sooty bark canker (*Encoelia pruinosa*) on 14 percent, Phellinus trunk rot (*Phellinus tremulae*) on 9 percent, and both foliar diseases and Cytospora canker (*Valsa sordida*) on 8 percent.

Aspen sprouts were present on 86 percent of plots. Sprout densities ranged from 0 to 31,600 sprouts per acre (spa); 46 percent of plots had 1,500 or more spa. The majority of plots (53 percent) also had seedlings of other species present with densities ranging from 100 to 5,300 seedlings per acre. Browse damage was recorded on 75 percent of plots with 38 percent of aspen sprouts affected. Other damages recorded on aspen regeneration included foliar diseases including ink spot (*Ciborinia whetzellii*) and Marssonina leaf blight (*Marssonina* sp.) on 28 percent of aspen sprouts, and shoot blight (*Venturia tremulae*) and insect defoliation on 16 percent and 10 percent, respectively.



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## Landscape Genetics of White Pine Blister Rust

Simren Brar<sup>1</sup> and Richard C. Hamelin

White pine blister rust (WPBR) is caused by *Cronartium ribicola* and is a severe disease of five-needle pines. The pathogen was introduced to North America in the early 1900s on infected seedling from Europe and has spread throughout North America causing heavy mortality in five-needle pine stands and plantations. The pathogen of WPBR has a complex life cycle including five different spore stages and requires both a five-needle pine host and a *Ribes* spp. host to complete its life cycle. Previous studies (Hamelin et al. 2000) have shown that eastern populations of *C. ribicola* are more genetically diverse than western populations, with only a third of the diversity in eastern Canada found in the west. However, there is little data on diversity at the landscape level. We hypothesize that genetic differences occur in the population of WPBR and landscape features are contributing, with altitude being an important factor and that increases in elevation will result in increases in homozygosity.

Single aecia were collected from infected trees in plantations and natural stands throughout British Columbia and Alberta. Samples were collected from both white bark pines and western white pines.

EST libraries of *Cronartium ribicola* are currently being used to develop SNPs markers, the most abundant class of polymorphisms in genomes. To maximize SNP discovery, the EST libraries were made from bulk aecia from Eastern Canada where more diversity is expected. The ESTs were then compared for homology in Genbank database to identify gene function. Primers were designed for any EST that had homology to fungi. Primers were designed for an average of 500bp for a given EST to allow for PCR amplification and sequencing. These primers were optimized and run on a panel of 16 samples, 12 from different locations in BC and 4 from eastern Canada. DNA from all PCR products obtained for the 16 sample panel were sequenced. The sequences were aligned and analyzed to identify DNA positions that comprised mutations.

To date 144 primer pairs have been designed, 36,153 bp screened and 13 confirmed SNPs. This represents 3.6 SNPs every 10000 bp which is a very low level of diversity. The next step will consist of genotyping the polymorphic SNPs in our range wide *C. ribicola* populations.

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## Spore Dispersal and Infection of *Dothistroma septosporum* in Northwest British Columbia

Kennedy Boateng<sup>1</sup>, Kathy Lewis<sup>1</sup>, and Alex Woods<sup>2</sup>

The spatial pattern and range of spore dispersal and infection of *D. septosporum* in northwest British Columbia were studied. The study examined the differences in frequency of dispersal by ascospores (sexual spores) and conidia (asexual spores) in rain water and air, the relative distance of spore dispersal from inoculum sources and the influence of climatic variables (rainfall, temperature, relative humidity and leaf wetness) on spore dispersal and infection of *D. septosporum*. The timing and number of spores dispersed was monitored in two infected lodgepole pine plantations at the Bulkley timber supply area within the Skeena Stikine forest district by using spore traps, and climatic factors were recorded using HOBOS and microclimate loggers from May 30 to September 26 in 2009 and April 23 to June 28 in 2010. Spores were trapped at different distances and heights from inoculum sources, and one-year-old lodgepole pine seedlings were exposed to infection at the study sites during the 2009 field season.

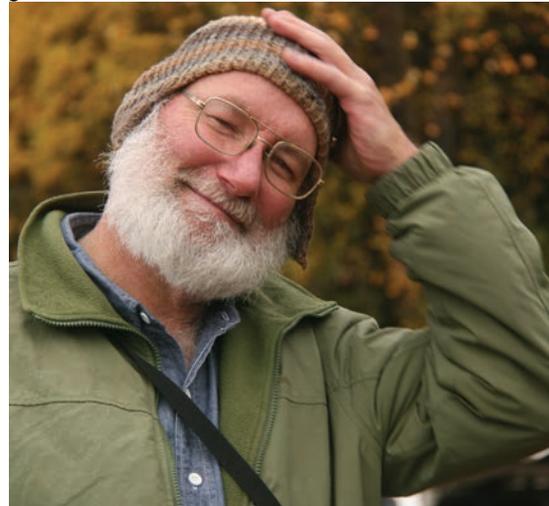
Ascospores of the fungus were never observed throughout the study period. Conidia were trapped from June to September in 2009 whenever rain fell; the dispersal reached its peak in July. During the 2010 field season, spores were first trapped in June and this confirmed the commencement of the spore dispersal in 2009 at the study area. No spore was observed during dry conditions or rainless days and the highest number of spores was observed on the slides positioned directly beneath the inoculum sources. The number of spores trapped decreased with increase in distance; it was rare to trap spores 2m away from inoculum sources. Based on mixed effects model used for the data analysis, the number of conidia dispersed at different distances from the inoculum sources was significantly different ( $P < 0.0001$ ) but there was no significant difference between the numbers of spore trapped at the different heights ( $P > 0.05$ ).

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Most spores were trapped when the daily mean rainfall, temperature, relative humidity and leaf wetness was ranging between 10 and 17 mm, 9.61 and 17.61 °C, 78.70 and 85.42 percent and 48.07 and 75.08 percent, respectively. The timing and number of conidia dispersed was strongly related to the climatic factors most especially under the control of rainfall. Temperature negatively influenced the spore dispersal while the other factors positively influenced the dispersal. From the mixed effect models, all the climatic factors significantly affected the number of conidia dispersed ( $P < 0.0001$ ). Results of the effect of climatic factors on *Dothistroma* infection is in progress.





## ***Cronartium ribicola* Impacts in Plantations of Improved *Pinus monticola* in Northern Idaho**

**John W. Schwandt<sup>1</sup> and Holly S.J. Kearns<sup>2</sup>**

Since its introduction into western North America, *Cronartium ribicola* has spread throughout the range of nearly all native five-needled pines. *C. ribicola* has played a major role in dramatically reducing the dominance of *Pinus monticola* on several million hectares and is primarily responsible for major changes in successional pathways throughout western North America. The current emphasis for blister rust control and management is to incorporate natural resistance mechanisms into regeneration. Over the past 35 years, more than 100,000 hectares (250,000 ac.) of *P. monticola* plantations with improved levels of resistance (F2) have been established in the northern Rocky Mountains. Surveys of 66 plantations 7–15 years after planting found infection levels varied from 0 percent to 95 percent, but F2 stock always out-performed natural regeneration. Over an 11 year period rust infection levels in permanent monitoring plots in 18 of these plantations increased from an average of 14 percent to 39 percent on F2 stock and from 12 percent to 63 percent on naturally regenerated white pine. Mortality levels during this time increased from 8 percent to 22 percent on F2 and from 7 percent to 47 percent on natural regeneration in the same plantations. We are also finding some trees with abnormal cankers with varying degrees of callus and resin that appear to be related to canker girdling rates in plantations of improved stock. Annual monitoring of over 190 branch cankers during a five-year period found an average canker expansion rate of 4.8 cm (1.9 in.) per year, which is similar to that reported on unimproved stock. Cankers that were less than 15 cm (6 in.) from the stem had a 74 percent probability of reaching the bole within five years, while cankers more than 50 cm (20 in.) from the bole had an 85 percent probability of dying before reaching the stem.



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## Firewood as a Potential Pathway for Native or Exotic Forest Pests in the Southern Rocky Mountains

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Firewood can harbor harmful insects and pathogens and facilitate the transport of these organisms across state or national borders. There are currently a number of invasive native or exotic species we hope to prevent from moving into new areas of the U.S. In 2008, approximately 4.3 million people camped in National Parks in the southern Rocky Mountains (Arizona, Colorado, Nevada, Utah and Wyoming). We propose that camper movement of firewood is a high risk pathway of non-native organisms within and into western states, especially if campers come from infested areas. In 2007-09 we interviewed campers at seven Colorado State Parks and 13 National Parks in the Southern Rockies. Firewood was purchased from a randomly stratified sampling of retailers in six cities in each of three states (Colorado, New Mexico and Utah) in winter 2008-09. Firewood properties (wood supplier, state of wood origin, bark presence, genera, and evidence of previous insect/fungal infestation) were determined, and purchased wood was placed in rearing cages and checked bi-monthly for insects.

Personal campground surveys indicated that 66 and 60 percent of campers had firewood at State and National parks, respectively. Of National Park campers with firewood, 39 percent brought wood from outside the park state. Based on visitor statistics, this proportion could equate to 329,920 yearly incidents of campers bringing out-of-state wood into the National Parks surveyed. Results varied by state: 23 percent of campers in Arizona parks brought firewood from outside the state, 8 percent in Colorado, 43 percent in Nevada, 57 percent in Utah and 24 percent in Wyoming. Of campers with firewood, 32 percent of the firewood was purchased inside the park, 25 percent was purchased outside the park, and 17 percent was cut or collected from the camper's home. Sixty-four percent of firewood had bark attached, 53 percent bore evidence of previous insect presence, and 39 percent showed evidence of fungal infestation.

A total of 412 firewood bundles were purchased and placed in 226 insect rearing bins. Insects emerged from firewood in 50 percent of the bins (totaling 2,621 insect specimens) from at least 19 wood species from 14 states, including the Southern Rocky Mountain states and California, Oregon, Nebraska, Montana and Texas. Most insects emerged from conifer species of firewood. Prominent genera included

*Polygraphus* (n = 977 individuals), *Pityogenes* (n = 330), *Ips* (n = 160), *Gnathotrichus* (n = 92), *Scolytus* (n = 28) and *Dendroctonus* (n = 26) genera; however two genera in the family Buprestidae have emerged from oak firewood (*Agrilus* and *Chrysobothris*, n = 8).

Transporting untreated firewood provides a pathway of introduction for forest pests to new areas. We recommend including firewood as a risk in invasive species education efforts, promoting local wood sales to incoming National and State Park visitors, buying and selling locally cut or heat-treated firewood near campgrounds, and banning the movement of firewood into parks and across state borders. Future movement of non-native pests might be prevented if we can manage and restrict the movement of infested wood, particularly firewood.

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## From the Big Blow-up to the Insect and Disease Slow Burn

Sandy Kegley<sup>1</sup>, Sue Hagle<sup>1</sup>, Holly Kearns<sup>1</sup>, Lee Pederson<sup>1</sup>, and John Schwandt<sup>1</sup>

The 1910 fires helped set the stage for insect and disease mortality now occurring throughout northern Idaho and Montana. While the effects of fire are immediate and obvious, insect and disease effects can resemble a “slow burn” by being less apparent and occurring over a longer period of time. However, the annual impacts of insects and diseases are often far greater than those of even the largest wildfires.

Each year nearly as much timber volume is lost to root diseases and bark beetles as was burned in the 1910 fires. From 2000 – 2009, average annual mortality from root diseases in northern Idaho and Montana was 4.39 billion board feet (bbf) and average annual mortality from bark beetles was 0.46 bbf, compared to the estimated volume loss from the 1910 fires of 7.65 bbf. Over that same period, wildfires resulted in average annual mortality of 1.34 bbf across the same area. During the past 11 years, bark beetles and root diseases killed trees on more than four times the acreage burned by wildfires in the US Forest Service’s Northern Region, which encompasses northern Idaho, Montana, and North Dakota.

Long-term forest impacts resulting from the 1910 fires are far greater than just acres burned and timber volume lost. Root diseases are increasing in severity in mature Douglas-fir and grand fir forests that developed after the fires. Root diseases can kill 25 – 50 percent of the mature trees in affected areas every 20 years and are the primary cause of tree death in 66 percent of northern Idaho forests.

Current bark beetle outbreaks are occurring on lands burned in 1910 and colonized by lodgepole pine that is now of an age and size susceptible to mountain pine beetles. Bark beetles can kill 90 percent of susceptible trees in less than 10 years during outbreaks.

White pine blister rust invaded Idaho and Montana in the 1920s and infected vast acres of white pines that regenerated after the 1910 fires. The invasion of white pine blister rust is a major factor in the 90 percent reduction of western white pine dominated forests on over five million acres, many of which shifted in composition to more root disease-susceptible Douglas-fir and grand fir.

The resulting losses from these “slow-burn” agents are often overlooked or unrecognized, yet they often play a more important role than fires in shaping the resiliency and health of our forests.

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<sup>1</sup>USDA Forest Service, Forest Health Protection, Coeur d’Alene, ID.





## Sensitivity of Western Redcedar Growth to Climate and Western Hemlock Looper in British Columbia's Inland Temperate Rainforest

Chris Konchalski<sup>1</sup>

Conflicting future predictions for British Columbia's inland temperate rainforest under climate change scenarios make it an area of special concern for research, management, and conservation. The inland temperate rainforest results from a globally unique combination of humidity and continental climate, and is host to rare lichens and bryophytes, and tree species that are normally considered coastal. The focus of this study is to quantify the sensitivity of western redcedar (*Thuja plicata*), a dominant canopy tree, to climate and western hemlock looper by analyzing the annual growth increments. The influence of slope position, aspect, and elevation on the sensitivity of western redcedar to climate and western hemlock looper will be analyzed. We placed six elevational transects each consisting of two sites in the cedar-hemlock forest approximately 100 kilometers east of Prince George, British Columbia, as well as one putatively sensitive climate site and one level, well drained site. Correlation and regression analysis of climatic variables will be used to explain variation in ring widths across time. Criteria will be developed to quantify ring suppression during known western hemlock looper outbreaks to extend the outbreak analysis into the past. Understanding the sensitivity of western redcedar to climate and western hemlock looper in different settings will help predict

future forest responses and processes in the inland temperate rainforest. Keywords: dendrochronology, dendroclimatology, western hemlock looper, western redcedar, inland temperate rainforest.

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## Characterizing Douglas-fir Tissue Colonization by the 'Sudden Oak Death' Pathogen, *Phytophthora ramorum*

Kathleen McKeever<sup>1</sup> and Gary Chastagner<sup>1</sup>

In 2001, Koch's Postulates confirmed Douglas-fir as a host for *P. ramorum*. Naturally-infected saplings have been observed in California forests, and studies on artificially inoculated Douglas-fir stems and shoots have established susceptibility parameters. Although naturally occurring stem infections in the U.S. have been limited to smaller diameter seedlings and saplings, *P. ramorum* stem cankers were recently observed on 8-year old plantation grown Douglas-fir in Great Britain in 2009. All previous research has served to substantiate the importance of performing studies to characterize the behavior of *P. ramorum* in Douglas-fir and assess the potential risk that this pathogen poses to Douglas-fir ecosystems.

Artificial inoculation experiments were carried out to provide a thorough analysis of the colonization of Douglas-fir by *P. ramorum*. Goals of this research included determining which tissues are colonized by the pathogen, whether woody tissues are able to support sporulation, the likelihood of stem infections occurring on Douglas-fir with intact bark, and the viability of the pathogen in foliage. Methods employed included isolation, ELISA, and histological examination of stem tissues; RT-PCR and isolation to determine colonization efficacy and viability of the pathogen in needle tissues; and baiting studies to determine the ability of Douglas-fir bark to inhibit colonization of Rhododendron leaves by the pathogen.

ELISA results showed that proteins of the pathogen were detectable in the phloem, cambium, and superficial xylem, with infrequent detection in asymptomatic tissues. The pathogen was able to be isolated from all symptomatic woody tissues tested, but not from non-discolored tissues. ELISA and isolation techniques produced results that were highly positively correlated ( $r^2 = 0.62$ ,  $p = 0.78$ ), and histological observations paralleled information

derived from these techniques. Douglas-fir bark reduced infection on Rhododendron leaf baits by up to 83 percent in the presence of inoculum. Results from foliage inoculations indicated that pathogen DNA is detectable using qPCR methods, but there was an inability to isolate the pathogen from needle tissue. Evidence of spore formation in woody stem tissues has not been observed.

It was concluded that the pathogen may be able to infect into the shallow xylem tissues of Douglas-fir in the presence of wounding, but there was no evidence of sporulation in tissues. The inability to isolate the pathogen from non-discolored tissues suggests that the proteins detected by ELISA outside of the lesion may be elicitors that are secreted in advance of hyphal colonization. The inability to isolate the pathogen from colonized needles may indicate the presence of chemical inhibitors that render the pathogen non-viable subsequent to initial infection. Finally, the ability of Douglas-fir bark to suppress infection of Rhododendron leaf baits suggests that the bark is inhibitory to *P. ramorum* and that infection of woody stem tissues through intact bark may be limited. Further research is required to identify the nature of this inhibition.

Characterization of *P. ramorum* colonization of Douglas-fir tissues is relevant to our current understanding of the behavior of the pathogen in different hosts and may enhance our ability to assess risk and create adequate regulations to thwart the spread of this exotic pathogen.

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## Alternatives to Methyl Bromide USDA-PAW Project Work Plan: Forest Tree Nurseries

Jerry Weiland<sup>1</sup>, Anna Leon<sup>2</sup>, Bob Edmonds<sup>2</sup>, Will Littke<sup>3</sup>, and  
John Browning<sup>3</sup>

This USDA/ARS study was the first replicated trial of methyl iodide and dimethyl disulfide (DMDS) coupled with metam sodium (chloropicrin) in the Pacific North West. The study design was replicated across three facilities with a combined total production of ~100 million transplant Douglas-fir seedlings in Washington and Oregon. Furthermore, low buffer fumigant rates (less than 100-foot) have not been previously tested in combination with VIF tarps, nor compared against current operational MBC 67:33 formulations. This study indicates that any of these chemicals could be considered as a replacement to methyl bromide based solely on control of Fusarium in bareroot Douglas-fir nurseries. Metam sodium was the only fumigant that showed levels of Fusarium growth that were significantly lower than other chemical treatments in all of the nurseries. Methyl iodide is another promising replacement fumigant; however in this trial its efficacy was less reliable than metam sodium. Under VIF tarp, methyl iodide was efficacious at Aurora and Canby; however it was in the less efficacious group of fumigants at Webster. There is no previous history of use of DMDS as an alternative fumigant in PNW conifer facilities. DMDS was only in the most efficacious group of fumigants at Aurora. Root and soil

isolations for Fusarium and Pythium pre- and post-fumigation were subjected to PCR analysis. The results confirm the isolation of *F. commune*, *F. oxysporum* and *G. fujikuroi* from all three nurseries. The Pythium complex included *P. irregulare* (1) as the most common at Olympia (64 percent), *P. irregulare* (2) at Aurora (59 percent), and *P. dissotocum* at Canby (48 percent). *Cylindrocarpon destructans* and *C. didymum* were routinely isolated from soil and seedlings. Fluorescent Pseudomonad populations (potential biocontrol agents) increased following fumigation but declined rapidly through the crop cycle. Packable seedling yields were not significantly different between fumigants, but all fumigants were significant against untreated soil. Recommendations for further testing and application of these treatments are contained in the final report.

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## Health of Whitebark Pine Forests after Mountain Pine Beetle Outbreaks

Sandra Kegley<sup>1</sup>, John Schwandt<sup>1</sup>, Ken Gibson<sup>2</sup>, and Dana Perkins<sup>3</sup>

Whitebark pine (*Pinus albicaulis*), a keystone high-elevation species, is currently at risk due to a combination of white pine blister rust (WPBR, *Cronartium ribicola*), forest succession, and outbreaks of mountain pine beetle (MPB, *Dendroctonus ponderosae*). While recent mortality is often quantified by aerial detection surveys or ground surveys, little information is presented to describe what stands look like following MPB outbreaks. This information may help prioritize areas for restoration. From 2008–2009, the severity of MPB impacts was measured in 42 whitebark pine stands in Idaho, Montana, and Wyoming. WPBR was recorded on remaining live mature whitebark pine and whitebark pine regeneration. Probable stand trajectory was determined by comparing abundance and health of remaining whitebark pine with other competing tree species.

### Mountain Pine beetle

During the recent outbreak, 30 to 97 percent of whitebark pine basal area tallied within each stand was killed by MPB. Whitebark pine density (BA) was reduced by more than 80 percent on over half of stands surveyed. Over 50 percent of whitebark pine basal area was lost on 81 percent of stands and, currently, 76 percent of sites have less than 50 ft<sup>2</sup>/ac of live whitebark pine basal area remaining.

In central Idaho, current outbreak losses were compared to losses from an outbreak that occurred circa the 1930s. In four of six stands attacked in both periods, more whitebark pine basal area was killed in the 1930s.

### White Pine Blister Rust

There were a total of 2,473 live, mature whitebark pine trees tallied on the 26 sites. The average WPBR infection was 23 percent and ranged from 0 percent to over 80 percent. Most infections (54 percent) tallied were branch cankers, and severity was light. Only 10 percent of all trees had severe infections with top kill that would affect cone production. The lowest average rust infection levels were in central Idaho (0 to 5.3 percent), while the highest were in northern Idaho, western Montana, and the Greater Yellowstone Area (GYA). WPBR infection levels on regeneration varied from 15 percent to 23 percent in northern Idaho, from 6.1 percent to 67.9 percent in western Montana ranged, and from 4.2 percent to 80.8 percent in the GYA.

### Stand Trajectories

The probable stand-composition trajectory for these whitebark pine sites was based on WPBR infection levels, whitebark pine basal area reduction due to MPB, live whitebark pine basal area and percent of stand composition, abundance of other mature species, and the abundance and health of all regeneration. Based on these criteria, we found that 24 of 42 (57 percent) of stands surveyed will likely convert from whitebark pine to other cover types without restoration efforts or wildfire. These stands should be considered high priority for active management alternatives that would assist in enhancing whitebark pine restoration.

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## ***Phytophthora ramorum* Detection and Monitoring in Western Washington Waterways, 2010**

**Daniel Omdal<sup>1</sup> and Amy Ramsey-Kroll<sup>1</sup>**

*Phytophthora ramorum*, an exotic plant pathogen, is the causal agent of Sudden Oak Death (SOD), ramorum leaf blight, and ramorum dieback. The pathogen can move aerially through landscapes with wind and wind-driven rain, such as in the forests of Oregon and California. In California, hundreds of thousands of tanoak and oak trees have been killed by *P. ramorum* since first detected there in 1995. The pathogen can also be moved long distances in nursery stock because of its ability to survive in plant material, soil and water.

Western Washington is at risk for *P. ramorum* caused diseases and *P. ramorum* spread due to the presence of known hosts in the natural environment, suitable climatic conditions (extended periods of moist weather and mild temperatures), and the presence of plant nurseries with positively identified *P. ramorum* infected host stock. To date, the pathogen has only been detected in locations that are either at or near plant nurseries in western Washington.

The Washington Department of Natural Resources has been conducting aquatic monitoring and forest and nursery perimeter surveys since 2003. Until 2006, *P. ramorum* had only been detected in western Washington nurseries. In 2006, an aquatic detection site established in a stream running through a *P. ramorum* positive nursery resulted in positive *P. ramorum* samples from the water. Since 2006, detection and monitoring efforts for *P. ramorum* primarily focused on water courses associated with nurseries identified as containing *P. ramorum* plant stock. From 2007-2009, *P. ramorum* was detected multiple times in the Sammamish River, King Co., WA using native *Rhododendron macrophyllum* leaf traps,

or stream baits. However, after the 2009 survey season, it remained unclear where the detected *P. ramorum* inoculum was originating from and whether there were one or multiple entry points of *P. ramorum* inoculum into the Sammamish River.

During our 2010 *P. ramorum* monitoring, seven positive sites were identified. All were associated with the Sammamish River. Four of these sites were also *P. ramorum* positive in 2009. During this year's sampling, efforts were made to place a majority of the Sammamish River stream baiting traps in water courses entering into the Sammamish River, yet outside of the influence of the river. These methods facilitated an increased precision of *P. ramorum* inoculum detections entering into the Sammamish River. Three likely points of inoculum entry have been identified in the Sammamish River.

The originating point of the *P. ramorum* inoculum detected in our survey remains unknown. The area is undergoing rapid development, making it difficult to track watercourses upstream and hindering vegetation surveys along the watercourses. Dr. Gary Chastagner's lab at Washington State University continues to conduct molecular fingerprinting on the positive *P. ramorum* samples in efforts to answer some of the questions associated with *P. ramorum* in the Sammamish River.

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## Identification of Control Agents and Determination of Factors Affecting Pathogenicity of *Phytophthora ramorum*- A Research Update

Simon F. Shamoun<sup>1</sup>, Grace Sumampong<sup>1</sup>, Elisa Becker<sup>1</sup>,  
Marianne Elliott<sup>2</sup>, Aniko Varga<sup>3</sup>, Saad Masri<sup>3</sup>, Delano James<sup>3</sup>, and Karen Bailey<sup>4</sup>

*Phytophthora ramorum* causes a canker disease of oaks which is known commonly as sudden oak death. This disease has resulted in the loss of tan oaks and live oaks in the coastal regions of northern California and southwestern Oregon, USA, and is a serious threat to the native forests of North America. The pathogen is believed to have been introduced to Europe and North America, with distinct sub-populations identified on the different continents. The known host range of *P. ramorum* is very broad and includes species such as rhododendrons, viburnums, beech, Oregon grape, salal, arbutus, and other woody ornamentals. There are many hosts for *P. ramorum* present in forested and urban areas in Canada. These are primarily foliar hosts that can serve as potential reservoirs for *P. ramorum* inoculum. Establishment of *P. ramorum* on these hosts creates the risk of disease spread to more susceptible hosts in other locations, especially through the nursery trade. The Canadian Food Inspection Agency (CFIA) confirmed the presence of *P. ramorum* in plants from a number of retail garden centers in the Vancouver, BC area in 2004. To date (2010), CFIA has completed its 2010 national survey for *P. ramorum*. Of the 133 nurseries sampled throughout Canada, *P. ramorum* has been detected at five retail nurseries and two propagation nurseries in urban areas of southwest BC. Five of the nurseries also tested positive for the pathogen in 2008 and 2009. The infested material at each site has been deep buried or incinerated under CFIA supervision. The infected plants included rhododendrons, pieris, camellia, kalmia, hybrid witch hazel and drooping leucothoe. The potential impacts of *P. ramorum* establishment in Canada are estimated to include direct and indirect losses to the horticulture industry and forest industry if SOD will establish in forested areas of Canada.

To date, we have delivered the following results:

1. Developed PCR-RFLP molecular markers that enabled us to identify three lineages of the

North American and European of populations of *P. ramorum*.

2. Evaluated the efficacy of several commercial biocontrol products and fungicides on North American and European populations of *P. ramorum*. There was great variability among *P. ramorum* isolates in their behavior with biocontrol agents, both in vitro and on detached leaves.
3. Of 12 fungicides tested in vitro on 9 different genotypes representing the three lineages of *P. ramorum*, all fungicides were effective at reducing the zoospore germination of all genotypes at concentration below the recommended amount.
4. Examined phenotypic plasticity and aggressiveness among isolates and within the three lineages of *P. ramorum*. Isolates of EU and NA2 lineages tend to be the most aggressive on detached leaves.

This research project is a comprehensive project with the potential for valuable outcomes that may contribute to protecting Canadian nursery and forest industries. This is the only group in Canada performing research of this scope and magnitude related to developing effective controls for *P. ramorum* and better understanding of its biology, pathogenicity, and molecular diagnostics of population structure and diversity.

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## Castilleja and Pedicularis Confirmed as Telial Hosts for *Cronartium ribicola* in Whitebark Pine Ecosystems of Oregon and Washington

Robin L. Mulvey<sup>1</sup> and Everett M. Hansen<sup>2</sup>

The primary objective of this research was to determine if native species of *Castilleja* and *Pedicularis* are naturally infected by *Cronartium ribicola* in whitebark pine ecosystems of the Oregon and Washington Cascade Range, U.S.A. Secondary objectives were to monitor the phenology of aecial and telial hosts to determine if there is sufficient time for *C. ribicola* to complete its life cycle within high-elevation stands; and to evaluate the variety of susceptible native host species within these genera through field and growth chamber inoculation. These objectives were approached through fieldwork in 2008 and 2009 in whitebark pine ecosystems at Mt. Rainier, Mt. Adams, Mt. Hood, Mt. Bachelor, Tumalo Mtn. and Crater Lake. Forty-nine observational study plots were established and monitored. Natural *C. ribicola* infection was detected on 84 *Pedicularis racemosa* plants and five *Castilleja* plants (*C. applegatei*, *C. miniata*, and *C. parviflora*). Field observation provided evidence that there is sufficient time for *C. ribicola* to complete its life cycle on hosts within high-elevation whitebark pine stands. In 2009, 18 field inoculation

plots were established at Mt. Rainier and Crater Lake. Field inoculation confirmed the susceptibility of two additional species within these genera, *C. arachnoidea* and *P. bracteosa*. All four *Castilleja* species inoculated in the growth chamber developed infection, with an overall infection incidence of 62 percent (167 out of 270 plants). The identity of the rust species on field specimens as *C. ribicola* was verified through PCR and sequencing of the ITS1-5.8S-ITS2 region of DNA. Improved understanding of the role of these newly-recognized hosts in white pine blister rust epidemiology should be used to prioritize sites for the restoration of ecologically-valuable whitebark pine.

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## Do Soil Properties Explain Root Disease in Western Montana Forests?

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The objective of this project, based in the Lolo National Forest outside of Missoula Montana, was to explore the relationship between the severity of root disease in western conifers and a number of soil properties, including texture, pH, and soil phosphorous (P) and nitrogen (N). Many species of fungi cause root disease, and they often overlap, making it difficult to link symptoms with fungal species. During the study, *Phaeolus schweinitzii* root and butt rot was suspected and both P (affecting ponderosa pine) and S type (affecting Douglas-fir and true firs) Heterobasidion sp. root rot and *Armillaria* sp. root disease were confirmed on the sites. I hypothesized that the presence and/or severity of root disease caused by fungi would vary as a function of one or more soil properties. I collected soil samples from 50 pre-established permanent plots on the Lolo National Forest, located in the Bear Creek and Deep Creek drainages. I then took soil samples (0-10 cm deep) from the N, E, S, edges of all plots. Following collection, I performed particle size analyses and examined soil nutrient content and pH. There were differing results depending on which statistical program was being used. Using a Spearman correlation there was a significant ( $P < 0.05$ ) positive correlation between inorganic nitrogen in the soil and the percent silt ( $P = .026$ ,  $R = 0.339$ ). With data log-transformed, in the Pearson method of correlation, I found the percent clay in the soil and the percent severity of root disease are significantly negatively correlated ( $P = 0.046$ ;  $R = -0.314$ ). Also, there is a negative correlation between the percent clay and the inorganic N in the soils ( $P = 0.02$ ;  $R = -0.377$ ). There was no significant correlation between any of the other variables. When the data was run through a program that compares environmental and species variables to see if any environmental variables are causing a variation in the species variables, there was no causal

variation found. The negative correlation between percent clay and root disease severity suggests that the more clay that is present in the soil, the more resistant the plants are to root disease. This could be because clay generally has more water-holding capacity than other soil textures. The water-holding capacity could allow trees to become more vigorous and allow them to enhance their defenses against the root disease. The negative correlation found between percent clay and nitrogen suggests soils with more clay may have less nitrogen, and the presence of less nitrogen within the soil may weaken the trees ability to defend against the fungus and enhance the efficacy of infection. I was surprised at the combination of clay and nitrogen because of clay's colloidal and cation exchange properties.

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## Multilocus Genotypes Indicate Differentiation Among *Puccinia psidii* Populations from South America and Hawaii

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Kadooka<sup>6</sup>, and R.D. Hauff<sup>7</sup>

### ABSTRACT

*Puccinia psidii* is the cause of rust disease of many host species in the Myrtaceae family, including guava (*Psidium* spp.), eucalypt (*Eucalyptus* spp.), rose apple (*Syzygium jambos*), and 'ohi'a (*Metrosideros polymorpha*). First reported in 1884 on guava in Brazil, the rust has since been detected in South America (Argentina, Brazil, Colombia, Paraguay, Uruguay, and Venezuela), Central America (Costa Rica and Panama), Caribbean (Cuba, Dominica, Dominican Republic, Jamaica, Puerto Rico, Trinidad and Tobago, and Virgin Islands), Mexico, USA (Florida, California, and Hawaii), Japan, and most recently it was potentially found in Australia. Of present concern is the recent introduction of the pathogen to Hawaii, where it infects an endemic tree species known as 'ohi'a, the dominant tree species in Hawaii's remaining native forests. The rust also poses serious threats to several Myrtaceae species including Eucalyptus, a genus native to Australia and planted extensively in numerous tropical and subtropical countries. Despite the potential threat toward many forest ecosystems worldwide and the expanding geographic range of this disease, little is known about the genetic structure of *P. psidii* populations, migratory routes and sources of introductions. To determine genetic structure

of pathogen populations in the putative center of origin, approximately 150 single-pustule isolates of *P. psidii* have been collected from diverse host species and locations in Brazil. These isolates were scored for variation at 10 microsatellite loci. Additional isolates have been collected from Hawaii, California, Paraguay, and Uruguay; and collaborators are currently being sought to obtain isolates from other regions. Collections from reputedly introduced populations will facilitate inferences about the spread of this rust pathogen throughout the world. Preliminary results for Brazil indicate that: 1) considerable genetic diversity is present; 2) species of hosts strongly influence population structure; and 3) distinct multilocus genotypes are uniquely associated with specific hosts across diverse geographic locations. In contrast, all 49 rust isolates collected from five different hosts in Hawaii (Oahu, Maui, Kauai, and Hawaii - "Big Island") share a unique, multilocus genotype, indicating a recent introduction of a single rust genotype. This information will help identify rust races that pose threats to global populations of Myrtaceae and help prevent their introduction into new regions. For example, none of the rust genotypes in Brazil are known to occur in Hawaii. At present, it seems prudent to focus on avoiding the introduction of any novel genotypes to new regions with populations of known hosts. Furthermore, potential recombination of pathogen genotypes could generate new virulent races with unpredictable consequences. Population genetics and molecular characterization of *P. psidii* isolates collected from around the world will provide critical information on pathways of spread and assessment of future risk.

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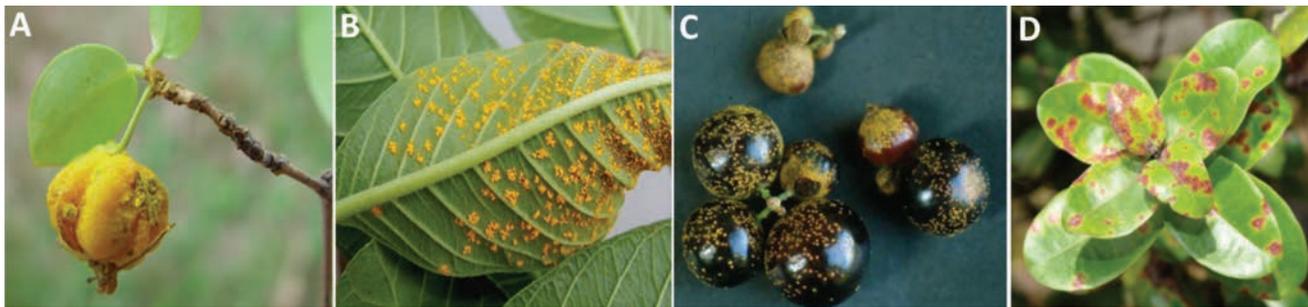
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### BACKGROUND

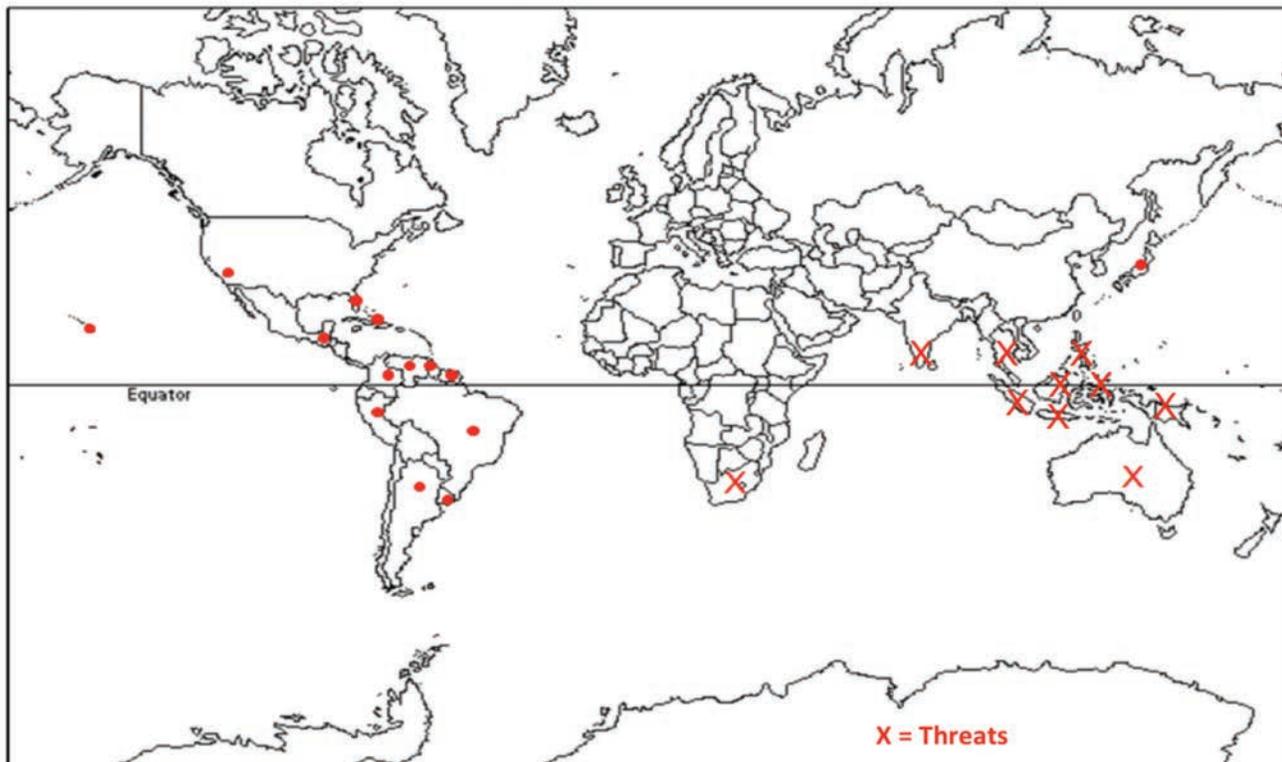
*Puccinia psidii* causes rust disease on many host species in the Myrtaceae family (Farr and Rossman 2010), including guava, Surinam cherry (*Eugenia uniflora*), and 'ohi'a (figure 1). First reported in 1884 on guava in south Brazil (Maclachlan 1938), the rust has since been detected in other regions of South America (Ferreira 1981; Lindquist 1982; Kern et al. 1928; Lindquist 1982; Chardon and Toro 1934),

Central America (Bernt 2004; Perdomo-Sanches and Piepenbring 2008), Caribbean (Spaulding 1961; Baker and Dale 1948; Kern et al. 1933; Dale 1955; Stevenson 1975), Mexico (Gallegos and Cummins, 1981), USA (Florida, Rayachhertry et al. 1997; California, Mellano 2006; and Hawaii; Uchida et al. 2006), and most recently in Japan (Kawanishi et al. 2009). Based on *P. psidii*'s widespread geographic distribution and its large host range, this rust pathogen is considered a serious threat to several hosts in the Myrtaceae family in numerous tropical and subtropical countries (figure 2) (Tommerup et al. 2003; Glen et al. 2007). Of special note is rust that was found infecting Myrtaceae species in Australia and reported as *Uredo rangellii*, based on the tomsure found in urediniospores surface (Carnegie et al. 2010). However, DNA sequence data did not differentiate the rust from

*P. psidii*. Studies are needed to elucidate the disease etiology. In Hawaii, *P. psidii* infects an endemic tree species known as 'ohi'a, the dominant tree species in Hawaii's native forests that provides habitat for much of the Hawaiian flora and fauna (Uchida et al. 2006; Loope 2010). This disease has also been highly damaging to the last remaining populations of endemic *Eugenia koolauensis*, a federally listed endangered species. Of present concern is the potential introduction of new pathogen genotypes into Hawaiian Islands (Loope 2010). However, despite the potential threats to many forest ecosystems worldwide and the expanding geographic range of this disease, little is known about the genetic structure of pathogen populations, migratory routes, and sources of introductions.



**Figure 1**—Some hosts of the rust caused by *Puccinia psidii*. A. *Eugenia uniflora* (Surinam cherry); B. *Psidium guajava* (guava); C. *Myrciaria cauliflora* (jaboticaba); D. *Metrosideros polymorpha* ('ohi'a) (From Alfenas et al., 2009).



**Figure 2**—Current (red dot) and potential (red x) geographic distribution of *Puccinia psidii*.

## OBJECTIVE

The main goal of this study is to provide baseline information to identify rust genotypes that pose threats to global populations of Myrtaceae. This information can be used to help prevent the introduction of critical rust genotypes into new regions and increase the efficacy in selecting rust-resistant host individuals.

## METHODS

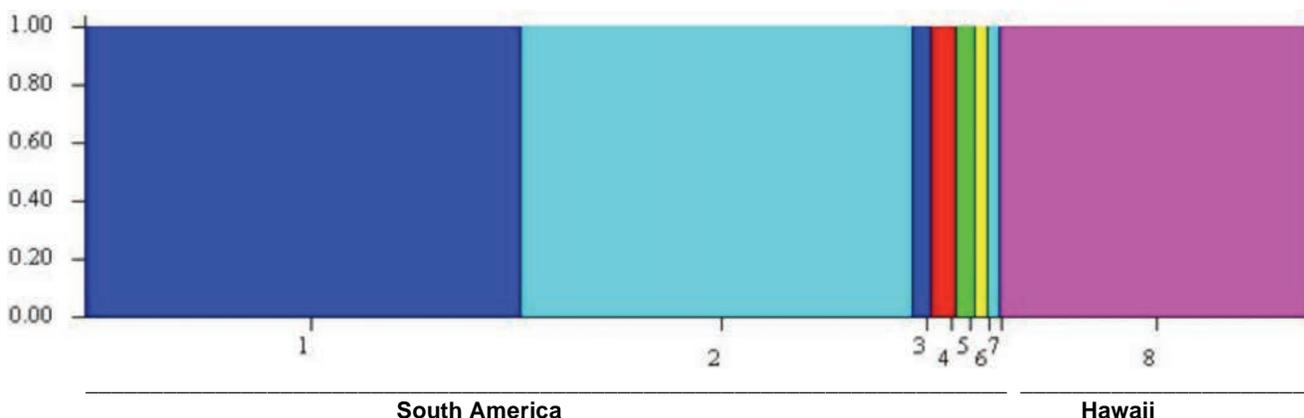
To determine the genetic structure of pathogen populations, approximately 150 single-uredinial isolates of *P. psidii* were collected from diverse host species and locations in Brazil, Uruguay, and Paraguay. DNA was extracted from each isolate and subsequently scored for variation at 10 microsatellite loci (Zhong et al. 2007). Additionally, 49 isolates were collected on *Metrosideros polymorpha*, *M. excelsa*, *Eugenia koolauensis*, *Syzygium jambos* and *Rhodomytrus tomentosa* from four Hawaiian Islands (Oahu, Maui, Kauai, and Hawaii - "Big Island") and, so far, scored for variation at 7 microsatellite loci (Zhong et al. 2007). Collaborators are currently being sought to sample isolates from other global regions, such as California, Florida, Puerto Rico, Central America, Colombia, Venezuela, and Japan. Collections from these diverse geographic locations will facilitate inferences about the spread of this rust pathogen throughout the world.

## RESULTS

Preliminary results indicate that host species strongly influences *P. psidii* population structure in South America (figure 3). Distinct multilocus genotypes were uniquely associated with specific hosts across diverse geographic locations in South America. Higher genetic diversity of *P. psidii* was detected in Brazil compared to Hawaii, which is consistent with the hypothesis that Brazil is the evolutionary origin of this pathogen. However, additional samples from diverse global locations are needed to confirm this hypothesis. In contrast, all of the 49 Hawaiian isolates had the same multilocus genotype across the three islands despite being sampled from six different species of hosts (figure 3). The low genetic diversity in Hawaii is consistent with a recent introduction of a single rust genotype into Hawaii. Four alleles at three loci in the Hawaiian sample were not found in the South American sample (private alleles, sensu Slatkin 1985) indicating that the hosts sampled in South America were unlikely to have been the source of the introduction to Hawaii.

## PRELIMINARY CONCLUSIONS

Structure of *P. psidii* populations in South America is strongly associated with species of hosts. Hosts may have selected particular *P. psidii* genotypes in South America followed by little or no gene flow among host-adapted pathogen genotypes.



**Figure 3**—STRUCTURE analysis showing posterior probability (y-axis) of each isolate belonging to one of six genetic clusters (K = 6), shown in different colors. Each isolate is represented by a vertical colored bar on the x-axis, where bars of the same color represent isolates assigned to the same genetic cluster. Thus, the width of the colored bar represents the number of isolates assigned to the genetic cluster. Numbers under genetic clusters are as follows: 1 = *Eucalyptus* spp.; 2 = *Psidium guajava*; 3 = *Syzygium jambos*; 4= *Syzygium cumini*; 5 = *Myciaria cauliflora*; 6 = *Eugenia uniflora*; 7 = *Psidium araca*; and 8 = *Metrosideros polymorpha* + *Metrosideros excelsa* + *Eugenia koolauensis* + *Syzygium jambos* + *Rhodomytrus tomentosa*.

*Puccinia psidii* populations in South America are not geographically structured. Although sample numbers are low, several host species in South America appear to be infected by a unique genotype of *P. psidii* that only infects that host species. This could represent recent host shifts by *P. psidii* in South America and the existence of host-specific genotypes of *P. psidii* in South America, which could also indicate the early stages of speciation in *P. psidii*.

Hawaiian isolates were all of the same multilocus genotype, regardless of host of isolation or geographic location. Furthermore, this genotype is not found in South America suggesting that South America is not the source of the introduction to Hawaii.

The apparent wide host range of *P. psidii* in Hawaii contrasts sharply with the high fidelity and narrow host range that is observed in South America. The wide host range of the *P. psidii* genotype found in Hawaii may indicate that this genotype represents a serious invasive pathogen risk if introduced to other global locations. Inoculation tests are needed to better understand the host range of the Hawaiian genotype of *P. psidii*.

The introduction of any new *P. psidii* genotypes into Hawaii represents a potential invasive pathogen risk for Myrtaceous trees. Furthermore, potential recombination among genotypes could create new genotypes with unknown risks. For these reasons, it seems prudent to avoid the introductions of new *P. psidii* genotypes into Hawaii and elsewhere, and studies should continue to better assess invasive risks posed by diverse genotypes.

## ACKNOWLEDGMENTS

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## Preliminary Report on the Ecology of *Armillaria* in the East Cascades of Oregon

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### ABSTRACT

As part of a larger effort to assess the distribution and ecology of *Armillaria* species throughout western North America, we present preliminary survey results for the East Cascades of Oregon. Surveys and sampling were conducted on 260 0.04-ha plots, which were randomly located across diverse environments and geographic locations. Using DNA-based techniques for the identification of *Armillaria* spp., we identified three genetically-distinct species groups that comprised the ca. 450+ *Armillaria* samples. The association of *Armillaria* species groups with habitat is summarized based on detailed vegetation data. When we understand habitats in which *Armillaria* species can potentially occur, forest management prescriptions can be developed to improve forest health at the stand level by reducing impacts of *Armillaria* root disease. These data also provide critical baseline information for evaluating larger-scale impacts in forest ecosystems as trees are subjected to climate-change induced stress.

### INTRODUCTION

The impacts of *Armillaria* on forested ecosystems are expected to generally increase under climate change (Sturrock et al. 2011, Kliejunas et al. 2009), but methods are needed to predict impacts at local and regional levels. Techniques are available to estimate climate data across the landscape. Climate data coupled with powerful statistical methods, such as maximum entropy (Maxent), can determine probable suitable climate space and potential distribution for

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a species, even with minimal presence data (Phillips et al. 2006, Pearson et al. 2007). Predicted potential distributions of both pathogens and host species under current climatic conditions can then used to predict potential future distributions, based on various climate-change scenarios (Rehfeldt et al. 2006, Klopfenstein et al. 2009).

Previous studies of the ecology of *Armillaria* root disease in the inland northwestern U.S.A. and Utah demonstrate that *Armillaria* distribution can be characterized with environmental parameters, exemplified by plant species coverages as indicators of environmental conditions such as climate (e.g., temperature and moisture) (McDonald et al. 1987, McDonald 1998). This method is well suited for predicting *Armillaria* presence at the stand level, which can perhaps be adapted for assessment at larger scales. One objective of the current study is to determine the relationship of plant coverages (vegetation subseries/potential vegetation groups) to the distribution of *Armillaria* species in the East Cascades of Oregon. A larger goal of this study is to determine the potential influence of climate and climate change on *Armillaria* spp. distribution, as reflected by plant coverages.

### METHODS

Two hundred and sixty 0.04-ha plots (FY2007-FY2010) were established across climatically diverse sites in Oregon's East Cascades. (e.g., Warm Springs Indian Reservation, Fremont-Winema NF, Deschutes NF, Mt Hood NF, and Ochoco NF). The majority of plots were selected based on climatic diversity and spatial separation across gridded sections (ca. every 7'30" N and 1° 52'30" W). For each tree and shrub species present, primary root systems and butts of three individuals were thoroughly examined, and samples (i.e., rhizomorphs, mycelial fans, rotten wood) of *Armillaria* spp. were collected along with precise location. Isolates were established in culture then identified with DNA-based techniques (Kim et al. 2006).



**Figure 1**—Cedar Hemlock/Moist Herb potential vegetation group, a highly suitable site for *Armillaria*.

The plant coverages on each plot were classified into vegetation subseries/potential vegetation groups. Each subseries/group represents several ecologically similar plant associations/habitat types based on the percent coverage of key indicator plants (McDonald 1998).

## RESULTS

*Armillaria* spp. were found on 146 plots (56.1 percent) of the 260 plots surveyed. *Armillaria solidipes* (= *A. ostoyae*) was found on 33 plots (12.7 percent), North American Biological Species (NABS) III-V-VII (*A. calvescens*-*A. sinapina*-*A. gallica* complex) was found on 73 plots (28.1 percent), and NABS X (including NABS X hybrids with NABS III/V/VII) was found on 67 plots (25.8 percent).

## KEY FINDINGS

(1) *Armillaria* spp. are common in the East Cascades of Oregon, occurring on 56 percent of 260 sampled plots. *Armillaria* incidence increases to 73 percent, when 95 of the driest, wettest, warmest, and coolest plots are excluded (table 1).

(2) Among the three *Armillaria* species groups identified, NABS X group was collected from the greatest range of climates as inferred from vegetation subseries. *Armillaria solidipes* group was rarely found on the warmest sites and the NABS III-V-VII (*A. calvescens*-*A. sinapina*-*A. gallica*) complex was rarely found on the coolest sites (tables 2-4).

## DISCUSSION

Ecological assessments of *Armillaria* are critical to address forest management needs. Habitat-based predictions of *Armillaria* distributions are useful for stand-level *Armillaria* risk assessments, such as the *Armillaria* Response Tool (ART) (McDonald et al. 2005). These stand-level indicators can be combined with climate-based predictions of *Armillaria* distributions at a much larger scale (Klopfenstein et al. 2009), which can be adapted for future predictions under various climate-change scenarios. Future predictions of both pathogens and hosts can provide information for National Insect and Disease Risk Maps. Climate-based predictions will continue to improve as higher resolution grids are developed that include influences of slope, aspect, and soil types. In addition, molecular techniques will also continue to improve species identification and allow classification of genetic diversity at the subspecies level, which will allow further refinements in predictions of environmental influences (Kim et al. 2006, Hanna et al. 2007, Ross-Davis et al. this volume).

**Table 1**—Occurrence of all *Armillaria* spp. in the East Cascades of Oregon.

	Dry Grass	Dry Shrub	Dry Herb	Moist Herb	Wet Herb	Wet Forb	West Shrub
Pinyon Juniper	0/1						
Ponderosa pine	4/42	3/15	0/0				
Douglas-fir	0/0	0/3	5/7	0/0			
Cool pine	0/5	3/3	1/1	0/0			
Cedar Hemlock			0/1	2/2	0/0	0/0	0/0
Cool fir	0/0	72/105	35/46	7/7	0/0	0/0	
Cold fir	1/2	5/9	6/7	2/3	0/1	0/0	0/0

**Table 2**—Occurrence of *Armillaria solidipes* in the East Cascades of Oregon.

	Dry Grass	Dry Shrub	Dry Herb	Moist Herb	Wet Herb	Wet Forb	West Shrub
Pinyon Juniper	0/1						
Ponderosa pine	1/42	0/15	0/0				
Douglas-fir	0/0	0/3	0/7	0/0			
Cool pine	0/5	1/3	1/1	0/0			
Cedar Hemlock			0/1	1/2	0/0	0/0	0/0
Cool fir	0/0	10/105	12/46	2/7	0/0	0/0	
Cold fir	1/2	3/9	1/7	0/3	0/1	0/0	0/0

**Table 3**—Occurrence of North American Biological Species (NABS) III-V-VII (*Armillaria calvescens*, *A. sinapina*, *A. gallica*) in the East Cascades of Oregon.

	Dry Grass	Dry Shrub	Dry Herb	Moist Herb	Wet Herb	Wet Forb	West Shrub
Pinyon Juniper	0/1						
Ponderosa pine	1/42	2/15	0/0				
Douglas-fir	0/0	0/3	4/7	0/0			
Cool pine	0/5	0/3	0/1	0/0			
Cedar Hemlock			0/1	1/2	0/0	0/0	0/0
Cool fir	0/0	44/105	19/46	1/7	0/0	0/0	
Cold fir	0/2	0/9	1/7	0/3	0/1	0/0	0/0

**Table 4**—Occurrence of North American Biological Species (NABS) X or hybrids of NABS X with NABS III-V-VII (*Armillaria calvescens*, *A. sinapina*, *A. gallica*).

	Dry Grass	Dry Shrub	Dry Herb	Moist Herb	Wet Herb	Wet Forb	West Shrub
Pinyon Juniper	0/1						
Ponderosa pine	2/42	1/15	0/0				
Douglas-fir	0/0	0/3	1/7	0/0			
Cool pine	0/5	2/3	0/1	0/0			
Cedar Hemlock			0/1	1/2	0/0	0/0	0/0
Cool fir	0/0	29/105	16/46	6/7	0/0	0/0	
Cold fir	0/2	2/9	5/7	2/3	0/1	0/0	0/0

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## DNA-based Identification and Phylogeny of North American *Armillaria* Species

Amy L. Ross-Davis<sup>1</sup>, John W. Hanna<sup>2</sup>, and Ned B. Klopfenstein<sup>3</sup>

### ABSTRACT

Because *Armillaria* species display different ecological behaviors across diverse forest ecosystems, it is critical to identify *Armillaria* species accurately for any assessment of forest health. To further develop DNA-based identification methods, partial sequences of the translation elongation factor-1 alpha (EF-1 $\alpha$ ) gene were used to examine the phylogenetic relationship among 30 isolates representing the ten North American *Armillaria* species: *A. solidipes* (= *A. ostoyae*), *A. gemina*, *A. calvescens*, *A. sinapina*, *A. mellea*, *A. gallica*, *A. nabsnona*, North American Biological Species X, *A. cepistipes*, and *A. tabescens*. The phylogenetic relationship inferred using the Maximum Likelihood method revealed separation of all ten North American *Armillaria* species. Thus, our preliminary results indicate the EF-1 $\alpha$  gene could serve as a potential diagnostic tool for distinguishing among currently recognized North American Biological Species of *Armillaria*.

### INTRODUCTION

The *Armillaria* (Fries: Fries) Staude genus is widely distributed across North America, where it displays diverse ecological behaviors ranging from beneficial saprobe to virulent root/butt disease pathogen. Currently, ten North American Biological Species (NABS) were identified through the work of Anderson and Ullrich 1979, Morrison et al. 1985, Anderson 1986, and Bérubé and Dessureault 1989, and are named as follows: (1) NABS I = *Armillaria solidipes* Peck (= *A. ostoyae* (Romagn.) Herink), (2) NABS II = *A. gemina* Bérubé and Dessureault, (3)

NABS III = *A. calvescens* Bérubé and Dessureault, (4) NABS (IV) V = *A. sinapina* Bérubé and Dessureault, (5) NABS VI (VIII) = *A. mellea* (Vahl: Fries) Kummer, (6) NABS VII = *A. gallica* Marxmüller and Romagnesi (= *A. bulbosa* (Barla) Kile and Watling or *A. lutea* Gillet), (7) NABS IX = *A. nabsnona* Volk and Burdsall, (8) NABS X is yet unnamed, (9) NABS XI = *A. cepistipes* Velenosky, and (10) *A. tabescens* (formerly known as *Clitocybe tabescens* (Scop.) Bres).

Because *Armillaria* species can display great differences in pathogenicity, it is critical to identify specific *Armillaria* spp. that occur on a site to assess disease risk. For example, *A. solidipes* and *A. mellea* are generally considered strongly pathogenic; whereas, other species (e.g., *A. sinapina*, *A. gallica*, NABS X, *A. cepistipes*) are generally considered less pathogenic in many situations (Shaw and Kile 1991). Unfortunately, many *Armillaria* spp. are difficult to identify based on morphology alone. Thus, DNA-based diagnostic tools are sought to aid in species identification for assessments of disease risks and other purposes. To examine phylogenetic relationships among 15 species of *Armillaria* from diverse global regions and hosts, Maphosa et al. (2006) showed that partial sequences of the translation elongation factor-1 alpha (EF-1 $\alpha$ ) were useful to separate *Armillaria* spp. More recently, Hasegawa et al. (2010) also showed the usefulness of the same DNA region to explore genetic relationships among eight Japanese *Armillaria* species.

The objectives of this study were to 1) examine phylogenetic relationships among the ten North American *Armillaria* species using partial DNA sequence data of EF-1 $\alpha$ ; and 2) assess whether DNA sequence differences have potential use as a diagnostic tool to distinguish among the North American *Armillaria* species, particularly those that are morphologically similar and/or closely related.

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## MATERIALS AND METHODS

Three isolates of each North American *Armillaria* species were included in this study (figure 1). Isolates were incubated in the dark at 21°C for approximately 2.5 weeks on 0.20 µm-pore, nylon membranes (Millipore, County Cork, Ireland) atop 3 percent malt agar. DNA was extracted from isolates using the DNeasy Plant Mini Kit (Quiagen Inc., Valencia, CA, USA) according to the manufacturer's protocol with an additional wash of 100 percent ethanol prior to elution. DNA was quantified using a NanoDrop™2000 (Wilmington, DE, USA) and stored at -20°C.

### PCRAmplification and DNA Sequencing

To amplify the EF-1α gene, each 50-µl polymerase chain reaction mixture contained 5 µl (equating to 35 to 520 ng) template DNA, omitted for negative control, 2.5 U Taq polymerase (Applied Biosystems, Inc, Foster City, CA, USA), PCR reaction buffer (supplied with Taq enzyme), 4 mM MgCl<sub>2</sub>, 200 µM dNTPs, and 0.5 µM of each primer (Forward: CGT GAC TTC ATC AAG AAC ATG and Reverse: CCG ATC TTG TAG ACG TCC TG (Wendland and Kothe 1997 in Kausarud and Schumacher 2001)). Thermal cycler conditions were as follows: 94°C for 2 min; 30 cycles of 94°C for 30 s, 57°C for 30 s, 72 °C for 1 min and 30 s; and 72 °C for 7 min. Sequences were edited and aligned manually with BioEdit software (Hall 1999). To minimize errors, both forward and reverse directions of the region were edited and aligned and two researchers performed all sequence editing independently. Polymorphisms were coded with the IUPAC codes for ambiguous nucleotides.

### Phylogenetic Analysis

The phylogenetic relationship was inferred using the Maximum Likelihood method based on the Tamura-Nei model. The 50 percent majority-rule, consensus tree inferred from 1000-bootstrap replicates was taken to represent the relationship among species. The percentage of replicate trees in which the associated species clustered together in the bootstrap test is shown next to the branches. A discrete Gamma distribution was used to model evolutionary rate differences among sites (four categories [+G, parameter = 1.0993]). The analysis involved 34 nucleotide sequences, since two different copies of the gene were present in four of the 30 isolates. All positions containing gaps and missing data were eliminated. There were 449 positions in the final dataset. Analyses were conducted in MEGA (Tamura et al. 2007).

## RESULTS

Phylogenetic analysis of the partial EF-1α gene revealed five major clades: 1) a basal clade of *A. tabescens*, 2) a clade containing *A. gemina*, *A. solidipes* (= *A. ostoyae*), *A. mellea*, and *A. sinapina*, 3) a clade containing *A. nabsnona* and *A. cepistipes*, 4) a clade containing NABS X, and 5) a clade containing *A. calvescens* and *A. gallica* (figure 1). Partial sequence data for the EF-1α gene delineated all ten North American species of *Armillaria* into separate clades or subclades, but *A. gallica* isolates were not comprised within a single subclade.

**CLADE 1:** The basal clade is comprised entirely of *A. tabescens*, the only exannulate species of *Armillaria* in North America. It is pathogenic on hardwoods in eastern North America, particularly on oaks and fruit trees in the Southeast.

**CLADE 2:** This clade consists of the aggressive pathogens *A. solidipes* (= *A. ostoyae*) and *A. mellea* as well as *A. sinapina*, which has been associated with *A. solidipes* disease centers as a secondary invader (Dettman and van der Kamp 2001), and *A. gemina*, which is similar to *A. solidipes* (Bérubé and Dessureault 1989; Kim et al. 2006).

**CLADE 3:** *A. nabsnona* and *A. cepistipes*, saprobes or occasionally weak pathogens found on hardwoods in the west, comprise the third clade.

**CLADE 4:** The unnamed NABS X, known only from British Columbia, Washington, Oregon, California and Idaho, comprises the fourth clade.

**CLADE 5:** This final clade is comprised of the morphologically indistinguishable species *A. calvescens* and *A. gallica*. Both species are found east of the Rockies primarily on hardwoods, but *A. gallica* is also found west of the Rockies. *Armillaria gallica* does not form a well-defined subclade. These data, as well as other sequence and AFLP data (Kim et al. 2006, Hanna et al. 2007), suggest that *A. gallica* is a genetically diverse species that may comprise multiple genetically distinct groups.

## DISCUSSION

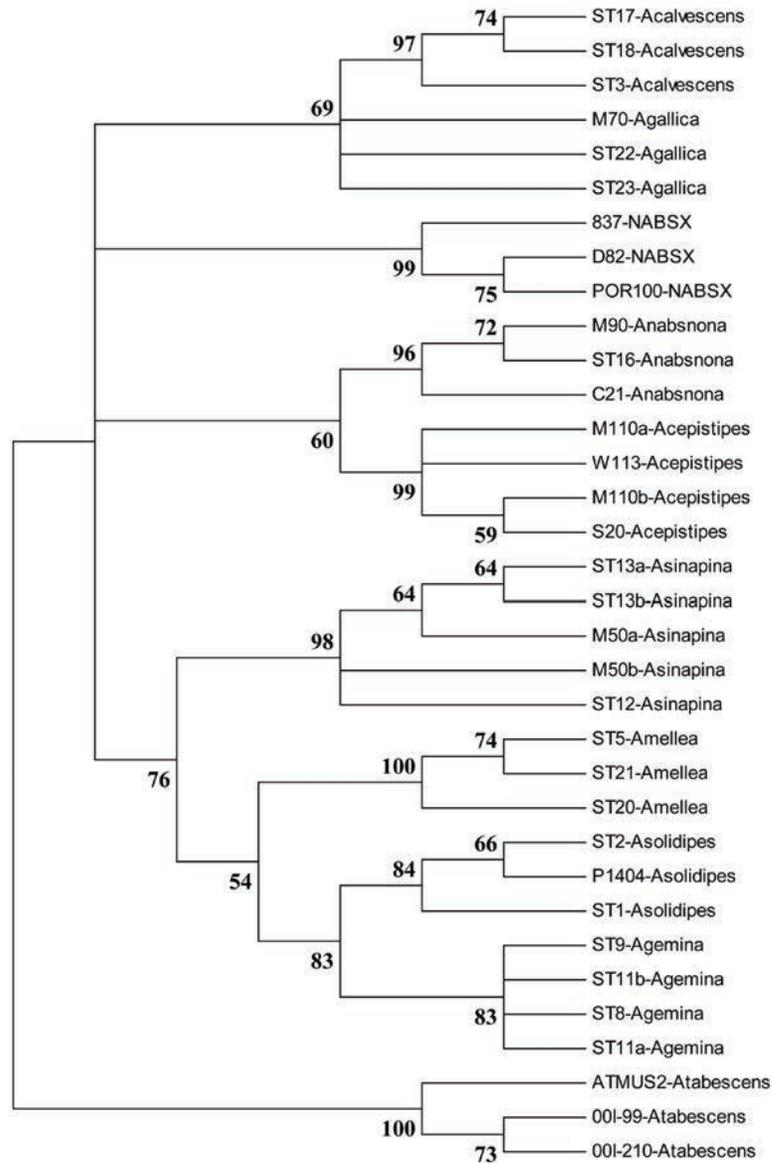
DNA-sequence-based identification of *Armillaria* is an essential step to monitor and predict *Armillaria* root disease in forest ecosystems. Previously, Kim et al. (2006) reported that rDNA sequence data are not sufficient to confirm species identification among the

closely related species *A. calvescens*, *A. sinapina*, *A. gallica*, and *A. cepistipes*. Other molecular characteristics, such as DNA content can help to identify some species, but facilities to determine DNA content are not widely available (Kim et al. 2000).

The results from this study show that the single-copy EF-1 $\alpha$  gene could potentially serve as a diagnostic tool to distinguish among all ten North American species of *Armillaria*. However, further work associated with morphological characterization and

mating/pairing tests of more isolates is required for verification of the utility of this region as a diagnostic tool. The region may also be useful for phylogenetic studies, particularly when combined with sequence data from additional genes.

This work is part of an on-going study to 1) examine relationships among the North American *Armillaria* species using sequence data from a variety of loci; and 2) confirm the validity of sequence for use as a diagnostic tool to distinguish among species.



**Figure 1**—50 percent majority-rule, consensus phylogenetic tree based on partial EF-1 $\alpha$  sequences of ten North American species of *Armillaria*.

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# Committee Reports



Mount Robson (Bilang) upstream of Emporor Falls BC. Wikimedia Commons



# Nursery Committee Report

## Acting Committee Chair – Will Littke

Attendees: Will Littke, John Browning, Ned Klopfenstein, Bob Edmonds, and James Blodgett

### OVERVIEW

Nursery regeneration pathology research is actively engaged in many facets of disease control and pathogen identification. Although Federal nurseries have reduced their growing stock, State and private nurseries continue to grow in excess of 1.3 billion conifer and hardwood seedlings annually across North America. Nursery pathology is undergoing the same taxonomic transformation as forest pathology with development of an implementation of molecular diagnostic tools. WIFDWC member scientists have been at the forefront of this development and testing process and application to both applied and basic pathology research.

### DISCUSSION AND ON-GOING RESEARCH TOPICS

**Forest Nursery Pest Handbook:** The chapter revisions have been submitted (thanks to all the volunteer authors) and the coordinators have now begun reviewing them. (Katy Mallams, Michelle Cram, Pathologist R-8 FHP, and Michelle Frank, Entomologist and Pesticide Specialist NA FHP). We hope to have the complete package ready to send to the Washington Office by the end of February so it can be published in September. I'm continuing to assist the federal seed orchards in SW Oregon prevent the spread of *Phytophthora* inoculum between facilities. I've been examining and testing Douglas-fir and sugar pine rootstock prior to shipping, and helped them set up standards for keeping growing areas free of contamination.

**Alternative's to MB Fumigation Metrics:** Jerry Weiland (USDA/ARS), Anna Leon (WSU), Bob Edmonds (UW), Will Littke and John Browning (Weyerhaeuser) presented data on the 2008 USDA/ARS West Wide Alternative Fumigation project. This USDA/ARS funded study was the first replicated fumigant trial of methyl iodide and DMDS coupled with chloropicrin in the PNW. *Fusarium* commune molecular markers (developed by Jane Stewart, Mee-

Sook Kim and Ned Klopfenstein) were used to track pathogen occurrence in three PNW bare-root nurseries. More details on this study can be found in the poster presentation. A final report can be obtained from (Jerry.Weiland@ARS.USDA.GOV)

A follow-up 2010 ARS fumigation study was installed (August 2010) in two Oregon nurseries to evaluate efficacy of low volume rate fumigants (25-foot buffers) using totally impermeable tarps. These treatments will evaluate the efficacy of treatments designed to meet the 2009 EPA guidelines for safety buffers zones around bare-root nurseries. Soil fumigant treatments include:

Methyl Bromide + Chloropicrin	250 lbs/A (50:50)	TIF
Pic-Chlor 60 (Chloropicrin+ 1,3-D)	339 lbs/A (60:40)	TIF
Metam Sodium + Chloropicrin	27 gal/200 lbs/A	TIF

**Control of *Cylindrocarpon* (Bare-root and Containers):** Bob Edmonds (UW), Nate Johnson (UW graduate student), Will Littke, and John Browning (Weyerhaeuser RandD) received funding from Washington Department of Agriculture for a trial to determine the efficacy of bio-control agents (*Trichoderma*, *Gliocladium*, *Bacillus*, and *Streptomyces*) and fungicide control of *Cylindrocarpon*. The occurrence and pathogenicity of *C. didymum* and *C. destructans* root pathogens in crops of Douglas-fir seedlings is being evaluated. Ned Klopfenstein (RMRS, Moscow, Idaho) is performing some initial molecular diagnostics on these two species of *Cylindrocarpon*. (will.littke@weyerhaeuser.com for project workplan)

***Fusarium* Nursery Research Project:** Anna Leon (Ph.D. student Washington State University Puyallup Research and Extension; (PI): Dr. Gary Chastagner. Objectives: (1) Test and verify the efficacy of a real-time PCR technique being developed by Dr. Lynne Carpenter-Boggs' lab for the identification and quantification of *Fusarium oxysporum* and *Fusarium commune* from Douglas-fir nursery soils; (2) Perform pathogenicity tests on isolates of *Fusarium* sp. to

determine their relative virulence on Douglas-fir; and (3) Compare genetic sequences of *F. oxysporum* and *F. commune* isolated from Douglas-fir nurseries and Christmas tree farms from locations across the United States to analyze potential methods of disease dispersal nationwide.

**Fusarium Biocontrol Project:** Ned Klopfenstein (RMRS, Moscow, Idaho), Mee-Sook Kim (RMRS, Moscow, Idaho and Kookmin University, Seoul, Korea) and Kas Dumroese (Plant Physiologist, RMRS, Moscow, Idaho), with the help of Bob James (retired Plant Pathologist, Forest Health Protection, Coeur d'Alene, Idaho) and Amy Ross-Davis (Post-Doctoral volunteer, RMRS, Moscow, Idaho), are working to evaluate potential efficacy of *Fusarium oxysporum* as a biocontrol agent for container-grown Douglas-fir seedlings against the pathogenic *Fusarium commune*. Mee-Sook and Kas are also working with Jane Stewart (PhD candidate, Department of Plant Pathology, Washington State University, Pullman, Washington) to further characterize the pathogenic *Fusarium commune* in conifer nurseries.

**Bessey Nursery Pathology Report:** James T Blodgett, USDA Forest Service, Forest Health Protection, Rapid City, SD. Diseases of conifer seedlings at Bessey Nursery include minor persistent problems with *Diplodia pinea* (*Sphaeropsis sapinea*), *Fusarium*, and *Phytophthora* or *Pythium*. From time-to-time *Phomopsis* can be an issue. Diseases of hardwood seedlings include some black-knot and shot hole in *Prunus*; and occasional foliage diseases including Anthracnose; powdery mildews; *Melampsora* rust on cottonwood; rusts on *Ribes*; and

*Gymnosporangium* rust ("cedar apple rust") on *Amelanchier*, *Malus*, and *Crataegus*. The nursery receives and stores all types of seed (hardwood and conifer), and I was involved a little with limber and whitebark pine seed collections. I tested some container-grown pine for resistance to *Diplodia* with Bill Schaupp. Seedlings of Austrian, jack, lodgepole, ponderosa, red, Scots, and jack pines were wounded and inoculated with water agar plugs colonized by four *D. pinea* isolates. These isolates were obtained from ponderosa pine. One isolate from a symptomatic host and one isolate from an asymptomatic host were collected in Nebraska and South Dakota. Hosts varied significantly ( $P < 0.001$ ) in their responses to inoculation from the isolates. Austrian pine was the most susceptible followed by lodgepole, red, Scots, and jack pines, based on symptom severity (distance from the inoculation site at which necrotic needles were present). Ponderosa pine, the original host of all four isolates, was the least susceptible to all isolates.



## Foliage and Twig Disease Committee Meeting

### Committee Chair - Harry Kope

#### **Jim Walla of North Dakota State University:**

*Stigmina lautii*, which has been found on spruce in eastern and central North America. Proof of pathogenicity of *Stigmina* is lacking, so it could be a pathogen, an endophyte, or a mycoparasite, but appearances are of a pathogen. Black fruiting bodies fill the stomata of the needles. Needles become necrotic in the second growing season.

#### **Bill Jacobi of Colorado State University:**

Red Oak canker, which has been detected on a very small population of trees (5) in Boulder Colorado. The problem appears to be bacterial in origin.

#### **Bruce Moltzen of USDA Forest Service, Arlington, VA:**

Thousand cankers disease occurs on black walnut (*Juglans nigra*) is caused by the fungus *Geosmithia* sp, vectored by the walnut twig beetle (*Pityophthorus juglandis*). The insect bores beneath the bark and the vectored *Geosmithia* causes a large number of small cankers to form under the bark in the phloem. More information can be found at <http://www.thousandcankerdisease.com/>

#### **Stefan Zeglen of the British Columbia Ministry of Forests and Range:**

*Septoria musiva*, a poplar disease introduced from eastern North America into BC. The pathogen was detected and identified in 2006 and was found causing stem cankers at multiple clonal poplar sites. Leaf samples have been collected from *P. trichocarpa* trees

along the Fraser River corridor and around positive finds on clonal poplar sites. Samples have tested positive for *S. musiva* from leaves collected from both clonal poplar and from native *Populus* trees. More work will be done determining the range of the disease on *P. trichocarpa* in southern BC along the Fraser River.

#### **Michael Murray and Alex Woods of the British Columbia Ministry of Forests and Range:**

Birch dieback is widespread throughout British Columbia and occurring to all species of *Betula*. A cause of the problem was suggested to be a complex involving climatic stress (drought) and asymptomatic endophytic fungi. The problem continues to be monitored, albeit, in a limited way.





# Root Disease Committee Report

## Chair - Michelle Cleary

The Root Disease Committee met for lunch on Tuesday, October 5, 2010. There were 43 persons in attendance.

### AGENDA ITEMS

Thanks! Michelle welcomed the opportunity to take on chairperson responsibility for the Root Disease Committee. I thank Brennan Ferguson for his enthusiasm and leadership of this committee over the last 5 years.

### ANNOUNCEMENTS

The 13th International Conference on Root and Butt Rot of Forest Trees is in Italy September 4-11, 2011 in Firenze (Florence) and Trento. A pre-conference field trip outside of Rome will look at *Heterobasidion irregulare* (the N.A. P-type) on stone pine introduced from the eastern US during military activities in WWII. More conference details are available at <http://www.iufro.org/science/divisions/division-7/70000/70200/70201/activities/>.

### PRESENTATIONS

**Dr. Jonas Rönnerberg, Associate Professor and Vice Dean, Faculty of Forest Sciences, SLU, Southern Swedish Forest Research Centre, Alnarp, Sweden**

Jonas Rönnerberg presented information on silvicultural management of *Heterobasidion* species in Scandinavia. Estimated losses attributable to growth reduction and degradation of wood are about €120 million annually in Sweden and Finland. Both *H. annosum* and *H. parviporum* occur together in forest stands, especially in the southern part of Sweden, whereas only *H. parviporum* is present in the north. In general, disease levels in plantation forests increase with successive rotations and with incremental silviculture activities. Options to mitigate potential damage caused by *Heterobasidion* spp. include reducing the number of thinnings, harvesting during low-risk periods, choice of tree species, and treating stumps with the biocontrol agent *Phlebiopsis gigantea*. Stump treatment must be carried out in a proper way and follow-ups are crucial to maintain treatment

quality. Hence, forest practitioners can potentially influence the spread of the disease and therefore reduce losses in forest stands by implementing flexibility and timely operational activities. To their help, models are available summarizing the existing knowledge and attributing different risks to different stages of the disease development in combination with silvicultural activities e.g. spore infection of stumps and stump treatment. Operational activities and goals have to be compared to the costs and risks involved in the operations and the infections of *Heterobasidion*. One future uncertainty regards global warming, likely causing a prolonged season for spore spread. It is unclear however, what the true spore load may be during mild winters in southern Sweden and what the potential effect will be on the distribution of *H. annosum* in areas where it is not present today. New and older research must be intimately linked to disseminate results and recommendations to the forestry sector. Practically oriented research and investigations can constitute golden opportunities for e.g. master's thesis reports, such as our latest studies on stump treatment of hybrid larch, high stumps in pre-commercial thinnings and the risk for infections in lodgepole pine in central Sweden. The work has to be consequent and cost benefit focused but will inevitably result in a more efficient and sustainable forestry.

**Dr. Fred Peet, Research Scientist (Retired), Canadian Forest Service, Pacific Forestry Centre, Victoria, BC**

Root Rot Tracker (RRT) is a simulator for the spread of forest root diseases (Peet and Hunt, 2005). Briefly, it grows individual roots, grows fungus along roots, keeps track of root crossings, keeps track of fungus transfer from infected roots to healthy roots at root crossings, uses fundamental biological variables, does not use fudge factors, and is invariant in space and time. The last characteristic is important for it means that one doesn't need different variants of the simulator for different geographical areas or for different time periods. Furthermore, if global warming affects the rate of root growth, the rate of fungus growth along a root, and probability of fungus transfer, then RRT can accommodate global warming effects without modification. The simulator's invariance property is consistent with Hartig's description of the

biological process of root disease spread that is also space and time invariant; it applies not only in Germany but also in other areas of the world and still applies after more than 130 years. RRT was tested against a number of published field data sets. Details of the tests, the results, and citations for Hartig's work and the data sets are below. This report gives a brief listing of the results. The first test is a root contact test. Reynolds and Bloomberg measured the number of root contacts in a field study at their Cowichan Lake site. They obtained 2.71 root contacts/m<sup>3</sup>. RRT gave an estimate for this site of 2.92 root contacts/m<sup>3</sup> (see publication). The second test is a mortality test. Tkacz observed the number of trees killed by *Phellinus* at his Mary's Peak site to be 430. RRT gave an estimate for this site of 449 with a standard deviation of 25 trees. The third test is also a mortality test. Morrison and Pellow reported the values of a number of variables at their Sugar Lake site. The observed year in which trees were first killed was 6 and RRT computed a value of 6. The observed number of trees killed in that first year was 4 and RRT computed a value of 5. The observed year with the maximum number of killed trees was 10 and RRT computed a value of 10. The observed number of trees killed in that year was 70 and RRT computed a value of 64. The observed average number of trees killed per year in the last 14 years was 34 and RRT computed a value of 35. The observed total number of dead trees at age 25 was 760 and RRT computed a value of 747. The fourth test was a root collar infection test using Morrison and Pellow's Sugar Lake data. The observed total number of trees with visible symptoms (root collar infection) at age 25 was 962 and RRT computed a value of 922. The fifth test was an actual (belowground) incidence test. Morrison et al. reported the belowground incidence of *Armillaria* in the Nelson Forest Region of 30 - 37 percent at age 19. Applying this to the Sugar Lake site gives a range of 1045 - 1290 trees with belowground symptoms. RRT gave an estimate of 1230 trees with belowground symptoms. (This may be a fortuitous result, as explained in the manuscript, because the Sugar Lake site is in a different forest region than the Nelson Region.) The sixth test consists of three confirmations of qualitative observations recorded in the literature. The first is escapes. Morrison et al. noted that some trees in an infected area escape infection. The graphical display of RRT shows trees that escape infection. The second is probability of infection. van der Kamp observed that 30 years after logging the probability that a tree becomes infected is not related to the distance to the

nearest stump. The graphical display of RRT illustrates this. The third is temporary infection decline. van der Kamp proposed that the decline of infection that is sometimes observed after regeneration is a temporary phenomenon. RRT's graphical output confirms this.

Peet, F. G. and R. S. Hunt. (2005) A New Simulator for the Spread of Forest Root Diseases by Individual Root Contacts. *Forest Science*: 51(5):425-437.

***Phil Cannon, USDA Forest Service, Vallejo, CA***

Phil presented information on a new and exciting club: The Pacific Forest Root and Butt Rot Club. At present, club members are from Saipan, Guam, Florida, California, Kosrae, N. Carolina, Hawaii, and Norway but there is an open invitation for anyone who wishes to join. The purpose of the club is to form a network of individuals who have similar interest in root and butt rots affecting trees in the Pacific Islands, discuss activities to take place or announce events (meetings) that deal with root and butt rot problems. A handout was provided by Phil detailing the occurrence of various root and butt rot pathogens on the islands. For more information about joining the club, contact Phil directly.

**Topics for Next Year's Meeting...**

Next year, the meeting is in Leavenworth, Washington. If there are any potential root disease related topics for discussion, I am certainly open to suggestions. Unfortunately, the meeting concluded without time for a round robin. Following the conference, participants were asked to submit a brief summary of any new or ongoing work. The following summaries were provided:

***Mike McWilliams, Oregon Department of Forestry, Salem, OR***

We continue to do root disease surveys (mostly *Phellinus weirii*) on state lands when requested. We have been working with our contractor on using GPS points within the stands and in disease centers to improve the spatial accuracy of our sketch maps.

***James T. Blodgett, USDA Forest Service, Rapid City, SD***

Along with my *Armillaria* in WY study, I have an *Armillaria*-Fire study, and include root disease as part of my general Forest Health surveys. The most recent surveys included an aspen survey in northern

Wyoming and the Black Hills National Forest (NF), a paper birch survey in the Black Hills NF, hail damage and Diplodia shoot blight survey in the Black Hills and Nebraska NFs, and an Annosus root disease survey with several others. I have done a few limber and whitebark pine surveys and found *Armillaria* in both cover types, but it does not compare with the damage from white pine blister rust or mountain pine beetle in those cover types.

**Recent Publication:**

Worrall, J.J., Harrington, T.C., Blodgett, J.T., Conklin, D.A., and Fairweather, M.L. 2010. *Heterobasidion annosum* and *H. parviporum* in the southern Rocky Mountains and adjoining states. *Plant Disease* 94:115-118.

**Ned Klopfenstein, USDA Forest Service, Moscow, ID**

In Moscow, ID, USA, RMRS Moscow plant pathology staff (Ned Klopfenstein, John Hanna, and Amy Ross-Davis) and collaborators (Gerald I. McDonald – emeritus, and Mee-Sook Kim – Kookmin University, Seoul, Korea) are continuing a collaborative STDP project with Region 6 (Helen Maffei) and Region 3 (Mary Lou Fairweather) to develop predictive models for *Armillaria* spp. in the inland western U.S.A. These predictive models will attempt to predict potential *Armillaria* spp. distributions under various climate-change scenarios. Amy Ross-Davis is further examining the genetic relationships among North American *Armillaria* spp., and is leading an effort to develop better DNA-based diagnostic tools for distinguishing closely related *Armillaria* spp. International collaborations are underway on a long-term project to examine the evolutionary relationships among *A. solidipes* and related species from world-wide sources.

**Recent publications:**

Kim, M.-S.; Klopfenstein, N.B.; Hanna, J.W.; Cannon, P.; Medel, R.; López, A. 2010. First report of *Armillaria* root disease caused by *Armillaria tabescens* on *Araucaria araucana* in Veracruz, Mexico. *Plant Disease* 94(6): 784.

Kim, M.-S., Klopfenstein, N.B., McDonald, G.I. 2010. Effects of forest management practices and environment on occurrence of *Armillaria* species. *Journal Korean Forestry Society* 99:251-257.

Kim, M.-S.; Hanna, J.W.; Klopfenstein. 2010. First report of an *Armillaria* root disease pathogen, *Armillaria gallica*, associated with several new hosts in Hawaii. *Plant Disease* in press.

Kim, M.-S., Klopfenstein, N.B. Molecular identification of *Armillaria gallica* from the Niobrara Valley Preserve in Nebraska. *Journal of Phytopathology*. in press.

Ota, Y.; Kim, M.-S.; Neda, H.; Klopfenstein, N.B.; Hasegawa, E. 2010. The phylogenetic position of an *Armillaria* species from Amami-Oshima, a subtropical island of Japan, based on elongation factor and ITS sequences. *Mycoscience* in press.

**Mike Cruickshank, Canadian Forest Service, Pacific Forestry Centre, Victoria, BC.**

I'm working with Duncan Morrison to get the results from the Skimikin stumping trial done.

I'm working on host resistance/tolerance systems in interior Douglas-fir with Barry Jaquish, Research Geneticist with Ministry of Forests, Mines and Lands. I'm also working on modelling sapwood area within several tree species and what effect disease may have on this within Douglas-fir. Rona Sturrock and I are mapping the distribution of several pathogens to produce national maps.

**Michelle Cleary, Ministry of Natural Resource Operations, Kamloops, BC.**

Work continues with Duncan Morrison to complete remeasurement and analysis of root disease pre-commercial thinning trials across the province. I'm also monitoring long-term species trials to determine root disease impacts on potential productivity. In collaboration with Rona Sturrock, we are continuing work looking at the distribution and impact of *Phellinus* root disease in the southern interior of BC.

**Recent publications:**

Cleary, M. and T. Holmes. 2011. Traumatic resin duct formation in the phloem of western red cedar (*Thuja plicata*) following abiotic wounding and infection by *Armillaria ostoyae*. *International Association of Wood Anatomists Journal*. 32(1) [in press]

Special thanks to all participants for sharing the great root disease work. I appreciate your interest and energy!

# Climate Change and Forest Diseases ‘sort of’ Committee, Meeting

## Co-Chairs - David Shaw, Susan Frankel, and Terry Shaw

Over 25 people attended. It was decided by all attending that the committee should be a formal WIFDWC committee, as the climate change issue will only increase in importance in the coming years. A great discussion was generated by the presentation from Harry Kope concerning what the BC Ministry of Forests forest health specialists (Alex Woods, Don Heppner, Harry Kope, Jennifer Burleigh, Lorraine Maclauchlan) think about how climate change will influence forest pathology. Here are a few highlights from their presentation:

**Foliar Diseases:** IF - Minimum temperatures and precipitation increased; THEN – Increased over-winter survival, and infection and spread (Climate becomes drier, < foliar diseases, > host stress).

**Stem Diseases:** IF - Warmer and wetter springs and summers; THEN – Increased infection and spread, > wave years (Climate becomes drier, < stem diseases, > host stress).

**Root Diseases:** IF – Drought; THEN – Host stress ⇒ root disease (Southern interior under greater drought stress and therefore at higher risk to root disease).

**Dwarf Mistletoe:** IF - Warmer winter temperatures; THEN – Geographic and elevational range expansion; (i.e., Douglas-fir mistletoe further north and coastward, and Hemlock mistletoe to inland areas of BC).

**Pest Complexes:** Climate is one driver in maintaining an ecological and physiological balance between host and pests. A change in this balance could favour innocuous insects and diseases ⇒acting together on stressed hosts.

**Decline Syndromes:** Physical factors (climate change) cause initial damage resulting in stressed trees, and secondary pests aggravate the problems. The decline and deterioration of stressed trees are more likely to experience growth impacts, a slow decline in health and death.

**Invasive Pests:** BC’s cold winters limit likelihood of establishment. Warmer winters, > invasive alien disease survival and spread.

They also noted major challenges in long-term timber supply, modeling, assisted migration and genetic diversity, gene conservation and tree breeding. They finished with the quote, “Beware the status quo - the risk associated with maintaining the status quo far exceeds the risks of implementing changes that provide for a broader range of future forest conditions, and uses.”



# Hazard Tree Committee Meeting

## Chair – Pete Angwin

Approximately 25 people attended the WIFDWC Hazard Tree Committee Breakfast. Pete Angwin chaired the meeting and Kristen Chadwick took notes. Four items were on the formal agenda:

1) Pete Angwin opened the breakfast meeting with a powerpoint presentation of photos from the 2010 Western Hazard Tree Workshop. Overall, the workshop was a tremendous success. Highlighted topics included the Region 6 roadside “danger tree” program and defects/hazards in oaks. The workshop “pre-ceedings” and presentations are in the Archives section of the Hazard Tree Workshop website: [www.fs.fed.us/foresthealth/technology/htwc/archive.htm](http://www.fs.fed.us/foresthealth/technology/htwc/archive.htm)

Attendance was greater than anticipated, with 79 total participants (65 paid registrants, 11 waived registrations and 3 guests). Three \$500 travel stipends were provided for speakers, one of which was paid for by the Oregon Dept. of Forestry. Because of the large attendance, the workshop cleared over \$4,000. All bills associated with the workshop are paid, and the current balance of the WIFDWC Hazard Tree account is now \$5401.96 (reported by John Schwandt, Jan 4, 2011).

Thanks to the many people who helped put the workshop together, including the Organizing Committee, Greg Filip (Program Chair), Don Goheen and Kristen Chadwick (Field Trip Organizers), Don and Ellen Goheen (Local Arrangements), Bill Jones and Jessie Glaeser (Workshop “Pre-ceedings”), Kristen Chadwick (Hazard Tree Photo Contest), John Schwandt (Treasurer), Katie Mallams (Registration Assistance), and Al Kanaskie and Paul Reis (ISA and SAF Credits). Special thanks are also due to Ellen and Don Goheen, who hosted a tremendous dinner gathering at their house!

The next Western Hazard Tree Workshop will be in 2013. The initial Organizing Committee meeting will be in the spring of 2012. Participation in the Organizing Committee is encouraged! Call or email Pete Angwin if interested (phone 530-226-2436 or email [pangwin@fs.fed.us](mailto:pangwin@fs.fed.us)). We also want ideas for the location of the workshop.

2) Kristen Chadwick and Bruce Moltzan led a discussion on the International Tree Failure Report Database (ITFD). Kristen provided an update on the ITFD Steering Committee meeting held in Jacksonville, Oregon, the evening before the Western Hazard Tree Workshop. The major item of discussion was future housing and maintenance of the ITFD. In recent months, the Director of FHTET (Frank Sapio) has stated that he feels that maintenance of the ITFD is not an appropriate long-term commitment for FHTET, and has asked Judy Adams to find an outside party to house and maintain the database.

Bruce Moltzan provided an update of the situation. Bruce has been asked to produce a needs assessment for Frank Sapio on the importance of the ITFD and how best to continue. Larry Costello will provide assistance in the response to Frank. In summary, we need to make the case to either keep the database at FHTET or provide other feasible options. Bruce solicited the involvement of all parties interested in the ITFD, particularly those interested in keeping it under FHTET, in this effort.

3) Bruce Moltzan led a discussion on the effort to revise sections of the Forest Service Manual (FSM) direction that outline policy on hazard trees and hazard tree management. Mary Lou Fairweather stated that in Region 3, revision of the FSM has become a pressing issue following an out of court settlement in a fatality case. Bruce responded that he is in contact with Chris Hartman (WO Recreation Sites Program Manager) in order to offer Forest Health Protection (FHP) assistance with the Manual update. Revisions to Chapters 2310, 2320, 2330 and 2350 were already underway, but delayed for further review following multiple fatalities of recreationists during a flash flood in Arkansas this last summer. The FSM revision will likely be out in about a year, with FHP assistance. Bruce solicited the help of all FHP staff, and Pete Angwin suggested that he contact the FHP Hazard Tree lead in each Forest Service Region for assistance.

4) Pete Angwin provided an update on the status of updates to the Forest Insect and Disease Leaflets (FIDLs). Kathy Sheehan is in charge of the FIDL

program. Two FIDLS on decay fungi were completed in 2010: FIDL 177- Schweinitzii Root and Butt Rot of Western Conifers (Hagle and Filip) and FIDL 52- Decays of White, Grand, and Red Firs (Mallams, Chadwick and Angwin). A schedule of upcoming FIDL updates was distributed. Some are further along than others are. Contact Kathy Sheehan if anything on the schedule sheet is incorrect. She also asked that everyone be aware that at some point in the near future, the FIDL webpage is going to have a new domain, along with all US Forest Service web material. She is working hard to ensure that the FIDL web site survives the move.

**After the discussions on the four formal agenda items, two additional round-robin items were discussed:**

1) Lori Winton spent several weeks testing an acoustic tomograph. Acoustic tomographs were developed in Germany about 15 years ago, and currently cost about \$8,000. The one she used had ten sensors (ones with up to twenty-four sensors are available) placed about three centimeters into the wood of the subject tree. Setup took about 15 minutes per tree. The sensors are at different heights to give a three-dimensional picture of the decay column. The sensors measure the time that it takes for sound to go through the tree, and the tomograph uses this data to generate

a computer diagram that shows internal areas of structural weakness, such as decay pockets and included bark. Trees of all sizes are assessed with a tomograph. For additional information, contact Lori.

2) Bill Jacobi is currently working on defects of Plains cottonwoods. Anyone with information that would be helpful in this endeavor should contact Bill.



# Rust Committee Report

## Chair - Holly Kearns

Approximately 45 people attended the Rust Committee meeting. We celebrated the Forest Pathology special issue: White Pines, Ribes, and Blister Rust, which was published in August after much arduous work by guest editors Brian Geils and Terry Shaw and contributions from many WIFDWC members. In addition, we had a formal presentation and a round robin discussion regarding current and up-coming rust-related projects. The following are brief summaries:

### **Michael Murray (BC Ministry of Forests, Nelson, BC) – Annual Observations of Conspicuous Canker Activity on Whitebark Pine (2003 to 2007)**

Rust-induced mortality is becoming well-documented across the natural range of whitebark pine. However, at a finer scale (i.e. individual tree), the biological interactions taking place between the infected host (whitebark pine) and disease are not well-studied. The objective of this study was to track the most apparent signs of canker activity on mature whitebark pine in Crater Lake National Park, Oregon over a 5-year study period. The magnitude and duration of inactive periods were documented. Further insight regarding possible differences between canker locations (branch vs. stem, location in study area) was also sought. For this study, activity was indicated by one or more symptoms: conspicuous resinosis, sporulation, rodent-gnawing, or bark discoloration. A total of 52 cankers from 46 trees were tracked. Overall, 42 percent of cankers changed their status (active vs. inactive) at least once. Cankers were significantly more active in 2003 and 2006 ( $\chi^2$ , Yates correction,  $P < 0.05$ ). Branch cankers were observed to be more active than stem cankers in all years except 2003, although not with significance ( $U = 5$ ;  $P < 0.05$ ). The east-side tended to have lower activity, however, the disparity between sides was not significant ( $U = 8$ ,  $P < 0.05$ ). The reliability of outward signs of activity closely reflecting fungal virulence is not well-documented in literature. The disease may continue to thrive, and possibly spread beneath the bark without conspicuous symptoms. Notwithstanding, cankers failed to re-activate after three years of inactivity. Additional studies could explore the utility of this potential threshold in determining when a canker is no longer infected with inoculum. Overall, half of all inactive

cankers failed to re-activate. Thus, classifying a tree as having blister rust based on the existence of a single canker, which appears inactive would risk overestimating disease incidence.

### **Bill Jacobi (Colorado State University, Fort Collins, CO) –**

- Limber planting trials along Front Range CO/WY, in second field season (A.M. Casper, MS candidate).
- Studies of periodicity of natural seedling establishment in planting trial locations and height by age relationships are ongoing (Anne Marie Casper, MS candidate).
- Five year evaluation of study of pathological and preventive pruning of high value limber pine infected with WPBR at Great Sand Dunes NP was completed in summer 2010 (FHM, NPS, CSU).
- Presented progress on WPBR epidemiology studies at High-5 Symposium.
- Continuing to refine the epidemiology assessments using on site meteorological data.

### **Anna Schoettle (Rocky Mountain Research Station, Fort Collins, CO) –**

- Continuing the screening for polygenic (partial) rust resistance of 74 southern Rocky Mountain (RM) limber pine families at Dorena - in cooperation with National Park Service (NPS).
- Rust screening of 184 RM bristlecone families is nearly complete (at Dorena) - there is resistance and it varies geographically - in cooperation with R2 FHM.
- Over 140 families of N. Colorado limber pine entered rust testing this year - inoculation at Dorena scheduled for Sept 2010 - in cooperation with NPS and R2-FHM.
- Continue to protect limber and RM bristlecone pine seed trees showing resistance or currently in testing, from MPB attack (in cooperation with NFS, NPS and R2-FHM).
- Confirmed the presence of WPBR on limber pine in Rocky Mountain National Park.

- Regen. surveys in 29 limber sites in N. CO completed to assess post - MPB population structure.
- Presented research results at the High-5 in Missoula (B. Goodrich, J. Klutsch, J. Coop and myself).
- Working toward completing range-wide seed collections for RM bristlecone for gene conservation, rust resistance testing and research. Unfortunately, 2010 is not a bumper cone yr (in cooperation with WO-FHP, R2-FHM, and help from many others!)
- We are developing a population genetic model to predict 5-needle pine population dynamics under different rust infection probabilities and resistance allele frequencies.
- Continuing to work with economists to evaluate the ecological and economic trade-offs of proactive and reactive management option for WPBR in high elev. 5-needle pine ecosystems.

**Jim Blodgett (Forest Health Management (FHM), Rapid City, SD) –**

- Collections and screening of bristlecone, limber, and whitebark pine are ongoing to conserve resistant seed trees (with RMRS, NPS, and various NFs).
- Remeasured comandra and white pine blister rust permanent plots of the Pest Trend Impact Plot System in South Dakota and Wyoming.

**Kelly Burns (Forest Health Management, Lakewood, CO) –**

- Aiding with RM bristlecone/limber pine cone collections, screenings and plus tree/superior tree MPB protection.
- Serving as the R2 regional contact for FHM's Monitoring on the Margins program.
- Marcus Jackson presented results from a R1-R2 EM project (Monitoring Limber Pine Health in the Rocky Mountains) at the High-5 Symposium in my absence. These results are also being published in the 2009 FHM National Technical Report.
- Confirmed the presence of WPBR on limber pine on Pikes Peak.
- Hazard tree issues are taking precedence as MPB decimates pine stands in no. CO/so. WY. There is great concern about the status of limber and whitebark, which have been particularly impacted.

**John Schwandt (Forest Health Protection, Coeur d'Alene, ID) –**

- Finished 2yr monitoring project looking at 42 mature whitebark stands severely impacted by mountain pine beetle, to document conditions following the outbreak. Results presented at High-5 Symposium.
- Presented a Whitebark Pine Restoration Program Summary (2010) at the High-5 Symposium.
- Establishing new prune and thin monitoring plots in western white pine plantations (2007-2011) to monitor differences in treatments. We are including weather instruments to document relative humidity and temperatures in different treatments.
- Summarizing data from prune and thin plots established 40 years ago (2011) to compare the effectiveness of pruning vs thinning.
- Planning to remeasure monitoring plots established in 22 F2 western white pine plantations 15 years ago to document changes in rust infection and mortality.
- Surveying new F2 western white pine plantations planted recently with Grouse Creek stock, which contains the latest tree improvement efforts to improve the level of rust resistance. Two stands examined in 2009 had very high levels of rust after less than 5 years.
- Writing up data collected on canker growth of nearly 200 cankers on F2 stock which indicate canker expansion rate is about 2" per year, similar to unimproved stock.
- Remeasuring survey plots established in 2007 in a young whitebark pine stand to create openings around potential crop trees (daylighting) to enhance survival. We are testing two different daylighting treatments on three size classes of trees.
- Writing up results of a small study that documented the age of small whitebark pine. We found trees that were only 3 to 8 feet tall were 20-80 years old.
- Analyzing and summarizing data from regeneration monitoring plots established 15 years ago in several natural stands of whitebark pine to monitor levels of rust and mortality over time.
- Continuing to install and monitor whitebark pine direct seeding trials. We established four in 2009 and two in 2010. Plantings are testing

different seed treatments as well as multiple seed caches vs. single seed plantings. We also are planting seed next to seedlings to provide long-term survival comparisons. The 2010 trials added a mycorrhizal treatment to enhance survival. Results through 2009 presented at the High-5 Symposium.

- Planning to survey whitebark pine stands in the Hells Canyon vicinity for rust and bark beetle impacts (in cooperation with R6).

**Michael Murray (BC Ministry of Forests, Nelson, BC) –**

- Have compiled results based on annual canker monitoring for conspicuous active/inactive status (2003-2007) at Crater Lake NP, Oregon. Results are from mature whitebark pine trees and indicate no cankers re-activated after three years of inactivity. Overall, half of all inactive cankers failed to re-activate.
- Re-measured three whitebark pine long-term monitoring plots in BC Kootenays. Mortality due to blister rust approaching 1 percent/year with disease incidence 13-63 percent among stands
- British Columbia lacks any programmatic development of rust resistance aimed at whitebark and limber pine. This is a reflection of the lack of financial support from the BC Government.
- Canadian federal government is formally assessing whitebark pine for endangered status. Final decision expected in July 2011.

**Ned Klopfenstein (Rocky Mountain Research Station, Moscow, ID) –**

The research group in Moscow has completed studies on range-wide genetic diversity of western white pine. International studies are continuing to assess evolutionary relationships among international sources of *Cronartium ribicola*.

FHP Region 5 (Phil Cannon, Anne Marie LaRosa), Universidade Federal de Vicosa in Brazil, Washington State University, University of Hawaii, Kookmin University in Korea (Mee-Sook Kim), Hawaii Department of Land and Natural Resources, and RMRS in Moscow, ID (Amy Ross-Davis and Ned Klopfenstein), and others are collaborating to understand the genetic diversity of the eucalypt/guava/myrtle/’ohi’a rust pathogen (*Puccinia*

*psidii*). Eucalypt rust, which is putatively native in Brazil, is currently threatening myrtaceous trees that are a large component of the native forest in Hawaii. Furthermore, eucalypt rust could threaten eucalypts and other myrtaceous trees worldwide. The collaborative studies aim to understand the threats posed by this invasive eucalypt rust pathogen better.

**Recent publications:**

- Butler, J.; Pearson, D.; Kim, M.-S., tech eds. 2009. USDA Forest Service, Rocky Mountain Research Station, Invasive Species Working Group: Research summary and expertise directory. USDA Forest Service, Rocky Mountain Research Station, Fort Collins, CO. 20 p.
- Klopfenstein, N.B.; Geils, B.A. 2009. Summary of taxa-specific research 2. Pathogens. pp. 7-8 in: Butler, J.; Pearson, D.; Kim, M.-S., tech. eds. Invasives Species Working Group: Research Summary and Expertise Directory. USDA Forest Service, Rocky Mountain Research Station, Fort Collins, CO.
- Klopfenstein, N.B.; Juzwik, J.; Ostry, M.E.; Kim, M.-S.; Zambino, P.J.; Vennette, R.C.; Richardson, B.A.; Lundquist, J.E.; D. Lodge, D.J.; Glaeser, J.A.; Frankel, S.J.; Otrosina, W.J.; Spaine, P.; Geils, B.W. 2010. Invasive forest pathogens: Summary of issues, critical needs, and future goals for Forest Service Research and Development. pp. 23-33 in: Dix, M.E.; Britton, K., eds. A Dynamic Invasive Species Research Vision: Opportunities and Priorities 2009-29. Gen. Tech. Report WO-79. USDA Forest Service, Research and Development, Washington, DC.
- Britton, K.O.; Buford, M.; Burnett, K.; Dix, M.E.; Frankel, S.J.; Keena, M.; Kim, M.-S.; Klopfenstein, N.B.; Ostry, M.E.; Sieg, C. Hull. 2010. Invasive species overarching priorities to 2029. pp. 3-11 in: Dix, M.E.; Britton, K., eds. A Dynamic Invasive Species Research Vision: Opportunities and Priorities 2009-29. Gen. Tech. Report WO-79. USDA Forest Service, Research and Development, Washington, DC.
- Richardson, B.A., Kim, M.-S., Klopfenstein, N.B., Ota, Y., Woo, K.S., and Hamelin, R.C. 2009. Tracking the footsteps of an invasive plant pathogen: Intercontinental phylogeographic structure in the white-pine-blister-rust pathogen, *Cronartium ribicola*. pp. 56-60 in: Noshad, D.; Noh, E.W.; King, J.; Sniezko, R., compilers. Proceedings of the Breeding and Genetic Resources of Five-Needle Pines: Ecophysiology, Disease Resistance and

Developmental Biology. IUFRO Working Party 2.02.15. 22-26 September 2008. Yangyang, Korea.

Ekramoddoullah, A.K.M.; Liu, J.-J.; Kim, M.-S.; Klopfenstein. 2010. Current and future molecular approaches to investigate the white pine blister rust pathosystem. *Forest Pathology* 40: 314-331.

Klopfenstein, N.B.; Geils, B.W., eds. 2010. RMRS Invasive Species Science Update (Number 4). USDA Forest Service, Rocky Mountain Research Station, Fort Collins, CO. September, 2010. 10 p. (<http://treesearch.fs.fed.us/pubs/36117>)

Kim, M.-S.; Klopfenstein, N.B.; Ota, Y.; Lee, S.K.; Woo, K.-S.; Kaneko, S. 2010. White pine blister rust in Korea, Japan, and other Asian regions: comparisons and implications for North America. *Forest Pathology* 40: 382-401.

Richardson, B.A.; Rehfeldt, G.E.; Kim, M.-S. 2009. Congruent climate-related genecological responses from molecular markers and quantitative traits for western white pine (*Pinus monticola*). *International Journal of Plant Science* 170: 1120-1131.

Kim, M.-S., Richardson, B.A., McDonald, G.I., and Klopfenstein, N.B. Genetic diversity and structure of western white pine (*Pinus monticola*) in North America: Implications for conservation and restoration. *Tree Genetics and Genomics* in press.

**Richard Snieszko (Dorena Genetic Resource Center, Cottage Grove, OR)** – Recently completed rust field assessments on some range-wide sugar pine provenance trials planted in the 1980's in OR and CA; and 6 linked progeny trials of sugar pine planted in the early 1980's in southern OR. Presented a poster at the annual American Phytopathological Society meeting entitled: White pine blister rust resistance in a seven-year-old field trial of 28 western white pine (*Pinus monticola*) families in the Coast Range of Oregon. Rust screening of whitebark pine and limber pine (the latter with Anna Schoettle).

**Mary Lou Fairweather (Forest Health Protection, Flagstaff, AZ)** – WPBR was discovered for the first time in Arizona in 2009, after two decades of searching. Forest Health Protection in Arizona is collaborating with Dr. Kristen Waring at Northern

Arizona University, who received Evaluation and Monitoring funding to assess the status and extent of white pine blister (WPBR) in Arizona. We are establishing long-term monitoring throughout the state, even though infestations are currently limited to moist canyon bottoms in the White Mountains of eastern Arizona.

**Robyn Mulvey (Oregon State University, Corvallis, OR)** – New paper coming out in *Forest Pathology* in 2011: Mulvey, R.L. and Hansen, E.M. Castilleja and Pedicularis confirmed as telial hosts for *Cronartium ribicola* in whitebark pine ecosystems of Oregon and Washington.

**Fred Baker (Utah State University, Logan, UT)** – Evaluating the effects of pruning and genetically improved stock on reducing the impact of WPBR in eastern white pine plantations in Minnesota and Wisconsin.

**Alex Woods (BC Ministry of Forests, Smithers, BC)** – Completed and submitted a manuscript that covers the increase in hard pine rust incidence in central BC and its possible link to recent weather. The paper should come out in *The Canadian Journal of Plant Pathology* under the symposium section.



# Dwarf Mistletoe Committee Report

## Chair - Fred Baker

Twenty-six dwarf mistletoes met for breakfast 0800 Friday morning.

### **Highlights of the discussion include:**

Broom pruning Douglas-fir had no effect (Filip, Chadwick and Maffei).

Dave Shaw (OSU) is studying the vertical fuels in lodgepole pine stands after mountain pine beetle infestation; he also mentioned an interesting book entitled Mistletoes of the Western Himalayas by YUPS Pundin of India.

Bob Edmunds is inoculating hemlocks as part of an ecological restoration at Cedar River.

Simon Shamoun continues his work on parasitic fungi of hemlock and lodgepole pine dwarf mistletoes.

Phil Cannon reported on pruning dwarf mistletoe infested trees in campgrounds and is seeking information on similar projects.

The mistletoes of hardwoods FIDL has been revised by Ellen Goheen and Katy Mallams and is available on the R6 FHP website.

Don Goheen reported that the Ashland, OR watershed lost 30 percent of its Douglas-fir to dwarf mistletoe over the past 30 years, and muttered something about overstocking.

Dave Conklin and MaryLou Fairweather revised the Dwarf Mistletoe Management Guide for Region 3, with specific changes in the percentage infection recommended for uneven-aged management.

Fred Baker reported that a paper on the distribution of dwarf mistletoe by crown thirds was hot off the press in the Western Journal of Applied Forestry (25:194-198). Another paper on Spatial distribution of dwarf mistletoe is in press in Ecological Modeling. Projects are underway to quantify the appearance of bird initiated mistletoe centers, and to explore the relationship between percent infection and DMR in FIA data.

**And, the only formal written contribution came from:**

**Bob Mathiasen** recently published a short article in Northwest Science on dwarf mistletoes that parasitize Brewer spruce in the Klamath-Siskiyou Mountains of northwestern California and southwestern Oregon (Northwest Science, Vol. 84, Pp. 295-301. R. Mathiasen and C. Daugherty, Northern Arizona University, Flagstaff.

We are continuing our systematic studies of dwarf mistletoes that primarily parasitize white pines in western North America and Mexico. We are using molecular techniques (AFLP analysis) and completing additional morphological measurements for *A. apachecum*, *A. blumeri*, *A. californicum*, *A. cyanocarpum*, *A. monticola*, and *A. guatemalense* to determine the taxonomic relationships of these taxa better. B. Reif and R. Mathiasen, Northern Arizona University, Flagstaff.

We have begun studies comparing the morphology and phenology of black dwarf mistletoe (*A. nigrum*), Gill's dwarf mistletoe (*A. gillii*), and Honduran dwarf mistletoe (*A. hondurensis*) in Mexico and southern Arizona. We are also re-examining ribosomal DNA ITS sequences of these taxa. R. Mathiasen and B. Reif, Northern Arizona University, Flagstaff and S. Kenaley, Cornell University, Ithaca, NY.

Limber pine dwarf mistletoe (*A. cyanocarpum*) was found infecting sugar pine (*Pinus lambertiana*) for the first time in the San Jacinto Mountains, California. This first host report was published in Plant Disease, Vol. 94, p. 134. Although only one tree was infected growing near severely infected limber pines, there were several infections on the tree and most of the infections were producing plants which were morphologically identical to the plants on the limber pines. R. Mathiasen and C. Daugherty, Northern Arizona University, Flagstaff.

Blumer's dwarf mistletoe (*A. blumeri*) was discovered parasitizing Mexican white pine (*Pinus ayacahuite*) and round dwarf mistletoe (*A. globosum* subsp. *globosum*) was found infecting Durangan pine (*P. durangensis*) in Sinaloa, Mexico. These first host

reports were published in *Plant Disease*, Vol. 94, p. 377. S. Quinonez, CONAFOR, Durango, Mexico and R. Mathiasen, Northern Arizona University, Flagstaff.

White fir dwarf mistletoe (*A. abietinum* f. sp. *concoloris*) was infecting Mexican spruce (*Picea mexicana*) on Cerro Mohinora in southern Chihuahua, Mexico. This is the first report of this dwarf mistletoe on this rare Mexican spruce. This new host report is in *Plant Disease*, Vol. 94, p. 635. Plants of white fir dwarf

mistletoe on Durangan fir (*Abies durangensis*) and Mexican spruce at this location were bluish-green and highly glaucous, rather than the more typical yellow-green to yellow of white fir dwarf mistletoe plants in the Sierra Nevada Mountains. Therefore, we began additional taxonomic studies of the white fir dwarf mistletoe populations in northern Mexico and Arizona in September 2009. R. Mathiasen and C. Daugherty, Northern Arizona University, Flagstaff.



## WIFDWC Business Business Meeting Notes

Written/compiled by Mary Lou Fairweather, 2011 WIFDWC Secretary

Chairperson Rona Sturrock called the Business Meeting of the 58<sup>th</sup> Western International Forest Disease Work Conference to order at 9:00 am on October 8, 2011. Thirty-two people attended.

### Old Business

A motion to accept the minutes from the 2009 WIFDWC (Durango, CO) business meeting included in the published proceedings is seconded, and passed.

The 2010 Organizing Committee is acknowledged for coordinating a great meeting: Michelle Cleary and Richard Reich (Local Arrangements); Don Goheen (Program Chair); Judy Adams (Web Master); John Schwandt (Treasurer); Michael Murray (Student Presentations); Simren Brar (Posters); Mary Lou Fairweather (Secretary).

### Treasurer's Report (John Schwandt)

In spite of uncertain budgets in the US and Canada, there are 83 total registrants including 50 regular members, 12 students, 9 retirees, and 12 spouses/guests/fiancés. The following is a summary of transactions for the WIFDWC account from 11/15/2009 through 12/31/2010. This year we initiated a silent auction and thanks to Blakey Lockman and Holly Kearns plus the great participation by everyone, we raised a total of \$1,364 for future student awards. After 15 years as WIFDWC treasurer, I have decided to transfer the treasury into the capable hands of Holly Kearns starting in 2011. Please note that our Federal Tax Id # is 35-2307554.

Transaction	Income	Expenses	Balance
<b>2010 Hazard Tree Meeting- Medford, OR June 15-18</b>			<b>\$971.54</b>
Registration	\$12,875.49		
Speaker Expenses		\$1,000.00	
Catering – breaks, lunches, social		4,255.00	
Field Trip Transportation		1,834.00	
Banquet		1,076.65	
Printing, copying		217.34	
Misc expenses		62.08	
<b>Ending Balance</b>			<b>\$5,401.96</b>

<b>WIFDWC balance as of 11/15/2009:</b>			<b>\$18,652.66</b>
<b>2010 WIFDWC Meeting –Valemount, BC October 4-8</b>			
Total registration	\$19,841.26		
Hotel meeting rooms, meals, breaks		\$9,845.77	
Field trip transportation		3,094.00	
Speaker expenses		1,650.00	
Field trip supplies and lunches		1,624.24	
Trinkets and awards		735.70	
<b>Net Proceeds (\$2,891.55)</b>			
Estimate for Valemount proceedings		(3,000.00)	
<b>Other Account Activity</b>			
2008 Proceedings (printing and mailing)		2,443.84	
2008 Registration adjustment	14.00		
2009 Proceedings (printing and mailing)		3,139.24	
2009 Registration adjustment		85.97	
2011 Meeting deposit		250.00	
Sales of proceedings	60.00		
Bank interest/dividends	72.83		
<b>Ending WIFDWC Balance</b>			<b>\$15,771.99</b>
<b>Student Award Fund</b>			
Silent Auction Proceeds	1,364.00		<b>\$1,364.00</b>
<b>Total Balance as of 12/31/2010 (WIFDWC + Hazard Tree + Student Award)</b>			<b>\$22,537.95</b>

### **Historian's Report (Rona Sturrock)**

Pete Angwin identified a need for clarification regarding the history of the WIFDWC Social Achievement Award.

Previous History of the Social Achievement Award as submitted in 2009 Historian's Report: "It appears that this Award was first given in 1957 to Stueie Andrews and last given in 1992 to Pete Angwin. The Social Achievement Award was replaced by the Outstanding Achievement Award (in 1993?)."

The History of the Social Achievement Award will now state the following: "The Award was first given in 1957 to Stueie Andrews, last given in 1996 to Ken Russell and officially retired in 1997. The Social Achievement Award was replaced by the Outstanding Achievement Award, the first of which were awarded in 2000 to a member from Canada - Duncan Morrison and a member from the USA - Lew Roth."

### **Nominations for 2011 WIFDWC Organizing Committee**

The Railroad Committee consisted of Dave Shaw, Mike McWilliams, and Richard Reich.

- 1) Committee Chair: Pete Angwin .
- 2) Program Chair: Alan Kanaskie was nominated, seconded, and closed.
- 3) Secretary: Stefan Zeglen was nominated, seconded, and closed.

### **2011 Outstanding Achievement Award (OAA) Committee**

This year Ellen Goheen completes her term. The current members are Pete Angwin (Chair) and Harry Kope (Canadian rep). Ellen nominated Bill Jacobi for the third member, which was seconded and closed.

## Future Meetings

**2011:** The 2011 meeting will be Oct. 10-14, in Leavenworth, Washington. Greg Filip secured a facility, with furniture imported from Austria, and is developing the field trip stops. Although it interferes with Canadian Thanksgiving, no one had an issue. Pre-WIFDWC trip on Lake Chelan may occur, a 20-minute drive from Wenatchee.

- Greg Filip discussed that during the 2009 Business meeting the idea of a "North American" scale meeting was raised and he was assigned to ask other forest pathology organizations of their interest in such a meeting. He contacted Glen Stanosz from UW at Madison, Bill Jones, USFS-FHP at Asheville, NC, Linda Haugen USFS-FHP in St. Paul, MN., and Bruce Moltzan, USFS-FHP in Washington, DC. Their responses were mixed, from "it's a good idea" to "we already meet nationally at the APS meeting." Greg reported that WIFDWC would probably have to host the first meeting, since none of the people that he contacted offered to do so.
- A motion was then made to invite the other forest pathology organizations to such a meeting, with 14 people in favor, 10 against. However, a followup discussion ensued over whether the vote was for inviting the other organizations or asking their respective organizing committees to collaborate on one large meeting. This question was not resolved; Rona suggested that scoping of all organizations should be done first. Rushing for a large North American-scale meeting in 2012 might not be feasible.
- Stefan Zeglen motioned that the 2011 WIFDWC Meeting Chair (Pete Angwin) send a letter to the other forest pathology organizations to start exploring interest in having a large joint meeting in 2013; 12 were in favor to 6 against, and a lot of folks abstained.

**2013:** Proposed locations for the 2013 WIFDWC meeting included Edmonton, Alberta, or somewhere in Alaska or US Forest Service Region 4. However, the location is contingent on whether or not WIFDWC and

**2012:** The 2012 meeting will be in California near Lake Tahoe or Big Bear. A discussion followed on whether or not to make this a North American Forest Disease Work Conference. Planners would need to make sure the facility was adequate with good airport accessibility to get there. Many people spoke, some in favor and some not in favor. A big concern was that some felt the annual APS meeting serves this purpose, while others felt that a North American-wide meeting like this would be too big.

other organizations decide to have a North American Forest Disease Work Conference.

Planning for the 2013 Hazard Tree Workshop will occur in 2012.

## New Business

Holly Kearns will be checking into WIFDWC gaining Tax Exempt Status. She will check to see if auction donations and proceeds are tax deductible.

The Climate Change Committee currently stands as an ad-hoc committee with co-chairs Dave Shaw, Susan Frankel, and Terry Shaw. Business meeting participants discussed allowing the committee permanent WIFDWC standing. This requires an amendment to WIFDWC Bylaws: passage with a 2/3 majority vote, queried at the Business Meeting and with a quorum of at least 25 members. Ellen Goheen motioned to allow the Climate Change Committee permanent standing; it was seconded and unanimous vote to become a full member committee.

Walt Thies proposed having the WIFDWC business meeting earlier in the week. The planning committee will consider this request.

The entire Membership List needs to be in the proceedings in total. A discussion took place about differences in the electronic availability of the list in emailing messages. The list is available in two forms; the US Forest Service has a personal distribution list (pdl) accessible through the email server, and everyone outside the agency must acquire a spreadsheet from Fred Baker and build an email list. A motion was made to form a committee to look into: 1) membership list and access and 2) review passed motions and revise and update bylaws. The committee would consist of Fred Baker, Judy Adams, Rona Sturrock, Mary Lou Fairweather, and Ellen Goheen. Seconded and passed.

A Student Aid Committee is necessary to draft a proposal on aid dispersal and the membership will vote by e-vote (see below). Bylaws will be updated to describe the protocol. A motion was made to form an ad-hoc committee consisting of Holly Kearns, Blakey Lockman, John Schwandt, and Bill Jacobi. They will draft a process, seek input/comment from the membership and finalize it by March 2011. The motion is seconded and accepted.

Terry Shaw motioned to utilize electronic email voting for proposals/motions, this is seconded and accepted. Such a vote was conducted for the first time in 2008, when Fred Baker asked the membership by email to vote yes or no on the question of "should the ad hoc Foliage and Twig Diseases committee gain standing committee status?". A return of yes votes by more than 35 WIFDWC members saw this motion approved.

***Rich Hunt gave the following verbal and written motion:***

In the interest of nomenclature stability, be it resolved that: a) over the course of the next year all WIFDWC members will use the specific epithet "ostoyae" along with "solidipes" for the taxon "*Armillaria ostoyae*" in all correspondence with clients, one another and initial journal submissions; b) Duncan Morrison and Rich Hunt be charged to investigate this issue and take appropriate action in the interest of nomenclature stability and that they (present a) preliminary report to the root disease chair by January 15<sup>th</sup> 2011 and

submit final report by June 15<sup>th</sup> 2011. The root disease chair will take whatever action is necessary to have this issue resolved by the 2011 WIFDWC. Rich's motion was seconded and accepted.

Meeting adjourned.



## Standing Committees and Chairs, 1994—2010

Committee	Chairperson	Term
Hazard Trees	J. Pronos	1994—2005
	P. Angwin	2006—present
Dwarf Mistletoe	R. Mathiasen	1994—2000
	K. Marshall	2001—2003
	F. Baker	2004—present
Root Disease	G. Filip	1994—1995
	E. Michaels Goheen	1996—2005
	B. Ferguson	2006—2009
	M. Cleary	2010—present
Rust	J. Schwandt	1994, 2005
	R. Hunt	1995—2004
	H. Kearns	2006—present
Disease Control*	B. James	1995—2002
Nursery Pathology	B. James	2002—2005
	K. Mallams	2007—present
Foliar and Twig Diseases**	H. Kope	2007—present
Climate Change***	S. Frankel	2007—2008
	S. Frankel and D. Shaw	2009—present

\*Disease Control committee was disbanded in 2002.

\*\*Foliar and Twig Diseases committee was made full charter member in 2009.

\*\*\*Climate Change committee was made full charter member in 2010.

## Past Annual Meeting Locations and Officers Meetings and Officers, 1953—2010

Annual	Year	Location	Chairperson	Secretary-Treasurer	Program Chair	Local Arrangements
1	1953	Victoria, BC	R. Foster			
2	1954	Berkeley, CA	W. Wagener	P. Lightle		
3	1955	Spokane, WA	V. Nordin	C. Leaphart	G. Thomas	
4	1956	El Paso, TX	L. Gill	R. Davidson	V. Nordin	
5	1957	Salem, OR	G. Thomas	T. Childs	R. Gilbertson	
6	1958	Vancouver, BC	J. Kimmey	H. Offord	A. Parker	
7	1959	Pullman, WA	H. Offord	R. Foster	C. Shaw	
8	1960	Centralia, WA	A. Parker	F. Hawksworth	J. Parmeter	K. Shea
9	1961	Banff, AB	F. Hawksworth	J. Parmeter	A. Molnar	G. Thomas
10	1962	Victoria, BC	J. Parmeter	C. Shaw	K. Shea	R. McMinn
11	1963	Jackson, WY	C. Shaw	J. Bier	R. Scharpf	L. Farmer
12	1964	Berkeley, CA	K. Shea	R. Scharpf	C. Leaphart	H. Offord
13	1965	Kelowna, BC	J. Bier	H. Whitney	R. Bega	A. Molnar
14	1966	Bend, OR	C. Leaphart	D. Graham	G. Pentland	D. Graham
15	1967	Santa Fe, NM	A. Molnar	E. Wicker	L. Weir	P. Lightle
16	1968	Couer D'Alene, ID	S. Andrews	R. McMinn	J. Stewart	C. Leaphart
17	1969	Olympia, WA	G. Wallis	R. Gilbertson	F. Hawksworth	K. Russell
18	1970	Harrison Hot Springs, BC	R. Scharpf	H. Toko	A. Harvey	J. Roff
19	1971	Medford, OR	J. Baranyay	D. Graham	R. Smith	H. Bynum
20	1972	Victoria, BC	P. Lightle	A. McCain	L. Weir	D. Morrison
21	1973	Estes Park, CO	E. Wicker	R. Loomis	R. Gilbertson	J. Laut
22	1974	Monterey, CA	R. Bega	D. Hocking	J. Parmeter	
23	1975	Missoula, MT	H. Whitney	J. Byler	E. Wicker	O. Dooling
24	1976	Coos Bay, OR	L. Roth	K. Russell	L. Weir	J. Hadfield
25	1977	Victoria, BC	D. Graham	J. Laut	E. Nelson	W. Bloomberg
26	1978	Tucson, AZ	R. Smith	D. Drummond	L. Weir	R. Gilbertson
27	1979	Salem, OR	T. Laurent	T. Hinds	B. van der Kamp	L. Weir
28	1980	Pingree Park, CO	R. Gilbertson	O. Dooling	J. Laut	M. Schomaker
29	1981	Vernon, BC	L. Weir	C.G. Shaw III	J. Schwandt	D. Morrison
30	1982	Fallen Leaf	W. Bloomberg	W. Jacobi	E. Hansen	F. Cobb
31	1983	Coeur d'Alene, ID	J. Laut	S. Dubreuil	D. Johnson	J. Schwandt
32	1984	Taos, NM	T. Hinds	R. Hunt	J. Byler	J. Beatty
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—2010 (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		
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	D. Morrison	
56	2008	Missoula, MT	G. DeNitto	F. Baker	J. Schwandt	W. Littke	B. Lockman M. Jackson		
57	2009	Durango, CO	G. Filip	J. Adams	J. Schwandt	D. Shaw	K. Burns B. Jacobi J. Worrall R. Mask	R. Sturrock	
58	2010	Valemount BC	R. Sturrock	M. Fairweather	J. Schwandt	D. Goheen	M. Cleary R. Reich		J. Adams

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.



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Ralph Williams  
9650 S. Powerline Rd.  
Nampa, ID 83686-9408

We have lost track of the following  
Honorary Life Members. If you know of  
their whereabouts, please contact Fred  
Baker at 435-797-2550 or  
fred.baker@usu.edu

Ed Andrews  
Alvin Funk  
Bob Harvey  
John Hopkins  
Paul Lightle  
Don Norris  
Art Partridge  
Jack Roff  
Jim Trappe  
Lori Trummer  
Bob Tinnin  
Mike Sharon  
Richard B Smith  
Ed Wood  
Conrad Wessela

## DECEASED MEMBERS

Stuart "Stuie" Andrews	Ray Foster	Paul Keener	Lew Roth
Jesse Bedwell	Dave French	James Kimmey	Dave Schultz
Robert Bega	Lake S. Gill	Andrea Koonce	Charles G. Shaw
Warren Benedict	Clarence "Clancy" Gordon	Don Leaphart	Albert Slipp
John Bier	John Gynn	Tom McGrath	Willhelm Solheim
Bill Bloomberg	John Hansbrough	Neil E. McGregor	Albert Stage
Roy Bloomstrom	Hans Hansen	Jim Mielke	Phil Thomas
Thomas "Buck" Buchanan	Homer Hartman	D. Reed Miller	Willis Wagener
Don Buckland	George Harvey	Vergil Moss	Charles "Doc" Waters
Hubert "Hart" Bynum	Frank G. Hawksworth	Harrold Offord	Larry Weir
Elmer Canfield	Dwight Hester	Nagy Oshima	Ed Wicker
Ross Davidson	Tommy Hinds	Lee Paine	John Woo
Oscar Dooling	Brenton Howard	John Palmer	Ernest Wright
Norm Engelhart	John Hunt	John "Dick" Parmeter	Wolf Ziller
		Clarence Quick	



## **In Memoriam - W. Thomas (Tom) McGrath (April 27, 1933 – March 6, 2008)**

### **Retired Professor of the School of Forestry, Stephen F. Austin State University**

Tom was born and raised in Lincoln, Nebraska. He earned his bachelor's degree from the University of Montana and his Ph.D. in plant pathology from the University of Wisconsin in 1966. Tom's major focus was on the infection of jack pine by western gall rust and fusiform rust.

Tom married Sylvia Freeman Wallace in 1966. They moved to Nacogdoches in 1969 where they both joined the faculty of Stephen F. Austin State University. Tom was professor at the School of Forestry, where he taught forest pathology, fire, and dendrology until his retirement in 1996. Tom was an adventurer, living abroad in Italy, New Zealand and Australia in his youth and serving interim professor for six months in Kuala Lumpur, Malaysia in 1991.

Tom had two children, Sandra Jean, born in 1968, and Charles George, born in 1971, who were the center of the McGrath family life personally and professionally.

Tom also had a passion for smoke jumping. He joined the National Smoke Jumpers Association (NSA) in 1957. He spent most summers in Montana smoke jumping, participating in trail crews, or simply enjoying the great outdoors. He even spent the summer of 1988 working on a fieldcrew for the Forest Pest Management staff out of Missoula. Tom also loved flying and held a pilot's license for more than 50 years.

Tom last attended WIFDWC in Sedona, Arizona, in 2007.

Tom is remembered for his candid honesty, integrity, wit, and for his ability to out-walk nearly every student who studied with him.

Tom was preceded in death by his wife, Sylvia, and daughter, Sandy. He is survived by his son, Charles, of Dallas, and his sister, Sarah Aiken, of Laguna Niguel, California.



## **In Memoriam - Andrea (Andi) L. Koonce (December 31, 1951 – July 24, 2010)**

### **Program Manager, Forest Health Protection, Southern California Shared Service Area**

Andi was born in Denver, CO, but she grew up in Los Alamos, NM, graduating from Los Alamos High School in 1969. Andi went on to earn a B.S. in Political Science at Arizona State University in 1973, and a M.Sc. and Ph.D. from Oregon State University in 1981. Her areas of graduate study included forest science, forest ecology and forest pathology with Dr. Lew Roth (OSU) and Dr. Robert Martin (FS). Her graduate research focused on the interaction between fire and dwarf mistletoe in ponderosa pine. Upon graduation, Andi went to teach at the National School of Forest Sciences in Honduras and directed a tree improvement program in tropical pines. She returned to the United States and joined the faculty of the University of Wisconsin at Stevens Point to teach forestry courses. While there in 1984, she established the Fire Science Center, supporting research and extension activities related to prescribed fire in the Midwest. In 1988, she left academia and joined Forest Service Research as Project Leader of the Prescribed Fire Research Unit at the Forest Fire Laboratory in Riverside, Calif. This was a trail-blazing move for Andi, as she became one of the first female US Forest Service project leaders in fire research.

Andi was very instrumental in the early formation of the International Association of Wildland Fire, first by conceiving of a professional organization, and later by serving as one of the very first members of the Board of Directors in 1990. She helped formulate ideas of a high quality, refereed scientific journal to help establish fire science as an international recognized discipline. With other collaborators, this resulted in the funding and development of what we now know as the International Journal of Wildland Fire.

When the US Forest Service Pacific Southwest Research Station eliminated several lines of research due to funding reductions in 1996, Andi joined the Fire and Aviation Management Staff in the Forest Service's National Office. She provided expertise in fire planning and fuels before moving to Milwaukee, WI, to work as a fire staff specialist in the Forest Service's Eastern Regional Office. In this capacity, she was able to provide technical expertise in support of prescribed burning and fuels management to national forests in this 20-state area. She later returned to California as a Forest Pathologist on the San Bernardino National Forest, and later oversaw the Forest Health Protection Program for the four national forests in Southern California.





**Top Row:**

Harry Kope, Stefan Zeglen, Terry Shaw, Lori Winton, Megan Dudley, Michael Murray, Alan Kanaskie, Alex Woods, Lindsey Myers.

**Bottom Row:**

Ned Klopfenstein, Mary Lou Fairweather, Pete Angwin, Erin Havard, Rich Hunt, Richard Reich, Dave Shaw.





Top Row:

Phil Cannon, John Hanna, John Browning, Bart van der Kamp, Adriana van der Kamp, Dan Omdal, John Schwandt

Bottom Row:

Mr. In Twophotos, Kristen Chadwick, John King, Bob Edmunds, Walt Thies, Gail Thies, Nancy Gillette, Bro Kinloch





Top Row:

James Walla, John-Erich Haight, Will Littke, Chris Konchalski, Kevin Buxton, Ellen Goheen, Don Goheen, Mike Cruickshank

Bottom Row:

Jim Blodgett, Bruce Moltzan, Kathleen McKeever, Simren Brar, Kim Camilli, Blakey Lockman, Robin Mulvey, Heather MacLennan, Norma Stromberg-Jones, Stephanie Beauseigle





Top Row:

Holly Kearns, Gregg DeNitto, Angel Saavedra, Bill Jacobi, Rona Sturrock, Anne Marie Casper, Von Helmuth, Kennedy Boateng, David Rusch, Chris Hansen

Bottom Row:

Mike McWilliams, Jonas Rönneberg, Greg Filip, Michelle Cleary, Duncan Morrison, Deanna Danskin, GERAL McDonald, Simon Shamoun

# WIFDWC Over The Years...

**PROCEEDINGS OF THE TWENTY-EIGHTH ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE**  
 VERNON, BRITISH COLUMBIA, SEPTEMBER 1986

**Proceedings of the Twenty-Eighth Annual Western International Forest Disease Work Conference**  
 Pigeon Fork, Colorado  
 September 18-23, 1986

Compiled by:  
 J. R. Spongberg  
 Northern Pacific  
 Forest Service  
 Missoula, Montana

**Proceedings of the Twenty-Seventh Annual Western International Forest Disease Work Conference**  
 Salt Lake, Oregon  
 September 22-26, 1985

Compiled by:  
 J. R. Spongberg  
 Northern Pacific  
 Forest Service  
 Missoula, Montana

**PROCEEDINGS OF THE 29th ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE**  
 TRENCH, BRITISH COLUMBIA  
 SEPTEMBER 19-23, 1987

EDITED BY:  
 J. R. Spongberg  
 Northern Pacific  
 Forest Service  
 Missoula, Montana

**PROCEEDINGS OF THE ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE**  
 VICTORIA, BRITISH COLUMBIA  
 OCTOBER, 1977

**Proceedings of the 24th Annual Western International Forest Disease Work Conference**  
 Grand Bay, Oregon, September 18-21, 1979

**Proceedings of the 26th Annual Western International Forest Disease Work Conference**  
 Missoula, Montana, October 27-31, 1983

**Proceedings of the 21st Annual Western International Forest Disease Work Conference**  
 Estes Park, Colorado  
 October 1-5, 1973

**PROCEEDINGS OF THE THIRTYFIFTH ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE**

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**THIRTY SEVENTH ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE**  
 AND THE  
**FORTYETH ANNUAL WESTERN FOREST INSECT WORK CONFERENCE**

**Proceedings of the Thirty-Fifth Annual Western International Forest Disease Work Conference**  
 Reno, Nevada  
 August 2-7, 1987

**Proceedings of the Thirty-Fourth Annual Western International Forest Disease Work Conference**

Edited by:  
 J. R. Spongberg  
 Northern Pacific  
 Forest Service  
 Missoula, Montana

**Proceedings of the 32nd Annual Western International Forest Disease Work Conference**  
 Okanogan, Washington  
 September 24-27, 1985

**32nd WIFDWC PROCEEDINGS 1984**

**31st WIFDWC 1983**

Proceedings of the Western International Forest Disease Work Conference  
 Wenatchee, Washington  
 August 15-21, 1983

**Thirtieth Annual Western International Forest Disease Work Conference**  
 Palm Springs, California, September 1982

**Proceedings of the 46th ANNUAL WIFDWC**

RENO, NEVADA  
 September 28 - October 2, 1998

**45th Annual WIFDWC**

Prince George, BC  
 September 15 - 19, 1997

**PROCEEDINGS OF THE FORTY-FOURTH ANNUAL WIFDWC**

RENO, NEVADA, OREGON  
 September 18-23, 1996

**43rd Annual WIFDWC**

Whitefish, Montana  
 August 29 - September 1, 1995

**Proceedings of the 41st Joint Western International Forest Insect and Disease Work Conference**  
 Albuquerque, New Mexico  
 March 7-10, 1994

**WIFDWC**

Boise, Idaho  
 September 13-17, 1993

**WIFDWC**

Durango, Colorado  
 July 13-17, 1992

**WIFDWC**

Missoula, Montana  
 1991

**Proceedings of the 5th Annual Western International Forest Disease Work Conference**  
 Missoula, Montana  
 1969-1971

**Proceedings of the 1st Annual Western International Forest Disease Work Conference**  
 Missoula, Montana  
 1969

**Proceedings of the Fifty-Fourth Western International Forest Disease Work Conference**

Don L. Kiser, Friendship Centre  
 Grand Rapids, Michigan  
 October 2 to 6, 2006

**Proceedings of the Fifty-Third Western International Forest Disease Work Conference**

WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE  
 SNOW KING RESORT, JACKSON, WYOMING  
 AUGUST 24 - 29, 2005

**Proceedings of the 51st Annual Western International Forest Disease Work Conference**  
 Riverside Inn Conference Center  
 August 18 to 22, 2003

**PROCEEDINGS OF THE FIFTIETH ANNUAL WIFDWC**  
 PUEBLO VALLEY AVENUE, BENTONVILLE, ARIZONA  
 OCTOBER 1-5, 2002

**PROCEEDINGS OF THE FORTY-EIGHTH ANNUAL WIFDWC**

WAIKONAUA AVENUE  
 AUGUST 14 - 18, 2000

**Proceedings of the Fifty-First Annual Western International Forest Disease Work Conference**  
 Breckenridge, Colorado  
 September 13 to 17, 1999