PROCEEDINGS OF THE 30th ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE

Fallen Leaf Lake, California
September 1982

Proceedings of the 30th Annual Western International Forest Disease Work Conference

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Proceedings of the Thirtieth Western International Forest Disease Work Conference
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Foreword

The Thirtieth Annual Western International Forest Disease Work Conference was held September 12-16, 1982, at the Stanford Sierra Lodge, Fallen Leaf Lake, California. A fine meeting was organized by the local arrangement group. The formal program had very informative panel discussions, special papers, workshops, a fine-rainy-cold field trip, reports on new and modified projects, the banquet and a business meeting. Posters were a great addition to this year's meeting and we hope to see more at the next WIFDWC.

Seventy-nine registered for the conference with twenty-two guests and spouses and four children.

Officers for the 30th Conference were:

Bill Bloomberg	Chairman
Bill Jacobi	Secretary-Treasurer
Everett Hansen	Program Chairman
Greg Denitto	Local Arrangements
Bob Scharpf	
Dick Parmeter	
Fields Cobb	
Dick Parmeter	Field Trip
Bob Scharpf	

Western International Forest Disease Work Conference

1982

Welcoming Address

W.J. Bloomberg, Chairman

Welcome to the 30th Annual Meeting of WIFDWC in beautiful Lake Tahoe. So WIFDWC is 30 years old - not a bad age to be. It implies that the foolishness of youth has passed, yet the stodginess of middle age has not set in. Searching past proceedings for some guide to chairmen's remarks, I noted that at the Vernon meeting last year, Chairman Larry Weir "found an increasing disregard for publishing the deathless prose expounded by conference chairmen". At Pingree Park, 1980, Bob Gilbertson's comments were considered too unimportant to be included in the proceedings, also the fate of Tom Laurent's "deathless prose" at the 1979 Salem meeting. In 1978, while plaudits were given to several sterling organizing efforts, including Mrs. Gilbertson's shopping tour, Chairman Dick Smith's utterances, if any, were roundly expunged from the record. I suggest therefore, that the crucial question for WIFDWC is not which geographical region has the honor of giving rise to the Mississippi River, nor even whether forks should be held in the left or right hand, but whether WIFDWC really needs a chairman.

Hurrying on before someone attempts to answer the question, I am going to ask your indulgence if I break with tradition and make a few remarks about the past year in which I have been chairman. For most forestry professionals, it has not been a good one, and forest pathology has more than its share of walking wounded. Since the only thing to do about a bad experience is to learn from it, I look critically at my profession. I look at my fellow professionals and see devoted workers. Enthusiastic, certainly. Willing, almost to a fault, to cooperate with other professionals in tackling complex problems. Seldom do they become exercised about claiming recognition for their contribution to the solutions. In brief, good fellows all; but as a profession, rather naive, one might say.

Comparing the self-image of the profession with those of other forestry professions, one is struck by a lack of "cutting edge". By this I mean the lack of distinct territorial expertise and professional "assertiveness". To assert the importance of one's profession is not distasteful; in today's administrational hurly-burly, it is essential. To claim an area of expertise in the forestry domain is an obligation to our profession. Perhaps because the discipline of forest pathology becomes often entwined with those of genetics, ecology and physiology in the solution of complex problems, pathological aspects become diffused. This has never deterred forest pathologists from collaborating with great gusto, but they owe it to their profession to extol the importance of the discipline in the solution of forestry problems. They owe it to the great forest pathologists who preceded us and to those who yet hope to make their careers in forest pathology.

PROGRAM

30th ANNUAL WIFDWC

1982

Sunday, September 12

3:00 p.m.: Registration and check-in

6:00 p.m.: B-B-Q Western style

Later: Conviviality, etc.

Monday, September 13

8:30 a.m.: Welcome - Bill Bloomberg

Keynote: Glen Smith, Planning Coordinator, Lake Tahoe Management Unit, USDA Forest Service

Project Status

1:30 p.m.: Panel-------Stress and Plant Disease

Terry Shaw, PNW, Juneau, Moderator

Rex Cates, Univ. New Mexico

Phil Wargo, NE Experiment Station

Walt Thies, PNW, Corvallis

Bill Farr, PNW, Juneau

Special Papers

J. Pronos - USFS, R-5 --- Trends in Ozone Injury to Pines in The Sierra Nevada of California.

D. Volger - USFS, R-5 --- Ozone Injury Symptoms and Height Growth of Planted Ponderosa Pine.

W. G. Thies & E. E. Nelson - USFS, PNW --- Survival of Douglas-fir Injected with the Fumigants Chloropicrin or Methylisothiocyanate.

7:30 p.m.: Workshop - Identification and Taxonomy of Wood Destroying Basidiomycetes. Bob Gilbertson, Univ. Arizona.

Tuesday, September 14

8:30 a.m.: Panel - The Future of Forest Pathology.

Everett Hansen, Oregon State Univ., Moderator
Bob Loomis, FPM, Washington, D.C.
Earl Nelson, PNW, Corvallis
Roy Whitney, Canadian Forestry Service, Ontario
Dave Adams, California Department of Forestry.

Special Papers

M. Wingfield, Univ. Minn. --- Insect Associates of the Pine Wood Nematode in Minnesota.

W. H. Livingston & M. L. Brenner, Univ. Minn. --- Response of Eastern Dwarf Mistletoe Infections to Applications of a Plant Growth Regulator.

J. Worrall, J. R. Parmeter, Jr., and F. W. Cobb, Univ. Calif. --- Host Preference in Fomes annosus.

1:30 p.m.: Panel - Who are the players in IPM, and is it something that new.

Fields Cobb, UC Berkeley, Moderator.
Mark Edwards, Woodland SErvices, Ukiah, Calif.
Bob Heald, U.C. Blodgett Forest, Georgetown, Calif.
Jerry Koenigs, International Paper, Research Triangle, N.C.
An Entomologist

Special Papers

E. Michaels & G. Chastagner, WSU. --- Conditions which influence ascospore release in Swiss Needle Cast of Douglas-fir.

R. Edmonds, C. Driver, K. Leslie, R. Hu, S. Frankel & T. Jordan, Univ. Wash. --- Overview of control and impact of Heterobasidion annosum in Western Hemlock.

6:00 p.m.: Banquet

Wednesday, September 15

8:45 a.m.: All day field trip. Pathology in high use recreation areas.

Thursday, September 16

8:30 a.m.: Panel - Cry Wolf -- The consequences of conflicting or erroneous information.

Greg Filip, FPM, Portland, Moderator
Craig Schmidt, FPM, Portland
Borys Tkacz, FPM, Ogden
Randy Fuller, FPM, Denver.

11:00 a.m.: Business Meeting

12:00 noon: Lunch and Adjournment.

INITIAL TESTING OF THE SHIGOMETER IN HEALTHY EVEN-AGED STANDS OF WESTERN HEMLOCK AND SITKA SPRUCE IN SOUTHEAST ALASKA

Wilbur A. Farr and Charles G. Shaw III[1/]

Introduction

Is the Shigometer[2/] a useful tool for detecting internal decay in trees or for measuring tree vigor? This question has no doubt been asked by many researchers since the instrument was introduced in the early 1970's. Even though some workers are convinced that the Shigometer is a useful diagnostic tool, many others remain skeptical.

The Shigometer is an instrument that measures electrical resistance (ER) in thousands of ohms (Kohms). For use on trees it has two different attachments. One is a 15-cm long probe (Shigo and Shigo 1974) that can be inserted into a drilled hole to measure internal resistance within living trees or utility poles. The other, a needle probe (Shigo, Blanchard, and Smith 1975; Wargo and Skutt 1975;), has two stainless steel needles that can be pushed through bark and cambium of a tree to measure electrical resistance within the cambial region. The former is used to detect internal decay, the latter for other uses, including studies of tree vigor.

Over 20 articles have been published on the use of the Shigometer. They include studies to detect internal decay in trees and utility poles (Shigo and Shigo 1974, Shortle 1982, Shortle and others 1978, Skutt and others 1972, Tatter and others 1972, Tatter and others 1974), to detect wound areas (McNiel and Hensley 1980, Shortle and others 1977, Sylvia and Tatter 1978), to monitor water potential (Dixon and others 1978), to develop hazard rating systems (Davis and others 1980), to indicate phloem thickness (Carter and Blanchard 1978, Cole 1980, Cole and Jensen 1980), to measure growth and stress in trees (Kostka and Sherald 1982; Schaeffer and others;[3/] Shigo and others 1975; Shortle and others 1977, 1979; Smith and others 1976; Wargo 1977; Wargo and Skutt 1975), and to select trees to be thinned in uneven-aged stands (Shortle and others 1979). Interest in the instrument continues and several more articles are being prepared.

[1/]Wilbur A. Farr is a mensurationist and Charles G. Shaw III is a pathologist with the USDA Forest Service, Pacific Northwest Forest and Range Experiment Station, Forestry Sciences Laboratory, Juneau, Alaska.

[2/]Mention of a product is for information only and should not be considered an endorsement by the U.S. Department of Agriculture or Forest Service.

[3/]Schaeffer, Bruce, Frank G. Hawksworth, and Paul Beemsterboer. Effects of dwarf mistletoe and vigor classes on electrical resistance in lodgepole pine. (For. Sci., in press).

The Shigometer was initially developed as a diagnostic tool to detect internal decay in trees. More recent efforts have included its use to detect stress in trees as a result of insect or disease problems. We are aware of only one attempt to use it to aid in the selection of trees to be removed by thinning (Shortle and others 1979). In one other instance the Shigometer was used in healthy stands to assess electrical resistance following thinning and fertilization (Smith and others 1976).

In 1979 we initiated a study on the use of the Shigometer to assess tree vigor in healthy, natural, even-aged stands of western hemlock (Tsuga heterophylla (Raf.) Sarg.) and Sitka spruce (Picea sitchensis (Bong.) Carr.) in southeast Alaska. The objective of the study was to determine if electrical resistance in the cambial region of young-growth western hemlock and Sitka spruce was correlated with various tree parameters (i.e., species, crown class, diameter, height, and diameter growth), site, or time of measurement. This report summarizes work to date and outlines plans for continuing work over the next few years.

Basic data

In 1974 we began a study of the effects of stand density on the growth and yield of hemlock and spruce growing in mixed, even-aged stands.[4/] Since then about 55 installations have been established in stands covering ages from 10 to 100 years and the range of sites commonly found on commercial forest land. An installation typically contains four 1-acre plots, including one unthinned control acre and three levels of thinning. Growth measurements are regularly taken on trees within a square 1/5-acre plot located in the center of each acre plot.

In 13 of these installations, we measured ER for all trees on thinned plots, and most or all trees on control plots. Measurements of ER were obtained by inserting the double needle moisture probe through the bark and cambial zone just above breast height (1.3 m). The needles were inserted vertically and three readings were taken per tree at approximately 120-degree intervals around the stem. Additional readings were recorded if a highly deviant ER was obtained.

Electrical resistance was measured in the spring and fall of 1979 and in the summer of 1982. Measurements of diameter, diameter growth, crown class, tree condition, site, age, and other variables were obtained from data files maintained in connection with the stand density study.

[4/] Farr, Wilbur A. The effects of stand density upon growth and yield of hemlock-spruce stands in coastal Alaska. Study plan FS-PNW-1652: S-8; 1973 (revised 1976). 22 p. On file at Forestry Sciences Laboratory, P.O. Box 909, Juneau, Alaska 99802.

Hypothetical relationships between electrical resistance, diameter, and diameter growth

Figures 1a, 2a, and 3a show hypothetical relationships between ER, tree diameter, and diameter growth for well-stocked, even-aged stands. We believe that ER should be relatively high for understory trees and lower for overstory trees (fig. 1a). The relationship should be curvilinear in form.

We also hypothosize that the relationship between diameter increment and ER is also curvilinear in form for well-stocked, even-aged stands (fig. 2a). Overstory trees should, on the average, have higher diameter increment and lower ER than understory trees.

The relationship between diameter increment and diameter should generally be linear in form for well-stocked stands (fig. 3a).

Actual relationships between electrical resistance, tree diameter, and diameter increment

Scattergrams were prepared for the relationships: ER over diameter, diameter increment over ER, and diameter increment over diameter. These were examined for both hemlock and spruce on all plots each time ER was measured.

Only one of over 100 sets of scattergrams is shown (fig. 1b, 2b, and 3b), but from these it is apparent that the hypothetical curves fit the basic data well if there is a wide enough range of tree diameters and crown classes present.

It is noteworthy, however, that if we had fit a mathematical curve through the data points in each figure, there would still remain considerable unexplained variation in all three relationships. For some plots there was no correlation between ER and diameter or between diameter increment and ER. For others these correlations were highly significant.

Other problems of differences between species, plots, and time of year

Hemlock has higher ER readings than did spruce for trees of similar sizes. This was consistent for all plots and times of sampling. Similar differences occur for other species (Davis and others 1979).

The scattergrams clearly indicate that ER has to be evaluated on a plot-by-plot basis because significant differences in ER occurred between some plots that could not be explained by differences in site, soil, or other stand characteristics. If relationships do vary over short distances within stands that appear to be on uniform sites, then the development of useful relationships becomes difficult. In contrast, other stands showed little difference in the relationships from one plot to the next.

Time of year measurements are taken is also important. Electrical resistance was lower in summer (during the active part of the growing season) than in the spring or fall (before or after the growing season). Similar effects of seasonal changes have been reported for red oak, red maple, and white pine (Davis and others 1979).

In all, there are several sources of variation, some easily accounted for and other not easily accounted for. Users of the Shigometer should expect considerable variation in ER readings both within and among trees and plots, even among a population of supposedly healthy individuals.

What has the data shown so far?

Analysis of these data indicate that the hypothetical relationships (fig. 1a, 2a, and 3a) fit most data sets reasonably well. There is, however, considerable variation in the degree of fit for some plots.

The scattergrams have proven very useful for detecting obvious outliers in the data sets. Invariably these outliers represent measurement errors or trees under severe stress resulting from excessive lean or poor crown development.

Trees with much higher-than-average ER typically have poor growth rates and are usually in the intermediate or suppressed crown classes. This is not always true, however, because even after the obviously poor producers are eliminated, there remains considerable variation in the relationship between ER and diameter. This variation can be seen in figures 1b and 2b where trees with lower-than-average ER cover a wide range of diameters and diameter increment.

Most of the poor producers can probably be identified by careful observation in the field or simply by removing all suppressed and intermediate trees from the analysis. The poor producers are usually overtopped, have relatively poor crowns, broken tops, damaged roots, or other problems. The important point is that even though there may be a good correlation between ER and diameter, the information may add very little to what we already know, or can be easily obtain elsewhere. In the case of selection for thinning, it is the trees in the upper crown classes with no apparent problems we are most concerned about.

In the next phase of this study we will identify all trees on the scattergrams for several plots by tree number, and then revisit them in the field in an attempt to evaluate the overstory trees, in light of knowing their ER readings and growth rates. We hope to gain insight into what characterizes relatively good producers versus poor producers in thinned and unthinned stands. From these examinations we may be able to improve the guidelines for selecting leave trees during thinning.

Conclusions

1. The Shigometer does provide a relative measure of tree vigor in even-aged stands of western hemlock and Sitka spruce in southeast Alaska, but there is often considerable variation in readings of electrical resistance within and among trees.

2. To be most useful, electrical resistance over diameter relationships should be plotted in the field. Trees having higher-than-average electrical resistance for their diameter should be carefully examined for characteristics possibly responsible for the high readings.

3. Additional research is needed to evaluate how useful the Shigometer may be in assessing tree vigor in healthy, even-aged stands and how the instrument might be used to select leave trees in thinning operations.

Future efforts in southeast Alaska

We will continue to use the Shigometer. Our principal effort will be to determine if the instrument can provide meaningful information that we do not already know about the 13 installations, or can easily learn from direct observation.

We believe the Shigometer's greatest utility may be in thinning research, especially if use of the instrument could easily identify the best trees to be left during thinning.

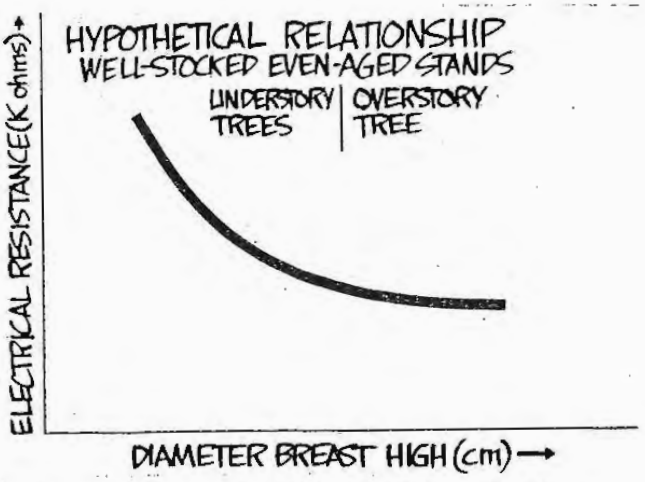

Figure 1a.--Hypothetical relationship between electrical resistance and d.b.h. for well-stocked even-aged stands.

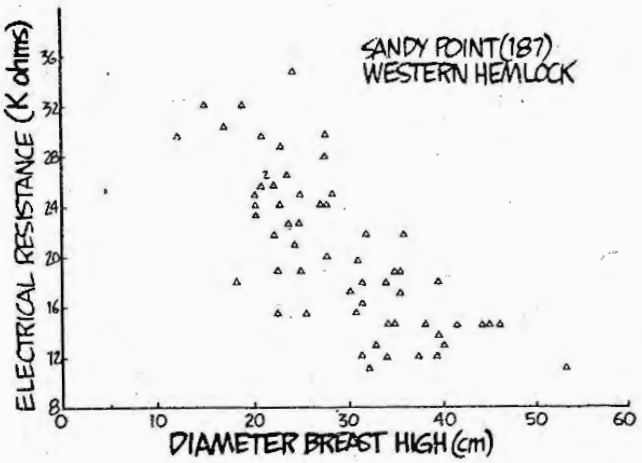

Figure 1b.--Relationship between electrical resistance and d.b.h. for western hemlock in a natural, well-stocked, even-aged stand at Sandy Point, Alaska.

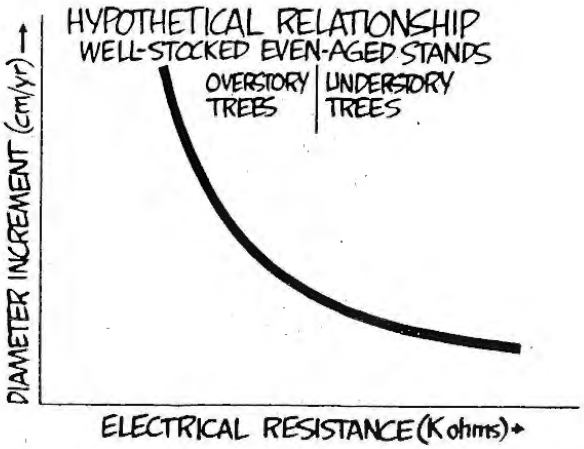

Figure 2a.--Hypothetical relationship between diameter increment and electrical resistance for well-stocked, even-aged stands.

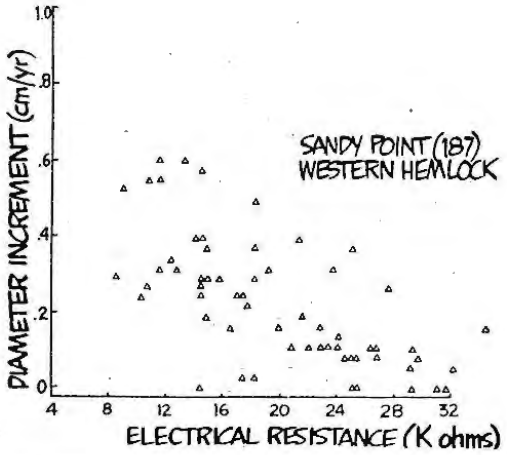

Figure 2b.--Relationship between diameter growth and electrical resistance for western hemlock in a natural, well-stocked, even-aged stand at Sandy Point, Alaska.

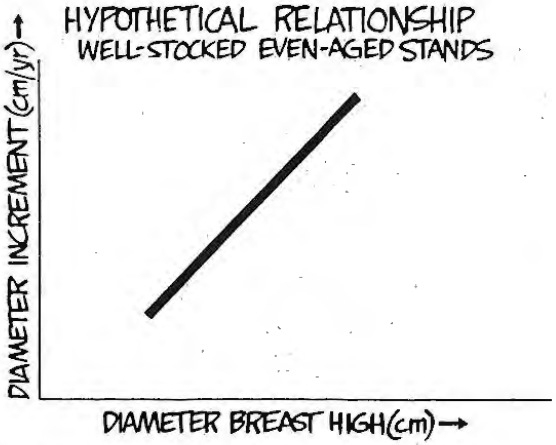

Figure 3a.--Hypothetical relationship between diameter increment and d.b.h. for well-stocked, even-aged stands.

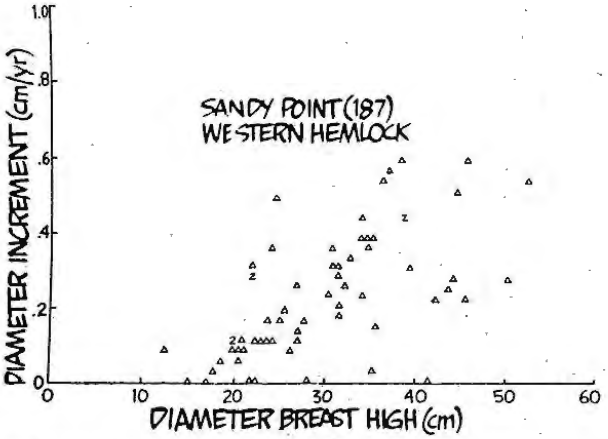

Figure 3b.--Relationship between diameter growth and d.b.h. for western hemlock in a natural, well-stocked, even-aged stand at Sandy Point, Alaska.

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GROWTH LOSS AS A MEASURE OF STRESS IN SECOND-GROWTH DOUGLAS-FIR INFECTED BY PHELLINUS WEIRII

Walter G. Thies[1]

Phellinus weirii (Murr.) Gilb., the cause of laminated root rot, can infect nearly all commercially important coniferous species in Western United States and Canada; it probably causes the greatest loss and has been most intensively studied in stands of second-growth Douglas-fir [Pseudotsuga menziesii (Mirb.) Franco]. When infected trees die, the pathogen persists saprophytically in dead roots. Infection begins in young conifers when roots contact infected, living root systems or infested roots from killed or harvested trees. The infection spreads between living trees via root contact (Wallis and Reynolds 1965). As the fungus advances along a tree's roots, roots distal to the fungus are killed, denying the tree water and nutrients necessary for growth. Crown symptoms may appear 5 to 15 years following initial infection (Wallis 1976). As roots decay, structural support declines. Eventually, the tree may be windthrown.

Although losses to P. weirii are most conspicuous as mortality, a tree may also suffer reduced height or diameter growth for several years before death (Gillette 1975, Ford 1977, Bloomberg and Wallis 1979). Significant growth loss (volume) was associated with P. weirii infected Douglas-fir from a stand in the Oregon Coast Ranges (Thies 1980).

I will present a summary of methods and results from an ongoing study. The data are from a study to measure the growth reduction induced by P. weirii in second-growth Douglas-fir.

METHODS

The study area is on the west slope of the Cascade Range near Foster, Oregon, on a nearly flat Saturn gravel alluvium at an elevation of 400 m. The study area classified as site II (McArdle and others 1949) occupies approximately 2.7 ha in a naturally regenerated 60-year-old stand. In 1980, stand density was 485 trees/ha, of which 93 percent were Douglas-fir, 4 percent were western hemlock (Tsuga heterophylla), and 3 percent were hardwoods. Total basal area was 23.6 m^2/ha.

Within the study area, each tree 12.7-cm d.b.h. or larger was numbered, its d.b.h. recorded, and its location plotted on a grid map (Thies and Hoopes 1979). Dead trees were included if all growth rings at breast height were distinct. After the area was mapped, 15 circular 0.04-ha plots were placed at random on the study area with the provisions that they not overlap nor be closer than 12 m from the study area boundary. Trees within plots were termed plot trees while all other trees in the study area were termed border trees. All trees in the study area were felled in April 1980. Each tree was measured for total height and height of primary nodes from

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the tip along the stem to the point where nodal location became indistinct, usually between the 25th and 45th node. Stem age at nodes was verified by counting annual rings at log ends and then sectioning as needed to locate missed nodes. A cross-section 5-cm thick was removed from each plot tree at the stump height of 1.4 m and at each 4-m interval thereafter for the length of the stem to a 10-cm-diameter (inside bark) top. One additional disk was removed midway between the 10-cm top disk and the tip. The height of each disk was recorded. From each of the border trees, a cross-sectional disk was removed at breast height only. Bark thickness and diameter outside bark were recorded for each disk. Each disk was labeled with tree number, disk number, and north point, placed in a plastic bag, and stored at -18°C.

Each stump top was examined for typical stain or advanced decay from P. weirii infection (Wallis 1976). When P. weirii symptoms were found, infection was verified by culturing from the stained or decayed areas. P. weirii was identified from morphological features of the colony (Nelson 1975).

The stump and roots from each plot tree were bulldozed from the soil, cleaned, and examined in August 1980. The presence of decay or ectotrophic mycelium of P. weirii (Buckland and others 1954) was recorded.

Sequential radial growth (SRG) measurements were recorded to the nearest 0.25 mm along three radii of each disk at azimuths of 0°, 120°, and 240°. The SRG measurements for each radius were reproportioned so that the length of radius equaled the mean disk radius. Mean disk radius was determined by subtracting mean bark thickness from half the disk diameter as measured with a diameter tape outside the bark. Data were combined for each disk to provide average radial growth. These data were used to calculate basal area and volume, and to produce stem profiles (Herman and others 1975). Stem profiles provide a visual edit of SRG data.

Tree volume was calculated for each year represented by an annual ring on the lowest (stump) disk. The segments cut from each tree were classified by origin as stump, lower bole, upper bole, or top. Volume at any year was found by summing the total volume calculated for each segment for that year.

Volumes of stem segments were calculated as follows. The stump was treated as a cylinder equal in height and diameter to the top of the lowest (stump) disk. Sections partly or wholly within the first 20 percent of total height (for trees over 7.6 m tall) were designated "lower bole" and were treated as the frustum of a cone. The upper 80 percent of a tree exclusive of tip and the entire bole except stump and tip of trees under 7.6 m tall were designated "upper bole" and were treated as the frustum of a paraboloid (Smalian formula). The top segment, from the top disk to the tree tip, was treated as a cone.

Arney's (1973) competitive stress index (CSI) was computed for each plot tree at specified years during stand development. This index represents a quantification of the relative stress placed on a tree by its neighbors. It does not attempt to identify the source of stress (e.g., competition for light, nutrients, moisture) but only to quantify relative

levels of competition for growing space (crown overlap). The degree of stress exerted on a tree is assumed to be directly proportional to the area of its growing space that is overlapped by that of neighboring trees. Computing the index requires data on diameter breast height outside bark and distances between trees in the study area. Appropriate data were collected from all border trees and plot trees. Western hemlock was treated as Douglas-fir for calculating CSI contributions. The hemlocks were small and scattered, so this treatment is assumed to have added little error to the total CSI of any plot tree.

Data from healthy plot trees were used to establish a regression equation defining the final 10 year (1970-80) volume growth based on volume growth during the preceding 10 years (1960-70). With this regression equation, the final 10 years of growth of each infected tree was predicted from its growth during the preceding 10 years. The difference between predicted and actual growth of infected trees is assumed to be a reasonable estimate of the growth lost due to the disease. A paired t-test was used to evaluate differences between predicted and actual volume increases for infected trees for the decade 1970-80. Selection of 10 years as the predictive period was a compromise between retaining a high correlation coefficient and predicting growth for the longest possible final period.

A preliminary determination of the impact of the disease on height growth was made by comparing the height growth of 75 randomly selected infected with noninfected plot trees. Each infected tree was paired with the most similar noninfected tree based on their CSI in 1980 (CSI 80) and their heights in 1960. A paired t-test was used to evaluate differences in height growth between infected and noninfected paired trees for the period 1960-80.

All statistical tests were made at the .05 (significant) or .01 (highly significant) probability levels using the standard statistical procedures (Snedecor and Cochran 1967).

RESULTS

There were significant differences between regression-predicted volume growth and actual growth of infected plot trees. The mean growth loss of all infected plot trees was 2.1 percent of their predicted harvest volume and 8.3 percent of their predicted final 10-year volume growth.

Of 111 live infected plot trees, 13 were found to have a hollow stump. Highly significant differences were found between predicted volume and actual volume of hollow, infected trees. The mean growth of hollow infected trees was found to be 3.3 percent more than their predicted harvest volume and 16.2 percent more than their predicted volume growth in the final 10 years. Additionally, none of the hollow trees exhibited the crown symptoms typical of advanced stages of the disease, and it was not possible to isolate P. weirii from wood collected more than a few millimeters beyond the limit of the hollow. All hollow stumps were associated with P. weirii infections.

Final 10-year growth volumes for infected trees were predicted using a linear regression equation determined from noninfected tree growth and CSI

data: Volume 1970-80 (m^3) = 0.008456 + 1.14297 volume 1960-70 (m^3) - 0.0001207 CSI 80. The sample number (n) was 159, the standard error of the estimate ($S_{y.x}$) was 0.0452219, and the coefficient of determination (R^2) was 0.93. A multiple regression analysis of volume 1970-80 on volume 1960-70 and CSI 80 yielded a highly significant R^2, with volume being a highly significant independent variable and CSI 80 being a significant independent variable.

There was a significant difference in the mean height growth of paired infected and noninfected trees. Although the mean heights of paired infected and noninfected trees (21.15 m and 21.21 m, respectively) were similar in 1960, by 1980 the mean height of infected trees was less than that of the noninfected trees (26.21 m and 26.79 m, respectively).

Of the 1,299 trees examined on the study area, 36 were hardwoods while 1,263 were trees susceptible to P. weirii (1,215 Douglas-fir, 56 western hemlocks). Of the 1,263 susceptible trees on the study area, 544 (43.1 percent) were found to be infected by P. weirii. Of the 235 dead susceptible trees on the study area, 195 (83.0 percent) were found to be infected by P. weirii. In 1980, 15.4 percent of the susceptible trees had been infected by P. weirii and were dead.

DISCUSSION

This study demonstrated that P. weirii can induce a volume growth loss in Douglas-fir in a stand on the west slope of the Cascade Range; however, that loss appears to be very small. The mean growth loss of infected trees was 2 percent of their predicted harvest volume and 8 percent of their predicted volume growth in the final 10 years. This comparison includes all plot trees identified as infected (including those on which only one root was infected but little if any of the root system destroyed) but excludes dead infected trees. Loss in height growth paralleled the loss in volume growth.

The growth loss reported for this stand in the Cascade Range is less than that reported from a similar study of Douglas-fir in a stand in the Oregon Coast Ranges (Thies 1980). In the earlier study, growth loss of live infected trees was found to be 6 percent of the predicted harvest volume and 12 percent of the predicted volume growth in the final 10 years (Thies 1980).

Two patterns of stump-top-stain were associated with P. weirii infection. Usually the stain was located near the sapwood-heartwood interface; in early stages the stain appears as spots or crescents. Later, a complete or nearly complete ring of stain forms and is indicative of a highly stressed tree. A second pattern occurs where the stain and advanced decay are limited to the center of the tree and always within the heartwood. In advanced stages, the tree will be hollow at the stump, but the hollow seldom extends as far as breast height. Statistically significant differences were found between predicted volume and actual volume of hollow-stumped infected trees. The mean growth of hollow-stumped infected trees was found to be 3 percent more than their predicted harvest volume and 16 percent more than their predicted volume growth in the final 10 years. The observation that some of the infected trees were associated

with both faster than expected growth and an apparent limitation of the fungus suggests the possibility of an active defense mechanism. An alternative explanation is that the hollow is the result of the site and method of attack by the fungus. Often the only infected root associated with a hollow stump will be small and originating from the bottom of the stump. The mechanism by which the fungus is apparently limited in its attack on the tree is worthy of additional study.

Losses to P. weirii on this study area from mortality were far more important than losses were from growth slowdown. Approximately 15 percent of the susceptible trees in the study area (approximating 15 percent of the stand volume) were killed by P. weirii, while growth loss amounted to 2 percent of the harvest volume of 43 percent of the trees. Thus unrecovered mortality, caused by P. weirii, will likely represent a much larger loss to the land manager than will growth loss induced by P. weirii.

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DEFOLIATION-INDUCED CHANGES IN TREE ROOTS FAVOR INFECTION BY ARMILLARIA MELLIA

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In the deciduous forests of northeastern United States, Armillaria mellea (Vahl. ex Fr.) Kummer (Armillariella mellea ex Fr. Karst) would be of little consequence were it not for stresses such as defoliation and drought. The fungus would function mostly as a decay organism because it apparently lacks the ability to infect and colonize roots of unstressed trees even though rhizomorphs of A. mellea colonize the outer bark of most tree roots. This external colonization of the roots by rhizomorphs seems to be the rule rather than the exception for most tree species and on most sites in the Northeast.

Colonization of the roots by rhizomorphs of A. mellea places the fungus in a position to take advantage of changes induced by stress; changes that stimulate vigorous hyphal growth and changes that reduce the ability of root tissues to respond and resist aggressive fungal growth. I propose that many of the chemical changes induced by defoliation (and other stresses) meet the physiological requirements of Armillaria mellea and that this interaction results in diseased roots.

The Interactions

Carbohydrates - The dominant changes in the roots induced by defoliation occur among the carbohydrates (Parker and Houston 1971, Wargo 1972, Wargo et al. 1972). Starch content is lowered substantially and in many trees that are defoliated completely and refoliate, starch content is depleted. Corresponding to this decrease in starch is a decrease in sucrose levels in the bark and outerwood of the roots. In contrast, levels of glucose and fructose increase, especially in the outerwood-cambial zone. Concentrations can be 4 to 5 times higher than the seasonal high that occurs normally in the roots in the spring when carbohydrates are mobilized for growth (Wargo 1971). The increase in glucose in the roots of defoliated trees is important to Armillaria mellea, because this fungus is a glucose fungus (Garraway 1974). Although it can grow on many carbon sources, its growth on glucose or polymers of glucose, like maltose and starch, can be 1.5 to 3 times higher than growth on other sources (Wargo 1981a). The enhancement of growth of A. mellea on extracts from roots of defoliated trees is attributable to higher glucose levels (Wargo 1972).

Glucose not only stimulates rapid growth of A. mellea but also enables the fungus to grow in the presence of inhibitory phenols such as gallic acid (Wargo 1980). Gallic acid, released when bark tannins are hydrolyzed, can inhibit and sometimes kill isolates of A. mellea. However when more glucose is available the fungus can not only overcome the inhibition by gallic acid but even use the oxidized phenol as an additional carbon source and grow more vigorously than on glucose alone (Wargo 1980, 1981b). This also occurs with other phenol compounds.

Nitrogen - Defoliation alters nitrogen metabolism and the levels of various amino acids. Alanine, asparagine, cysteine, histidine, leucine, isoleucine, proline, theronine, and tyrosine increase in the bark and wood of roots of defoliated trees and seedlings (Wargo 1972, Parker and Patton 1975). Of this group alanine and asparagine were shown (individually) to be very satisfactory and leucine moderately satisfactory for growth of A. mellea (Weinhold and Garraway 1966). The best nitrogen source in vitro was casein hydrolysate, which is a mixture of all the common protein amino acids. The fungus responds to the overall increase in amino nitrogen. Defoliation, like drought, another stress that predisposes trees to attack by A. mellea, causes increases in the overall level of amino nitrogen (Parker and Patton 1975).

In current studies of the growth of A. mellea on extracts from the bark of oak roots, supplemental nitrogen is critical for the oxidation of phenolics in the extract. Without supplemental nitrogen (asparagine), oxidation of the phenols, as indicated by an increased level of "browned" compounds (absorbance 450 Å), is limited and so is growth. Additional growth and eventual utilization of the oxidized phenolics as carbon sources depends on supplements of both nitrogen and glucose. Growth in the extracts in the presence of glucose and asparagine was 3 to 4 times as great as with either alone. Growth in the presence of phenolics in the extract appeared to be a two-phase reaction, first the oxidation of the phenols; then the utilization of the oxidized phenols as energy sources.

Successful colonization of root tissue may depend on the fungus's ability to oxidize phenols, and high concentrations of phenols would make the tissues more resistant to colonization. Defoliation can decrease certain phenolics in root tissue (Parker and Patton 1975). Also the general level of phenolic compounds available for mobilization against fungal attack may be reduced by defoliation. In oaks, phenolics or their precursors are produced in the leaves and translocated to the lower portions of the tree (Hathaway 1958). When leaves are removed by defoliating insects the source of phenols is also removed.

Lytic Enzymes - Defoliation also affects activities of enzymes that are capable of lysing polymers of chitin and glucans (Wargo 1976). Since the hyphal walls of A. mellea contain chitin and Bl, 3-glucan, they are vulnerable to the lytic action of these enzymes in the root tissues. Indeed, enzymes with activities for chitin and laminarin (B-1, 3-glucan) extracted from oak and maple tissues were able to dissolve prepared cell wall of A. mellea (Wargo 1975).

While the role of these enzymes in resistance to diseases caused by aggressive primary parasites, such as in Verticillium wilt of tomatoes, is doubtful (Pegg 1977) they could function in healthy tissue against weakly pathogenic agents (Wargo 1976).

Ethanol - Defoliation may result in ethanol production in the roots. Removal of leaves reduces water loss from the soil through transpiration and this, coupled with heavy or frequent rains during defoliation, could result in temporary anaerobic conditions on certain sites. Our studies with naturally defoliated trees and artificially defoliated seedlings confirm that ethanol is produced in roots of defoliated trees under anaerobic soil conditions. Our studies also confirm that ethanol concentrations tend to be higher in roots of defoliated seedlings. Because water is not being transpired, ethanol is not carried away from the roots by the sapstream.

Like glucose, ethanol enables A. mellea to grow rapidly in the presence of potentially inhibitory compounds (Wargo 1980), and it also increases the ability of the fungus to use carbon sources other than glucose for growth (Garraway 1974).

The production of ethanol in roots of defoliated trees has an additional effect. The tree's ability to survive anaerobiosis may depend on the amount of food reserves present. Since defoliation reduces the stored reserves, roots of defoliated trees are more vulnerable to the adverse effects of anaerobic conditions. Their deaths could provide A. mellea with initial entrance points from which the fungus could readily spread into still living but stressed tissues (Wargo 1981c).

Strain of fungus - A final consideration and perhaps most important in this "stress-attack" interaction is the strain or biological species of the fungus we call Armillaria mellea. There are at least ten reproductively distinct groups of A. mellea in North America and these are the equivalent of biological species (Anderson and Ullrich 1979). Within each of these species several clones can exist with as many as six in close proximity to one another (Ullrich and Anderson 1978). The larger number of clones is characteristic of the eastern deciduous forests in contrast to the western conifer forests (Anderson et al. 1979). This genetic diversity can result in diverse pathogenicity with some "species" being more pathogenic and/or more virulent than others (Rishbeth 1982). This explains why some "isolates" of A. mellea can be primary pathogens on pine (Shaw 1977) and why other "isolates" are secondary pathogens that attack trees only after they are defoliated (Wargo 1977, Wargo and Houston 1974).

Isolates are also distinct physiologically, as indicated by their different abilities to metabolize phenolic compounds (Wargo 1980, 1981b). These metabolic differences certainly become involved in the stress-change-fungus interaction. The genetic and consequently the physiological diversity of A. mellea helps to explain why in the Northeast mortality caused by the fungus can be localized when the stress has been widespread, why different species are attacked preferentially, and why stress is needed to initiate attack.

Hypothesis

Rhizomorphs of Armillaria mellea are on the surface of the root's bark. At many places among the rhizomorph network, hyphae penetrate the outer bark and "challenge" the inner tissues. Lytic enzymes continually dissolve the invading hyphal tips. The glucose level of the tissue is not high enough nor is enough nitrogen present in a form readily utilized for vigorous fungal growth. Tannins and other phenols in the bark inhibit further growth of the fungus. Phenol oxidase production by the fungus is low and any phenols that are oxidized are quickly reduced back to the phenolic state. The healthy tree resists invasion by A. mellea. Then the tree is stressed; changes occur in the roots that adversely affect the host's enzyme defense mechanisms, increase the energy available to the fungus, and enable it to alter and metabolize inhibitory compounds; then colonization takes place.

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Panel: The Future of Forest Pathology

THE FUTURE OF FOREST PATHOLOGY
Everett Hansen - Moderator

Introductory Remarks

In true scholarly fashion, I want to start this morning's discussion on the future by looking to the past. I'm always surprised at how much fun it is to read old WIFDWC's. There seems to be no subject immune to the pathologist's wit - except the future; there pathos seems to rule. Just last year Norm Alexander, in his keynote address, admonished "Don't huddle about in dark caves asking your fellow caveman if it's worth it!" We are a paranoid bunch when it comes to the future. WIFDWC proceedings are full of such musings.

In 1972, my first WIFDWC, Gus Loman addressed this same topic. Speaking of the future role of universities, he predicted "The basic function of universities ..... will be teaching and research aimed at training students in scientific investigation. The publish or perish syndrome will be removed from the career development of university teachers."

So much for predicting the future! With that lesson, I haven't even put a university representative on the panel. Given the proclivity of professors to talk, I'm sure they'll get their licks in one way or another.

I tried to share my concerns about the future of forest pathology at the recent APS meeting in Salt Lake City. I didn't get much sympathy from anyone, but several comments were particularly galling. A forest pathologist from Florida accused me of regional paranoia. It seems that things have never been better in the South, despite the recent Forest Service RIF. An assistant director at a Forest Service Experiment Station asserted that forest pathology has never been healthier. "Witness the constantly increasing number of papers in forest path sessions at the APS meetings." I didn't believe him at first, but a quick count seems to support his point.

APS Meeting	Total Papers	Forest Path Papers
1971 Philadelphia	218	18 (8%)
1974 Vancouver, B.C.	442	38 (9%)
1980 Minneapolis	552	68 (12%)
1982 Salt Lake City	653	71 (11%)

Is our western plight just a demographic quirk? Although most of the forests are in the West, most forest pathologists are in the East, and both are moving South. There is more than that. My friends have lost their jobs; in the South as well as the Northwest, and the process has been going on for a long time. My A.D. friend ensured my anger with his next, off-hand comment, "This is only a temporary slump in Forest Disease Research. Another 10 or 20 years and we'll be right back where we were!"

Great! I have asked our panel members to assess where we are today, and where we are going. They represent the various branches of Forest Pathology endeavor in the United States and Canada. Their remarks will trigger, I am sure, a wide open discussion amoung the audience.

THE FUTURE OF FOREST PATHOLOGY

David Adams

It is difficult at best to look into the future. I have found however, that looking into the future can be made somewhat easier if one first looks into the past. The past can act as a guide in pointing out possible directions the future may take. We as a people, and forest pathology as a science, are in a time of great and immediate change. We must understand what is happening about us so that we can make the necessary changes in our traditional thinking style.

Predicting the future of forest pathology seems to me to be particularly difficult, as it is tied into political whims as well as real needs. The following bit of verse seems to represent my feelings:

> Said a point being approached by a locus;
> I consider this sheer hocus-pocus.
> What good will it do me if it never gets to me?
> Will someone please tell it to focus?
>
> Conrad Patter Aiken
> A Seizure of Limericks, 1964

Nevertheless, I have turned to a recent book by Alvin Toffler, "The Third Wave" 1/ for inspiration and guidance. Toffler brings the future into focus through concentrating on the past and present happenings in this very complex world in which we live and work.

Let me make clear at the outset that I am not especially knowledgeable in this area, and therefore, don't necessarily follow Toffler's thinking. He has the advantage of many years of research behind him. He is also the author of "Future Shock", and "The Eco-Spasm Report". But, in any event his arguments are very interesting, and I believe carry a strong note of truth. You will have to form your own opinion, if you care to.

Toffler traces the development of man in society from earliest times to present day. Early man wandered about to find his food. Some ten thousand years ago, man first learned to till the soil to provide food for his needs. This was the agrarian revolution, or the first wave. This wave is still developing in some parts of the world today.

Some three hundred years ago an industrial revolution, the second wave, began and ultimately changed consumer first wave society into a producer society. Our Civil War in 1861 was based on this change taking place.

Now, Toffler sees a new change is taking place all over the world. This is his "Third Wave". Whereas, the first wave lasted ten thousand years, the second wave has been with us some three hundred years, the third wave will displace the second in only twenty to thirty years.

1/ Bantam Book, 1981

Toffler believes that third wave changes are reflected in our work, family life, life-styles and political acts. He claims that most of us, whether we realize it or not, are either of the second wave committed to maintaining that situation, or third wave, or some totally confused self-canceling combination of the two. It appears that as pathologists, many of us are not prepared to become part of the third wave; our philosophies are firmly rooted in second wave thinking. To be competitive in these times, one must compete for support not only on the scientific level, but also in the political arena.

For the sake of illustrating my theme, which is that forest pest control must be plainly visible to those we serve, both scientifically and politically, I will make a comparision. This comparison is for illustration purposes only. The underlying principles, not the overall comparison, is what I'm looking at.

In the broad science of "Forest Protection," notice I did not say "Forest Pest Protection," we find two major areas: forest fire control (and related activities) and forest pest control. The California Department of Forestry has responsibility for protection against fire and forest tree pests over some 8.5 million acres. Fire control, including fire prevention has a very large share of our budget, while pest protection receives much less.

Let's examine for a moment why fire control assumes the monetary importance that it does. Fire control people are very much under pressure to do "more with less," so it isn't a matter of funding to suit their wishes. Obviously, forest fire has a high visibility impact: life and property are threatened. Can we say that about forest pests? Occasionally we can, but not very dramatically. Insect damage becomes quite apparent during drought years; disease gets attention when the wind "blows over" a (root-rotted) tree onto someone's house. Really, where does disease enter public awareness? Would the public know a diseased tree if they saw one? Or even what disease is? If you as a forest pathologist were to be the recipient of a game of "Twenty Questions," do you think your questioneers could guess your occupation: I think not!! Upon learning of my profession, several people have thought that I studied dead bodies (human!!) in the forest. They were amazed, and rightly so, how anyone could earn a living that way.

It is easy to blame the "economy" for lack of forest pest monies with which to fund research and other positions. In good part this is true, but not completely so. Certainly, all governmental forest related activities have less money to work with than they had a few years ago. Fire control is no exception. However, as their funds have been reduced, their public awareness relationships have been intensified. What have we, as pathologists, been doing to enhance our public image and importance?

What should be obvious to even the most casual observer is that forest pest control (not to mention forest pathology) as a science has and continues to have a severe identity problem. We know who we are, but who else does? While annual board foot losses may be high, who sees it? Let alone who knows that there is a profession composed of scientists who know how to do something about reducing those losses?

While the topic of this panel is to discuss the "future of Forest Pathology," I really perceive it to mean the "Future of Research in Forest Pathology" for research is fundamental to all aspects of our profession. Without continued research forest pathology as a science would stagnate, and soon become outmoded.

There are very few, if any here who do not conduct research as part of their job. My assignment, however, is not one of research (though I do sneak a little in now and then under some guise) but really one of practicing extension work in forest pest control. From my point of view then, if you will permit me, I feel that the days of pathological research in the strict or historical sense of the word, are nearly over. The word I get from industry and my own CDF people is that research is fine (and we'll help), but don't let it interfere with our production goals. In other words, research, if you would call it that, must become a part of the production process and becomes not only cost efficient, but of practical application as well. The term "applied research" comes as close as any to describing the situation. For example, we are attempting to reduce incidence of Fusarium hypocotyl rot in true firs and sugar pine in a CDF nursery. Trials may go on, but production isn't allowed to be reduced because of our work. This type of study isn't new, but in these days when budgets are looked over very carefully, we can't let our work in pathology stand in the way of production.

From the standpoint of my work as a state forest pathologist, the form of my output must be service, and the product of output must lead to increased production for the grower. It is simple, he has less money to spend to produce more product. I can't interfere with that through my aid to him. We are in one of those "vicious cycles." Present intensive forest management requires more research, more research requires more money, etc. We will not get more money until we make ourselves known.

My own work in the Department of Forestry encompasses all areas of forest tree production from nursery to harvest, Christmas trees, hazardous tree evaluation, urban forestry, in addiiton to forest management plan preparation. In these times, the pathologist cannot hide under the shell of his/her knowledge of forest pathology, but he/she must be capable of considering pathology not as a discreet entity, but as a part of the whole ecosystem. It is, I believe, through the broadening of our concept of pathology that we will once again receive our due of respect and money for research and practice.

The future of forest pathology as I see it, depends upon the interest of some very diverse groups. On the one hand we have researchers who are the backbone of our science. At the other extreme we find a group of people who, for the most part, have no knowledge of what our life's work is all about. However, the latter group, being duly elected officials of our State and Federal Governments control our future through control of our funding. Obviously then, our future lies not in enlightening ourselves about what we do, but in bringing out to the public our message. It is through public concern and demand that our science will continue.

The IPM (integrated pest management, not increased political meddling) concept that many of us have been involved with, either directly or indirectly, is our most viable approach to public awareness. However, adoption of the IPM concept brings us into another cycle in which we need funding to fully develop our IPM programs in forest pest management.

Very few would argue against the benefits to be derived from a well managed IPM approach to disease and insect control. To develop and maintain a reasonable IPM program takes a totally committed effort by all of us, even if it means redirecting our own present efforts. Each pest situation needs to be looked at from a variety of control methods to evaluate which method(s) work best under the given circumstances. For some pest situations this has been done, for others very little has been done. Much research, compiling of data, and trial and error application is yet to be done. Public awareness needs cannot be over emphasized. Fully implementing an IPM program will be expensive, and that brings us back to why this panel was called into existence.

Forest pest control is in a transition period. Where we will go from here no one knows. But one thing is certain to me; the trend is towards direct results of applied control methods. I expect this to be the area of emphasis for many years to come. We all must be a part of this trend if we are to go on from here.

THE FUTURE OF FOREST PATHOLOGY

Vision of what the future holds for forest pathology is not any clearer for us than for anyone in this room. However, since we in Forest Pest Management (FPM) are routinely asked to predict future trends and consequences of insect and disease pests, not only months, but years in advance, we have grown accustomed to the uneasy feeling which accompanies these sometimes incompletely supported predictions. So, with this in mind, I have consulted the FPM crystal ball and, without fear or trepidation, will briefly review what FPM is and does; what personnel, budget, and program changes have taken place over the past 12 years or so; and then finally, offer a few observations about where we may be headed in the future.

The FPM staff groups nationwide are responsible for leadership, consultation, and support in forest pest management and pesticide-use management and coordination activities within the Forest Service. These responsibilities are unique. They not only include direct technical support of Forest Service and other Federal agency resource management activities but also the cooperative aspects of the Forest Pest Action Program and cooperative pest suppression projects with those States participating in this part of the Forest Service's State and Private Forestry program.

On National Forest and other Federal lands, FPM staffs provide technical support to resource managers by detecting and evaluating potentially damaging pest outbreaks to determine (1) nature and extent, (2) biological, economic, and environmental consequences, and (3) available prevention or suppression strategy alternatives.

Recommendations are made to resource managers, who ultimately select the final course of action from a variety of alternatives and who conduct the operational aspects of most pest prevention and suppression activities. FPM staffs also provide direction and coordination for pesticide use on Federal forests and rangelands as well as conduct special pest loss assessment projects and other pilot and demonstration projects to test and demonstrate promising technology.

The Cooperative Forest Pest Action Program is designed to assist States in financing the recurrent phases of forest insect and disease management such as prevention, detection, evaluation, and technical assistance. This program is authorized by the Cooperative Forestry Assistance Act of 1978, which recognizes both the need for close Federal-State-private cooperation in reducing forest insect- and disease-caused losses and for Federal

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Summary prepared by Robert C. Loomis, Staff Plant Pathologist, USDA Forest Service, Forest Pest Management, Washington, DC, for presentation at the 30th Annual Western International Forest Disease Work Conference, South Lake Tahoe, California, September 12-16, 1982.

coordination and financial assistance when widespread, damaging outbreaks exceed State and local capabilities to deal with them. The Cooperative Forest Pest Action Program helps insure that State forest pest management specialists are available to assist State and private resource managers in preventing losses and dealing with emergency outbreak situations. Technical and financial assistance is provided to States for these intermittent, damaging pest outbreaks through cooperative suppression projects which are also authorized by the Cooperative Forestry Assistance Act of 1978.

This briefly outlines much of what FPM is and does. The organization has changed over the last 12 or so years, and by examining these personnel, budget, and program changes, some insight to the future may be gained.

Personnel Changes

In 1970, FPM was called Forest Pest Control and had 94 staff professionals: 25 percent were pathologists, 63 percent were entomologists, and 12 percent of the professionals represented other disciplines. By latest count, we now have about 165 staff professionals--a 75 percent increase. The distribution by discipline remains very similar to what it was 12 years ago. That is, 28 percent pathologists, 57 percent entomologists, and 15 percent professionals from other disciplines. During this same period, the Forest Service as a whole increased by about 20 percent.

Viewed another way, pathologists have increased by about 95 percent, entomologists by 60 percent, and other disciplines by almost 120 percent. The "other" category has not only increased greatly on a percentage basis, but it has also undergone significant changes in its makeup. In 1970, this group was composed largely of personnel with general forestry backgrounds. In recent years, biometricians, economists, chemists, ecologists, statisticians, and computer progammers have made up the bulk of this group. We should also recognize that, as this "other" group increased, there was a 6 percent decrease in entomologists. It was at the expense of this group that some of the diversified staffing needs were met. This relatively modest drop in overall entomologists percentage masks that as many as one-fifth of this group spend all or a large percentage of their time on pesticide-use coordination and environmentally related administrative tasks. This further reduces the number of entomologists available for on-the-ground entomology. In contrast to this, the pathology group has not been tapped as much for these other duties.

Budget Changes

Since fiscal year 1972, the total annual forest pest management expenditures have increased from about $9 million to about $25 million in fiscal year 1981. This represents a 177 percent increase over the last 9 years. In real dollar value terms, however, the increase represents less than one-fourth of that or about a 42 percent increase. The relationship between our two major work categories--survey/program management activities and suppression--has fluctuated somewhat over the years but was almost identical in 1972 and 1981, with about 40 percent going to suppression and 60 percent going to survey and program activities. The Federal share of the Cooperative Forest Pest Action Program and pest suppression projects is

included in each category. The Cooperative Forest Pest Action Program expenditure has grown from about $435,000 with 29 States participating in 1971 to about $2 million with 44 States participating in 1981.

Program Changes

A significant change in pest management philosophy has evolved over the past decade. This change, represented in its simplest form, is a change from reliance on direct suppression to a recognition that the most effective results can be obtained by preventing and reducing damage of outbreaks by applying integrated pest management (IPM) principles in forest resource management activities.

During the late 1960s and early 1970s, forest pest management and fire management were viewed in much the same context. When the seeming inevitable insect and disease or fire outbreaks occurred, it was then time to gear up and put them out. Too often, important pest considerations were omitted from routine resource management decisions. Entomologists and pathologists were not yet part of the resource management team.

Encouragement to use prevention and practice IPM did not develop overnight. For one thing, not many opportunities existed for applying IPM principles where little or no silvicultural manipulation was done. For many years, we have been dealing with "unmanaged" stands. In recent years, more intensive forest management has been practiced on an ever-increasing number of forest acres and more opportunities are available for practicing IPM.

Another force which helped push this change along has been the passage of several Federal laws which required both Federal resource managers and managers of programs and projects supported by Federal funds to respond differently to the rising clamor of voices expressing divergent views of how the Nation's forest resources should be managed. For example, the National Environmental Policy Act of 1969 (NEPA), the Forest and Rangeland Renewable Resources Planning Act of 1974 (RPA) and the National Forest Management Act of 1976 (NFMA) require public participation in the decisionmaking process as well as thorough assessment of alternative management actions to facilitate balanced approaches to resource protection and development. The NFMA, in particular, requires the use of sound management practices which apply the principles of IPM to prevent excessive losses due to pests. In total, this and other legislation have more specifically outlined requirements of the resource management decisionmaking process and have required resource managers to better define and consider the biological, economic, and environmental aspects of major decisions. These two things--more intensive forest management and recent Federal legislation--have helped focus attention and provide the opportunity for more completely incorporating IPM principles in the resource management process.

The Future Outlook

In the short term, we expect a continuation of currently tight or reduced budgets. For example, the fiscal year 1983 budget battle continues between the Administration and Congress. The President's budget request represents a one-third cut from the fiscal year 1982 FPM appropriations. If passed,

this would significantly change the program as it is today. We understand some congressmen disagree with the proposed budget and may work to restore it at least to the current level. In any event, adjustments which may be made include reducing the Federal cost-share for suppression projects on State and private lands, or limiting the cooperative suppression we do undertake, to Federal lands and to State and private lands intermingled with Federal lands.

Other savings may have to be made by reducing FPM staffing levels and by further reducing our involvement in loss assessment, pilot, and demonstration projects. Available professional expertise will be stretched by turning much more of the routine pest-related surveys and evaluations over to forest management personnel. This can be accomplished in part by better incorporating pest considerations into the resource evaluations that are already being done.

The Cooperative Forest Pest Action Program remains a key link in fostering the Federal-State-private coordination needed to deal with widespread outbreaks. With this in mind and in spite of possible reduced budgets, we hope to update the present program and increase the Federal contribution to States to better reflect today's increased cost of doing business.

In the longer term, we will continue to emphasize the need to incorporate IPM principles into all forest management decisions. We know that many management decisions will be made with pest management considerations omitted or included only as an afterthought. This is partly because many of the forestry graduates of today receive little or no pest management education. It will be our job to somehow provide this basic education and supplemental training. Another reason why pest management is not adequately considered is because our message has not been convincing enough. Getting more consistent application of IPM principles will require both increased professional credibility with resource managers and well-supported recommendations. Professional credibility requires that we be knowledgeable both in our field of special expertise and in forest resource management. In addition, to be more persuasive, future recommendations will have to better recognize the interaction of pests as faced by resource managers and will have to predict with much more accuracy the biological, economic, and environmental consequences of alternative courses of action. We simply must continue to make progress in more accurately predicting outbreak consequences with and without control and in describing the benefits and costs of proposed pest-related activities.

In all likelihood, the professional makeup of our organization will continue to change. The "other" category will continue to grow as the need for special expertise grows. Also, an effort will be made to get pest management out closer to where the resource management decisions are made--at the Forest or District level. This could open up a number of new opportunities and will greatly affect how we interact with and provide technical assistance to Forests and Districts.

Finally, the currently tight budgets and proposed additional cuts in both Research and FPM budgets raise some serious questions. What organizations will pick up the slack? How will the quality and quantity of pest-related research be affected? With our reordered priorities, who will carry out pilot testing of promising research results?

Well, that is what FPM is and does, how we have changed over the past decade, and some questions to think about for the future. We must remember that even though change is unsettling for all of us, it is inevitable. It is through change that many of the improvements we take for granted have come about. Individuals and organizations which can foresee and understand future problems and take steps in advance to develop contingency strategies to deal positively with them will grow and prosper. We know that change will occur and that it is in our power to accept as well as to reject the opportunities the future holds. Which shall we choose?

The Future of Forest Disease Research in the Forest Service

Earl E. Nelson

Topics like this are frustrating because no matter how thoroughly one reviews the past or how hard one scrutinizes the present, the future remains clouded. Yet the future is closely tied to the past, and by taking some time to examine past events we can perhaps better understand why we are where we are today, and what to do to get where we want to be tomorrow.

Forest pathology research in the United States Department of Agriculture (USDA) grew out of early work at the Mississippi Valley Laboratory existing 1899-1907. Initially, much of the early work was on fruit tree diseases, but in later years emphasis turned to forest pathology. In 1907, the concern over chestnut blight was largely responsible for the formation of the Division of Forest Pathology within the USDA in Washington, D.C., and the transfer of forest pathologists to that Division.

Although there was a close working relationship between pathologists and the U.S. Forest Service (indeed, pathologists were stationed at Forest Service Experiment Stations in the late 1920's), the Division of Forest Pathology was not brought into the Forest Service until 1954. Forest pathology continued to grow in the Forest Service, along with economic prosperity and national emphasis on science, peaking about 1970 when more than 75 scientist-years (SY's) of research were accomplished. This compares to the past fiscal year when 61.5 SY's of research were accomplished.

At the turn of the century, forest pathology was a tiny organization, but early pathologists such as Von Schrenk, Hedgcock, and Spaulding had the determination and energy to make the profession grow. The introduction of white pine blister rust, as well as chestnut blight, substantially prodded that growth. Rapid spread of these highly visible diseases in such valuable timber species was alarming. Later, introduction of Dutch elm disease and phloem necrosis of elm again prompted increased research efforts (Hartly, 1980).

Forest Disease Research (FDR) can trace its roots, then, back some 80 years and in that time we can site a large number of successes: among them, the control of damping off and fusiform rust in nurseries; control of brown spot of longleaf pine, dwarf mistletoe of western conifers, annosus root rot of southern pines; and control of numerous foliage diseases of special importance to the ornamentals and Christmas tree industries.

But these examples are only a few of those diseases of major importance in our forests. Control of forest diseases is not always practical. Much of our research today looks not toward total control, but toward management of diseased forests to minimize losses. Successes here are more difficult to demonstrate and harder still to sell to politicians, high level administrators, and even to forest managers who are often looking for easy-to-apply cures. We have a lot of work left to do both in finding solutions to disease problems and doing our best to see that these solutions are implemented.

There is presently in FDR a growing uncertainty of the future. I have asked FDR project leaders and Washington Office Staff for their thoughts on the health of our organization and their insight on what we can do to strengthen FDR in the future. I would like to share some of their ideas with you.

Recent reductions in funding are certainly not unique to FDR. They are signs of economic trends in general and of current philosophy of reducing spending in government. Proposed reductions for Forest Insect and Disease Research (FIDR) for 1983 are about 11 percent below 1982. I do not know how much of the total reduction will fall upon FDR but projected reductions are substantial. Reductions in FIDR will be painful because so much of FIDR's funding is tied up in salary. Wicker (1980) stated that FIDR had the lowest average operating funds/SY at $45,000. The average for all of Forest Service Research (FSR) was $69,000; a difference of $24,000. Schipper (personal communication) found for the period 1969-1982 that FDR was funded at levels below FSR in general on a per SY basis. In Fiscal Year 1982, this difference amounted to over $21,000.

There are several reasons why FDR is hurting for lack of research funds. With few exceptions, diseases are not highly visible and, therefore, do not attract wide attention. Because of their insidious nature, not only are losses unapparent to most people, but in many cases we, as forest pathologists, have only hazy ideas of how much loss diseases cause. This lack of disease impact data has been a stumbling block to increased funding for as long as I can remember. If we cannot demonstrate significant disease impacts, we cannot justify working on disease problems at all. Cost/benefit ratios certainly cannot be determined using costs alone.

The remedy for this problem would seem to be to collect necessary data to make the needed realistic estimates of disease loss. In many cases, however, techniques for doing this are too crude to make it an attractive proposition for Forest Pest Management (FPM) or other agencies responsible for measuring impact. The beginning of a solution to this problem may lie in the establishment of a multidisciplinary project to develop technology to better quantify insect and disease impacts. This is presently being seriously considered by the Washington Office of FIDR. But developing better techniques could take years and the need for impact data is now. Until new technology is developed to provide a means of acquiring data supporting the need for forest disease research, some of the more workable techniques now at our disposal should be used to gather broad-based impact data for at least some of the more important diseases.

All of the woes of FDR cannot be blamed on world economics, lack of disease visibility, or lack of good impact data. We have been guilty of poor salesmanship in getting into practice some of the gains we have made toward solution of important disease problems. We need to sell ourselves to users of research information, to the public, and to politicians. We need their collective support if we are going to be funded to continue producing salable knowledge.

Is FDR a productive organization? I believe it has been and that it will be in spite of some of the problems that face us today. Budget reductions and personnel ceilings (problems in themselves) have led, first, to an undesirable age stratification among FDR scientists and, more recently, to a sharp reduction in numbers of research scientists. It is pretty obvious that with fewer dollars and fewer people we cannot accomplish as much as an organization as we once did. But, perhaps of even more importance to our long term productivity is the absence of new staff--the fresh ideas, concepts, and stimulation provided by young scientists. We could all profit by having a few bright, ambitious, young people "nipping at our mental heels." A better balance of age in our organization would also provide continuity in tending long term research projects, so often a major part of FDR programs.

Another problem we often face is the paradox between primary research responsibility and reward for accomplishment. Problem solving research requires an analysis of the problem, separating its components, and solving all subproblems to arrive at the final solution before new problems are tackled. Some of the subproblems may require a substantial amount of time and resources. Problem solving research is encouraged by research administrators and only makes good sense if, indeed, we exist to help forest managers solve their disease problems. Reward for accomplishment, however, is too often based on numbers of publications (too often aimed at ourselves) when we are evaluated by our supervisors or, for that matter, by our peers. Perhaps we have yielded too often to the temptations of reward and have not done our best to truly solve management problems.

There is strong feeling among many forest pathologists that the future of FDR lies with how well we approach and solve disease problems. There is a true need to work closely with forest managers, to understand their problems, and to attack those problems in a way that leads most directly to their overall solution. In so doing, we will often need to work with scientists of other disciplines, and where expertise is lacking within FSR to cooperate with universities or other research organizations. Perhaps today's emphasis on integrated pest management will make this easier to accomplish.

There is no easy solution to the problems we face today. The way out of our predicament is the reverse of the way in. Some of our problems are beyond our power to solve, but there is much we can do toward solving the others. If the world economic situation does not improve, we must do the job with fewer resources. If our numbers grow smaller, we must further focus our efforts to make meaningful contributions in some problem areas. If we lack visible evidence of disease-caused forest devastation to impress congressmen, we must press for the statistics to do so. If we cannot hire young scientists, we must seek out those desirable interactions with them, perhaps with increased efforts to support graduate study. If we feel unjustly rewarded for pursuing research in a problem solving mode, we must convince those who judge us of the system's unfairness.

Forest disease research may suffer additional setbacks in future years before we are again firmly back on our feet. We cannot predict the future. But, as long as we believe in ourselves, the future is in good hands.

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Future of Forest Pathology in Canada

by

R.D. WHITNEY, R.S. HUNT and J.A. MUNRO

If Paul's Motor Inn in Victoria had exploded between April 13 and 16 this year, all but two research forest pathologists in the Canadian Forestry Service could have been wiped out. Dr. C.E. Dorworth was on sabbatical in Italy and Dr. J.T. Basham was in England because of family illness. Otherwise they too would have been attending the Forest Pathology Workshop to which all CFS pathologists and almost all from universities and provincial governments were in attendance. This Workshop was convened to review, discuss and revise a working paper on Canadian Forest Pathology--why it is needed, what is has contributed and what it can contribute to better forest management in Canada in the foreseeable future.

A Review of Forest Pathology research in the CFS has been undertaken by a committee of three appointed by the Director-General of Research and Technical Services, in the CFS of Environment Canada. The terms of reference indicated that the committee would gather information, chiefly through correspondence and from reports, on tree disease problems and research opportunities and priorities with a view to ensuring that forest pathology research was not neglected in future CFS Research Programs.

The committee spent 4 months gathering information and preparing a 119 page report which was the background paper for a 4-day Workshop which refined the report, proposed research needs and finalized recommendations. The nuts and bolts of it were that the committee of three took information and comments from the workshop and incorporated them into a revised report which was only submitted to the Director General September 10. This report with its recommendations is to be considered by the Committee of Establishment Directors at their meeting September 21.

Understandably, we are not at liberty to discuss matters of a controversial nature contained in the report or the recommendations prior to them having been seen by those authorizing the review. Information on tree diseases in Canada contained in the report is mostly published and can be freely used. Much of what we have to say are our impressions of forest pathology in Canada gained from being involved with the forest pathology review.

Forest pathology in Canada grew up around mycological studies and decay studies. Pathologists worked on individual fungi and the handful of diseases that were known to be important such as white pine blister rust, Dutch elm disease, birch dieback, pole blight and dwarf mistletoes. Decay studies resulted in better net volume estimates in standing timber.

Impact of Tree Diseases

In Canada the forest products industry contributed $22 billion to the economy in 1980 accounting for one dollar in every seven of manufacturing shipments (3). Forest products resulted in a positive balance of trade of $12 billion which was more than any other sector including agriculture and

mining. This high productivity has been maintained for some decades and probably will continue for a few more years.

Actually you might say there has been little or no effect of tree diseases on Canada's economy or standard of living of its citizens. The cost of wood and of Christmas trees may be somewhat higher as a result of measures being applied for control of nursery diseases and Dutch elm disease may have indirectly reduced some property values and necessitated the use of some other species for furniture stock. The great reduction in the annual white pine cut from the early part of this century to the present is due less to white pine blister rust than to lack of reforestation. Decay, which reportedly destroys some 25 mm m of wood per year in standing timber in Canada, does not prevent our mills from running at full capacity. Other factors might and do. Armillaria mellea kills about 2% of stems per year in conifer plantations in Ontario but if final stocking is affected at 60 or 70 years of age in spruce or pine stands this is not known or heeded at present.

In a recent estimate, total annual losses or consumption by diseases exceed 65 mm m^3 of timber in all of Canada (2). Diseases and insects have been getting a large share of the forest estate for a long time and they will continue to do so as long as people are satisfied with the share they are getting. On a Canada-wide basis diseases account for more than 1/3 as much timber as is harvested annually. It should be remembered that for the entire country only about 75% of the economically accessible allowable cut is harvested (3). However, the excess of growth over cut is all concentrated in only 4 provinces and it is only marginally economic. Regional shortages are already being experienced as was indicated in a recent federal forestry sector strategy paper. Tight regional wood supply problems could possibly be alleviated by reducing current losses to diseases, insects and fire. Reduction in disease losses of about 30% would make available an additional 20 mm m^3 per year and this would be most advantageous if concentrated regionally where the wood shortages existed.

Two aspects of the above statements are pertinent to disease considerations. The first is that the consumption of trees by diseases will eventually be felt by wood-working industries and at that time controls or some means of reducing the effects of diseases will be wanted in a hurry. This will likely occur when the old growth timber is used up. The second is, what are the volumes being consumed on a specific watershed, leased area, stand or hectare of land? This was the most sought after information by forest industrialists and provincial forest managers in answer to solicitations concerning important diseases in their jurisdiction. How much of a disease problem do I have? What effect will diseases have on future stocking, yield, stand composition and seed production in my stands--10, 20 or 50 years hence? Most forest managers are unaware of the losses diseases are causing in the forest, and unless they are catastrophic many diseases may not be noticed at all. We as pathologists aren't able to give all the answers either.

This leads to a topic considered to be of utmost importance at the Victoria Workshop--that of Loss Assessment or Damage Appraisal. Horsfall and Cowling in the first Vol. of their book (5) p. 8, state the most urgent need in forest pathology today (1977) is improved understanding of the magnitude of losses. It is no easy matter. Damage by diseases consists of a variety of effects--mortality, growth reduction, decay, deformation, predisposition to windfall

or other pests including other diseases and pollution. In long-lived plants like trees all of these can be present until mortality occurs. Measurement of these damage factors are difficult enough but attributing economic loss to them is even more difficult. Losses from nursery diseases for example, can be determined with a high degree of accuracy because damage can be easily tallied, cost of new seed and seeding operations are definable, and most of land, and other material and labor can be found. The only difficult problem with estimating losses from nursery diseases is costs resulting from adjusting planting programs if the seedlings are not available. But, for other diseases not only are damage factors such as growth reduction, more difficult to measure and isolate, but ultimate economic losses to productivity such as from liquidating a stand prior to maturity for a lower value product, must be taken into account.

Research managers are reluctant to assign scarcer and scarcer research resources to study of a disease problem until the economic importance of the disease has been shown. This often cannot be done due to the above difficulties. Furthermore, researchers have been reluctant to work on damage or loss aspects of diseases when both their training and inclination lean more to solving the problem. In the absence of good damage and loss appraisals, justification and hence support for disease research have declined steadily since the disposition of research resources has been called into question.

Lack of support for disease research was further aggravated by inability of researchers to develop directly applicable controls. Where direct controls could not be found, silvicultural or indirect methods of reducing losses were resorted to and developed for many diseases. These methods included such procedures as altering stand composition, reducing logging injuries, reducing rotation ages, eliminating cull or diseased trees or parts of trees, or avoiding silvicultural practices that increase disease susceptibility. Since levels of forest management in Canada were such that most of these silvicultural techniques were not used, such disease control was held in abeyance until forest management practice intensified.

Not all the disease control recommendations were impractical, however.

Forecast Forest Management Problems and Disease Implications

Forest Management will probably become a reality in Canada in the 1980's with an increase in intensity beyond that. During the last five years there has been a very noticeable increase of public and political awareness of the importance of forests and of their problems. A prominent silviculture researcher in Canada, W. M. Stiell, stated in 1976 "The intensification of forest management is inevitably accompanied by a sharper awareness of forest enemies..." Their impact often looms most prominently in plantations, where it is readily perceived and where the costs of stand establishment are best appreciated.

In Canada only 200,000 ha/yr. are reforested while the annual area of cutover is 800,000 ha. An additional 200-300,000 regenerate naturally (3). There are signs already that reforestation will increase dramatically to try and bridge this gap and reforest the backlog of unforested cutover land. Seed orchards, seed and seedling storage, and nursery production, each with known disease problems are being increased. The application of selected

mycorrhizae to stimulate seedling growth and confer other desirable characteristics to trees may come into general practice. This will necessitate the development of a mycorrhiza technology.

With liquidation of old-growth forests and bringing on stream of new forests, rotation ages will decrease and stem decay will be very much reduced. In the immediate future trees formerly considered as weed species such as aspen, red alder and balsam fir, will be utilized, necessitating more intensive study of their pests including diseases. Intensive management has begun with site preparation, pre-commercial thinning and fertilization. In addition, tree improvement and selection, pruning and commercial thinning will start.

A pathological benefit accompanying thinnings or tree improvement cuts is that an opportunity is provided to remove diseased trees or those of low vigour that are prone to disease. At the same time, however, stand improvement with machinery inevitably results in wounds to residual trees which can be ideal entrance courts for disease fungi and log degrade through deformation. Treatments or refinements of methods will have to be developed where these procedures come into use. Dead root material may also provide unwanted inoculum of some parasites.

Scarification procedures conducted to prepare sites for conifer regeneration frequently do not kill, but only badly damage poplar, birch, alder, etc., that continue to form crop trees. Unless these damaging procedures are altered, the diseases resulting will have to be dealt with.

The application of fertilizers, while increasing tree growth, can result in more disease, and faster-growing black spruce on upland sites have been found to have much more root and butt rot than slower-growing ones on the same sites. These facts should be taken into account before extensive fertilizing is conducted.

As mentioned earlier, information on the losses caused by diseases becomes increasingly important as silviculture intensifies. The effects of formerly weak parasites may be vastly different in younger stands grown under different conditions. More intensive disease surveys and better damage information will be required.

In the future there will have to be more pathology input in forest planning for seed production, raising of seedlings, selection of planting sites, tending of stands and harvest and utilization of trees. Lack of pathology input at the planning stage has resulted in silviculture failures in nurseries and planting programs. Before investing scarce resources in large-scale planting programs, forest managers must be shown how to assess pathological consequences of various silviculture options.

Present Pathology Program in CFS

Present research is largely conceived by regional establishments in consultation with provincial resource managers. From 1968 to 1979, the Canadian Forestry Service research personpower was reduced from 2181 to 1063, a reduction of 51 percent. At the same time professional pathologists were reduced from 54 to 25, a 54% reduction. Of these 25, 8 are in survey work, leaving 17 person-years in research.

The Pacific and Great Lakes Regions are best represented with forest pathology staff, with the other four Regions below the critical mass or effective working level (4, 9).

Good biological information has been obtained on many of our important disease problems such as decay, stem rusts, root rots, fume damage, Dutch elm disease, stem rusts, mistletoes, nursery diseases, cankers, pole blight, birch, maple and ash dieback, and mycorrhizae. Research results have been incorporated into forest management plans where appropriate, but some recommendations were impractical because of the low level of forest management as indicated above. At present, root diseases employ the highest number of professionals 4.3 (26%) followed by studies on Scleroderris canker (17%), DED (15%), and stem decay and deterioration (10%). Rust studies and technology transfer employ 6% and 5% of the person-years and other disease problems employ 1-4%.

The number one disease problem for both provincial and industrial foresters, root diseases, is in fact receiving the most research resources (26%) by the CFS. Two of the other three research areas receiving large proportions of the research effort, Scleroderris Canker and Technology Transfer, also rated high or moderately high as problems for provincial or industrial forest managers. Dutch elm disease research, receiving 15% of CFS pathology research resources, was rated as moderately important by provincial forest managers and as unimportant by industrial foresters. However, DED is a major concern of municipal authorities who were not solicited in the survey.

Strengths in the CFS pathology research program are the fund of basic information on many diseases built up over the years and in expertise in the various regions. A strong diagnostic capability has been developed and perspectives have been established on a large number of relatively less important disease agents. Studies of several unsolved diseases such as birch dieback and pole blight, while not resulting in specific controls, have contributed substantially to the understanding of the ecology of the trees concerned.

Weaknesses in the program to-date have included inappropriate or impractical recommendations for disease control and inadequate explanation or transfer of control information to forest managers. Lack of a sound relationship with other disciplines at conceptual and planning stages of research work on diseases has been another weakness. There has been a serious lack of reliable estimates of damage or losses from specific diseases. This has not only frustrated control procedures, but has failed to provide the necessary justification for badly needed research on many diseases.

Future of Forest Pathology in Canada

I cannot say what the future will bring for forest pathology in Canada. I can indicate what I hope will happen and what I hope will not happen.

First, what I hope will not happen is a continued retrenchment and cutting back in forest pathology research in Canada. I think I was expected to bring a ray of sunshine to this panel so I won't dwell on this. But the sun has definitely gone behind a cloud because the Dutch Elm Disease research project at the Great Lakes Forest Research Centre was terminated in August this year!

As silviculture comes of age in Canada anything interferring with tree growth will also become important and I hope the development of disease management will follow. With the rising value of forest products there will be a general improvement in the economic climate for investment in good forest management in general, and better pest management in particular. A declining land base will force the forest manager to maximize his yield of usable product by whatever means available to him, including pest control. Forest managers are becoming aware of the other managers of the forest--insects, diseases and environmental conditions. These other managers have always been there. If man, the intruder, wants a larger slice of the pie, he must recognize this and learn to better compete with those having nature on their side.

Timber and forest products in British Columbia, are of the utmost importance, and the B.C. Ministry of Forests, in recognition of an expected serious shortfall in forest yield in some districts, is developing a vigorous pest (especially disease) management program. A Forest Pest Management Section has been established (presently 52 person-years) by the BCMF which is dependent mainly on the Pacific Forest Research Centre for pest control information. Currently emphasis is being placed on reduction of root rot and mistletoes, which combined were estimated to result in losses of 6-5 billion m^3 of timber in British Columbia in 1978 (1). Additional information on these pests will be required for implementing a disease management program that is effective and economical.

Two aspects of Canadian Forest Pathology must be addressed if an analysis of the immediate future is to be made. These are: 1. Convincing management that diseases are important and should be studied. 2. Combating the diseases--new approaches.

Earlier in this report the inadequacies of damage appraisal and disease loss estimates were pointed out. Managers, not only of the forest, but of Research also, must be shown the importance of diseases to their operation. National average damage or loss estimates are of little use when trying to estimate yields on a specific property. Site characteristics and other environmental factors affecting tree quality are not described in national or provincial loss estimates, and sampling on the specific property by stand types is required to get accurate estimates of current disease or insect losses. Making forest managers aware of tree disease in their area and what they should be looking for is a current technology transfer challenge. This challenge is beginning to be met by such mechanisms as root rot workshops for forest managers in British Columbia (8). Being aware of diseases is not the whole answer of course, but knowing their presence and potential for damage can help considerably in management planning. Disease awareness among forest managers is where the impetus for research must originate, but this awareness must be fostered by pathologists. Most forest pathologists that I know would rather work on a pesky disease problem than try to convince a busy management forester of the importance of diseases. But it is largely up to us as the only body of people knowledgeable of tree diseases to show foresters and research managers what diseases are doing.

Another attack on this job of technology transfer could be made through our educational institutions. Lack of knowledge about tree diseases among forest managers is partly due to forest pathology being taught chiefly in isolation and in an atmosphere of research rather than as an integral part of

forest management. Forest pathology and entomology and fire control and use, should always be taught in the context of forest management and tree physiology. Curriculae on forest management should recognize that diseases are a part of nature and we must assume they will be there unless preventive steps are taken at appropriate times in management planning and during the life of a stand.

When the forester does decide that disease control or minimization of diseases is a worthwhile silvicultural option, pathology expertise and control techniques often must be incorporated early in the life of a stand as they are designed to prevent diseases from developing. Preventive disease management can only be practiced if knowledge of tree diseases is utilized during forest management planning. If the lush looking seedlings a forester gets from the nursery have hockey-stick roots from nursery transplanting, or the roots are dry and moldy from improper storage, they must be rejected. Otherwise problems of root rot, premature windfall, and disease associated with low vigor trees will develop later on. If the area being planted is sandy and rapidly drained he must insist on a species such as jack pine that will survive and grow there. Spruce will most likely survive poorly and those that do survive will become chlorotic and many will contract root rot and die. If only spruce is available, the planting must be postponed until the desired species is available. Perhaps the forester should insist on mycorrhizal seedings--up to a certain mycorrhizal standard, or even specify the mycorrhizal species. Our technology on mycorrhizae is certainly not developed to that level in Canada, but regeneration problems are paramount in parts of the country, and maybe mycorrhizae could help. Here we need more knowledge.

Of course diseases develop later on in the life of a stand as well, and unexpected diseases occur. Many of these are with us now--white pine blister rust, Phellinus root rot, birch dieback, etc.

Innovative ideas and procedures will have to be developed for disease reduction and control. Genes controlling virulence in pathogens and resistance in host trees should be investigated. Use of the fused cell technique for hybrid production in biotechnology, if applicable to forest trees, could ultimately lead to plants with greater disease resistance. Direct control of forest pathogens involving the use of virus-like agents as in hypovirulence in the chestnut blight fungus, should be tried on more diseases. Biotechnology, still in its infancy, requires an in-depth subcellular scientific base. This usually means amalgamation of expertise from several institutions, but the ultimate payoff in tree disease control could be great.

The incorporation of disease information into stand models and simulation techniques can help to ensure use of tree disease technology, as well as point out the gaps in information that exist. The use of these techniques could be much more widely applied in Canada.

As in human medicine, emphasis should be placed primarily on the preventive rather than the corrective approach to disease management. Understanding the ecosystem in which the trees grow is essential if trees are to be grown under conditions where prevention can be practiced. Biological control is an ideal method of control where environmental conditions are manipulated so a third organism or entity works against the pathogen or disease agent, but not the host. Biological control of plant diseases has not been a roaring

success in North America, but one of the two commercially available biocontrol agents for plant diseases in the United States is for a tree disease--Annosus root rot. Other examples, as with Trichoderma viride parasitizing Armillaria mellea and mycorrhizae protecting against Phytophthora cinnamomi, seem to indicate promise in this field. But unless you have a lot of luck, biological control entails a detailed knowledge of large numbers of interacting factors and is a tremendously large undertaking.

We hope that a new era is dawning where diseases will be studied in concert with insect attack and adverse environmental factors. Too often in the past one of the three above aspects of a problem only has been addressed. There are some notable exceptions. Long-lived perennial plants (trees) can be expected to have combinations of problems acting together in a disease syndrome. In volume 5 of their treatise, Horsfall and Cowling (6) state "rarely is disease caused by a single agent; cause is complex, and its study is just as complex...." Probably research on most diseases would be more successful if expertise from several other disciplines were also brought to bear on the problem. Examples of where this had happened in Canada are the blue stain bark beetle complex of lodgepole pine and white spruce, Dutch elm disease and nursery diseases.

Tree stress is probably a basic cause of a tree succumbing to many diseases. Shigo (7) recently pointed out that if sufficient energy flows are present in a tree, that many infections that occur are walled off and the tree survives. This cannot happen if energy is low. The part that predisposing stress factors play in pathogen infection and disease development is complex but is basic to understanding tree diseases. Even if all the intricate stress relationships are not clarified, at least what the stress factors are should be defined.

During the recent review exercise pathologists in each of the six regions of Canada were asked to priorize important diseases in their region. Armillaria root rot was in the top 6 important diseases in all regions. Stem decay and deterioration studies and white pine blister rust were included in the first 10 important diseases in all regions but one. Scleroderris canker was rated in the top 3 diseases in all but the two western regions. Damage appraisal or impact studies were indicated as a requirement for most diseases.

To sum up let me say: 1. It is not easy to build a case for tree disease research when forest management is at a low level or is non-existant. 2. In Canada accurate loss statements from tree diseases by land jurisdiction are probably the most urgent requirement as an impetus to disease research. 3. Aside from nursery diseases, controls for diseases of commercial trees have been unspectacular. The greatest pathology contributions have been in inventory improvement through estimates of cull. Our two most important diseases (by volume)--stem decay and mistletoes, are being greatly reduced through reduction of rotation age and sanitation--not through any control breakthrough. 4. A breakthrough in controlling important diseases such as pine stem rusts, root rots or mistletoes in young stands is badly needed. 5. Disease prevention through application of sound knowledge about diseases to forest management is ultimately the best control. Let's hope that research on tree diseases does not disappear because of unspectacular cures. 6. Better knowledge about tree disease among practicing foresters so they can appreciate

the effect of diseases on forest management, should be an aim of our forestry education institutions.

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Panel: Who Are The Players In IPM, And Is It Something That New ?

Fields W. Cobb - Moderator

Much has been said about IPM during the past several years, and two years ago we had a lively discussion on the subject at Pingree Park, CO, which was very ably led by Dick Parmeter. Hence, we do not wish to belabor the subject. Rather, we'd like to try to answer a couple of questions that we think are important. Most likely, you will find that there is not complete unanimity even among the panel.

The answer to the first question, "Is it really new?" depends upon one's view or definition of IPM. The terminology is new; entomologists came up with it to replace another of their terms, "integrated control." IPM is an expanded concept of integrated control, and there are many entomologists who claim both concepts as theirs. As for the IPM concept, I contend that plant pathologists have been practicing much of that ever since our triad concept (host-pathogen-environment) replaced the old germ concept several decades ago. This is especially true with respect to forest pathology, particularly because we could not justify the costs of multiple applications of fungicides over a 50-100 year rotation.

However, that is not to say that there is nothing new within IPM. There certainly is. For example, IPM has a very strong economic component, one that often necessitates the inclusion of economists on an IPM implementation team. In turn, the economists are going to need much better quantitative data from us on the effects of disease alone and in combination with other pests.

IPM also demands a strong input concerning the requirements, physiology and growth of the host plants. If we are truly interested in integrating the management of pests, it means that we and the entomologists are going to have to communicate with each other more effectively. And, in spite of all the negative reactions, we must learn to make computers and their programmers work for us. A fully-integrated pest management plan should include inputs from many disciplines. To integrate those inputs into predictive models, we must use the computer and the mathematical models that are being developed. This again brings us to the need for good, quantitative data. The data must come from surveys and other studies that are well-conceived to give the types of data (input) needed. Used correctly, the computers and models can assist us greatly in identifying gaps in our knowledge that need filling before we can develop sound plans.

Is IPM new? Frankly, my opinion is, "no." Most of us have been practicing it in a somewhat crude form all along. However, we now have a new generation of tools and a broader recognition of how we should be working together for the common goal--optimization of forest yields in the long term.

Which brings me to the second question, "Who are the players?" To me, this is a very important concern. Often, we've heard people speak as though IPM exclusively, or at least primarily, involved pathologists and entomologists. Others might add weed control specialists. Sometimes, we might even include the silviculturist, which is certainly a move in the right direction. However, as in our discussion of "who is our audience," we have often excluded the "bottom line." The bottom line is, of course, the forester--the man or (more and more) the woman who must recognize the value of our knowledge and put it to good use. If the forester does not see the value of our input, it won't matter how much the public on the streets of San Francisco may like the pictures in our publications. If we do not include the forester in our IPM team from the very beginning, we are making

a big gamble that our efforts will be overlooked once again in the management of forest resources. Maybe the term "IPM" should now be discarded in favor of the old "forest resource management." That is where our ultimate goals are and that is where we have been for decades, except for short lapses when we may have forgotten those goals.

IFM, NOT IPM

Oscar J. Dooling[1/]

What we're really talking about is integrated forest management (IFM), not integrated pest management (IPM).

The manager is constantly bombarded from all sides with advice; pathology is only a small part of it. If you can prove your advice is important, the manager will listen. But remember--even though the manager understands the problem and would like to follow your advice, there may be other factors that you aren't aware of that take precedence over anything you say: esthetics, watershed, soils, or wildlife. Politics can be either external (public) or internal. Policy is also important--what you suggest may be 180 degrees out of phase with organizational policy.

Your part of IFM is to help the manager understand the tradeoffs by playing the pathology game of "If you do this, then you can expect..."; but be ready to play the management game of "But I can't because...."

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WHO ARE THE PLAYERS IN IPM--IS IT SOMETHING NEW?

C. S. Koehler, Entomologist
Cooperative Extension
University of California, Berkeley

From the perspective of an entomologist more acquainted with urban forestry and agriculture than commercial forestry, there are three points I wish to make:

(1) On the question of whether IPM is something new, we must go back, in the West, to about 1950 when the term supervised control was coined. This described an agricultural system involving grower employment of an entomologist to sample his acreage for pest and beneficial insects and to advise on the need for treatment. In the early 1960's a new term, integrated control, was coined. This involved a more deliberate attempt, on the part of the crop protection entomologist, to integrate various components of insect control practices--usually chemical and biological control. In the early 1970's a new term emerged, integrated pest management (IPM), which described an integration of all the crop protection disciplines into a unified and compatible protection system. IPM was an image-building term, one designed to imply that we had left the dark ages of pest control and entered a new and enlightened era.

Each of the first-named systems lasted about 10-12 years each. On that basis, we may be approaching the end of the natural life of IPM. It is likely to be replaced by a term emphasizing integration of all the essential components of an entire cropping system. Already in agriculture there are signs that such a new philosophy is emerging.

The leadership of each of these successor systems has been provided by entomologists, and each system has been inflicted upon counterparts in the other plant protection disciplines. Where are the spokespersons who will assure that plant (forest) pathology will get its proper billing in whatever succeeds IPM?

(2) The future of traditional forest IPM is cause for concern. You are attempting to provide protection of a crop with a 40-80 year harvest cycle while dealing with the vagaries and realities of 2-5 year funding cycles. Superimposed on this climate are the uncertainties of housing starts. Finally, U.S. Congressional reapportionment has stripped the vote from the rural sector and given it to the urban sector--to folks who see our forests more as recreational lands than as timber production areas.

Consider exploiting the phenomenon called urban forestry. In today's climate it appears to be one of the few growth areas for forestry and forest protection, at least in the short term. Much of the research reported at this Workship could have been conducted under the umbrella of urban forestry. So long as the public sector is likely to remain a major sponsor of forest IPM, consider taking advantage of what urban forestry has to offer.

(3) In the real world of urban forestry, the probable inevitable establishment of insects such as the gypsy moth are likely to afford new challenges to forest IPM. In 1982 gypsy moth was discovered in 14 California counties, and in about 12 counties collectively in Oregon and Washington. From the Eastern

experience it is well known that several consecutive years of defoliation of broadleaved trees, and a single year of defoliation of conifers, results in stressed trees and in increases in pathogen incidence sufficient to kill such trees. If or when the gypsy moth succeeds in becoming established in the West, and if the insect performs here as it does in the East, we will confront a problem quite unlike anything we've had to deal with in the Western urban forest.

IPM -- THE PLAYERS, AND NOVELTY -- AN INDUSTRIAL PATHOLOGIST'S VIEW

Jerry Koenigs
International Paper Co.

I approach this topic with both enthusiasm and trepidation. Enthusiasm because, although I was hired by International Paper Company to develop a plan of basic research in the area of pest protection, I soon realized that a good deal of pest research had not been applied. Consequently, I tailored a series of management recommendations for major southern pests to satisfy IP's specific objectives and constraints. Out of this activity an IPM program was born and adopted by our Land & Timber Business Division. Besides some research projects that I conduct, my efforts still focus heavily on aspects of pest management particularly in the areas of hazard rating and computerized mapping for fusiform rust and annosus root-rot. I feel particularly fortunate in being able to wear the two hats of researcher and pest manager because of the insight this provides as to the intimate interdependency between research and pest management. Although I researched at the Intermountain Forest & Range Experiment Station in 1961-1965, my apprehension initially arose from my feeling of ignorance of the current status of forestry management, forest pest research, and pest management practice in the West. Also some of my comments may appear to be arbitrary and critical of pest research and researchers and to overlook much of the good research, but I offer them in good faith and with some awareness of the dynamic research underway. Please realize that I feel equally vulnerable to such criticism because of some of my past attitudes while I was purely a researcher.

Who are the players in IPM? Most obvious is the pest manager. He is the technologist who applies knowledge about the pest in order to reduce losses and control costs. Less obvious is the researcher who supplies the basic biological and ecological knowledge to the pest manager. Without him there would be no technology -- technology being simply applied science. Least obvious, especially to some researchers, is the forest manager -- the player with the pest problem. I consider him a if not the principal member of the IPM team. He is a forest pest researcher's and pest manager's "reason for being". Without him there is no need for us. Furthermore, if we can't convince him to use our research or accept our recommendations, what have we accomplished? Additional members of a pest management team may at times include economists, statisticians, growth and yield specialists, silviculturists, and eventually geneticists, meteorologists and soil scientists.

As to the second part of the panel topic -- is IPM new? If your view of IPM is that it is a loose system for managing pests by coordinating control options -- usually silvicultural measures -- into a coherent plan, then, no, IPM isn't all that new. You may also believe that view is sufficiently comprehensive or dismiss IPM as a vague program based on glittering generalities. For the researcher, pest manager or forest manager with these beliefs, I suggest that some changes are necessary in your thinking if forest productivity is to be increased through pest management.

What are some of these changes? First, lets change the name of the game -- from IPM to IFPM. You guessed it, the "F" is for "Forest". This is more than an academic issue or a psychological ploy associated with buzzwords. Nor was it meant to differentiate the pest management of forests vs. agronomic crops. It was done to introduce a concept of scale, to change our focus from tree pathology to forest pathology. It's the difference between autecology and synecology -- the difference between studying the effect of environment and man on the pest in single trees or perhaps a part of a stand to one of populations of pests in populations of trees within stands, and of stands within forests or over a region. This perspective is required for acquiring pest information on a scale that permits estimating impact and development of management strategies at the regional level as well as at the individual stand level. Basically, it describes the epidemiological approach to forest pest research and pest management.

Epidemiological studies involve four general activities: (1) developing disease progress curves along with a knowledge of the biological and ecological factors affecting rates of change, (2) developing incidence - impact curves, (3) rating, monitoring, mapping and forecasting hazard and (4) evaluating the effectiveness of various pest control measures under varing environmental conditions and levels of hazard. The flow of epidemiological knowledge from research to pest management takes the form of hazard evaluation and control effectiveness. I do not undervalue the importance of basic research on basic biology and ecology of the pathogen, host and the disease, but I do stress that this information be acquired with an understanding of the epidemiological framework into which it should eventually be fitted if it is to be ultimately worthwhile to the pest manager and forest manager.

The "Forest" concept in IFPM requires determining the probability distribution of pest impact or hazard zones and probability estimates of the effectiveness of the individual control options over a broad range of environmental conditions at different levels of hazard. Probability estimates of both hazard and control effectiveness are required for calculating the cost effectiveness of management options and for integrating options into a final pest management plan. The "Forest" perspective also provides insight for developing new research and management tactics and strategies at the regional scale, strategies such as genetic and functional diversity -- that would not present themselves when considering pest dynamics at the single tree or single stand level.

What are some of the changes necessary if IFPM is going to contribute to increased forest productivity? Regarding "Integration", the researcher needs to plan his research so that results can be integrated with the objectives and constraints of the forest manager. Questions he needs to ask are: Who is the forest manager that will be using his information -- a small landowner, or a public or private forester? What is the product -- pulpwood, sawlogs and veneer or some multiple resource? What are the forest management variables -- the stocking density, rotation age, thinning schedules, silvics of the host, silvicultural practices, etc.? The researcher should clearly understand the separate but interdependent roles of himself, the pest manager and the forest manager but adopt the attitude that his main function is to assist the forest manager in increasing productivity by helping him meet his objectives within his constraints.

The pest manager must evaluate pest management recommendations for single pests, and for multiple pest, host and disease combinations. He must resolve conflicts between recommendations and make the recommendations for each pest enhance one another. He must pilot-test control recommendations, determine there effectiveness and calculate costs. The pest manager must integrate and assign control recommendations into a pest management system according to levels of hazard, and their cost-effectiveness. He must integrate pest management recommendations with the objectives of the forest manager.

The forest manager needs to list his objectives and constraints. He needs to supply information on the value of his product or product mixture so that the economic threshold and hazard zones can be established and that the benefits and cost elements of the benefits/cost ratio can be calculated. He also must evaluate pest management recommendations and integrate them with his forest management objectives and constraints.

Changes are needed in our view of "Pests". The researcher and pest manager must remember that a pest only becomes a pest when impact reaches or exceeds some specified economic threshold. Depending on his valuation of benefits and control costs and his economic threshold the definition of "pest" will vary among forest managers. Up to the limit of his own economic threshold, the forest manager can tolerate some level of pest loss. Similarly, control measures need not be 100% effective. They simply have to reduce impact to a level somewhat below the economic threshold.

Researchers need to understand the importance of the incidence - impact curve because impact represents one cost element in benefit/cost analyses and it, plus a risk factor, is the basis for establishing hazard levels. Hazard rating and forecasting enhance the economics of control by allowing the pest manager to apply control measures only when and where impact is or will be significant and to forget about them elsewhere. Impact estimates should include losses from mortality, growth and yield, product devaluation, non-timber resource devaluation and costs of changes in the forest manager's scheduling and operations. Impact should be evaluated at the tree, stand, and forest level. Even the geometry of impact needs to be considered. For example, the effect of tree mortality on stand yield for a disease like Phellinus or annosus, which kill trees in groups to produce nonproductive holes, will be much greater than one like the blister rusts which kill more randomly and act as a thinning agent. The pest management recommendations especially in plantations must consider the differing effects on stand productivity of the two types of disease. Appropriate stand and regional, impact and decision models need to be constructed for the major pests.

In relation to the "Management" aspects of IFPM, the researcher should furnish probability estimates of the effectiveness of control options over the range of hazard. Such estimates are required for evaluating economic risks and by the pest manager for coordinating an integrated management plan. When an economic analysis of a pest management decision is not possible, the researcher should understand that his perception of risk in decision-making may not be the same as the forest managers. The researcher may require confidence limits of 95 or 99% in his experiments. The forest manager, operating in the absence of good pest information, may be happy with 55% correct decisions. If the researcher and the pest manager cooperate to improve this level to 75%,

they have made a valuable contribution. Researchers need to use imagination in devising and testing new control tactics and strategies. For example, what are the opportunities for employing silvicultural practices such as site preparation, genetics, fertilization, prescribed burning and thinning. The pest manager should explore systems analysis procedures and computer technology in evaluating impact, mapping hazard, and calculating the cost effectiveness and applying management recommendations at the stand and regional levels. The forest manager should be willing to incorporate pest management recommendations into his forest management operations when they are shown to increase productivity.

Intensive forest management has created pest problems at a rate beyond our capacity to cope using past research approaches. Tree improvement programs, seed orchard and genetic engineering technologies promise to accelerate the pace. The need and opportunities for pest research and pest management are greater than ever. Epidemiology and IFPM represent reasonable, realistic approaches to pest research and pest management if forest managers, pest managers, and researchers each understnad their separate but interdependent roles and work together toward a common goal of increasing forest productivity. This will involve forest managers stating their objectives and constraints, and furnishing realistic economic valuations of all the resources to be protected. Researchers will have to acknowledge that their function is to develop the basic biological and ecological pest knowledge from an epidemiological orientation. The pest managers can then apply this knowledge to evaluate and forecast hazard and assign control options on the bases of their cost-effectiveness and suitability in meeting the objectives of the forest manager. The bottom line for all of us is increasingly going to be an economic one. If IFPM is not new, I believe it is time to acknowledge that it will have to be viewed differently than in the past if it is to effectively increase forest productivity.

Crying Wolf and the Consequences of Giving Conflicting and Erroneous Information to Land Managers

Panel Members: Greg Filip, Borys Tkacz, Randy Fuller, Craig Schmitt, and Bill Livingston

OPENING REMARKS:

This panel will be quite different from most anything presented at other work conferences. We will give no papers. We will present no new data. We won't even present old data and make you think it's new data. Instead, we would like to explore the consequences of giving erroneous and conflicting advice from pathologists to the forest land manager. As professionals, we will always have slightly different opinions about exact causes of disease and management recommendations. But how is this perceived by the land manager who often wants a quick solution to his problem? How does he view so-called "friendly differences" in interpretations between pathologists? We would like to further explore this subject with your help. The main idea for this panel originated with Jim Hadfield, who, unfortunately, could not be here. I'm not sure if this was unintentional or by design. Jim may have been inspired by Bill Bloomberg's presentation last year concerning the consequences of failing to conduct surveys.

We would first like to present five situations that Joe, the Land Manager, has to face when managing for disease on his working circle. I have distributed a summary of these five situations for you to use as a reference for the spirited discussion that will follow. As you view these situations, don't be embarrassed if you can identify with one or more of the cases. You are certainly not alone. We all may be guilty of some of these mistakes at some time in our careers. Hopefully, they were or will be much less flagrant than the situations that will be presented here.

The main purpose of our presentation will be to serve as a stimulus for discussion. Hopefully, you may see and discuss other situations of a similar nature that may occur between the pathologist and the land manager. Also, I hope that we can discuss ways of minimizing the inevitable disagreements that arise between pathologists so as to avoid the subsequent problems for the land manager when attempting to make management decisions.

SCENE 1

Slide #1 - Title, Christmas Tree Plantation

Narrator: Our first scene opens at the Christmas tree plantation operated by Joe, the Land Manager. Now this is the first time that Joe has grown Christmas trees on this portion of recently cutover land. He has a root rot problem and has decided to consult a pathologist.

Enter Pathologist and Joe, the Land Manager.

Slide #2 - Douglas-fir Plantation

Pathologist (shaking hands with Joe): Hello, my name is Randy, the Pathologist. You must be Joe, the Land Manager?

Joe: That's right. I'm sure glad you could make it. I've been tearing my hair out worrying about what to do with my trees.

Pathologist: Well, it looks pretty bad, but let's take a closer look, and we'll see what you have.

Slide #3 - Swiss needle cast

Pathologist: Look here at these needles. See these little spots. These are caused by a fungus, *Phaeocryptopus gaumanni*. That's Swahili. Anyway, it causes a disease called Swiss needle cast. Infections occur in the spring at budbreak during wet years. I suggest you spray once with chlorothalonil at budbreak. Here's a card from a local chemical representative. Get in touch with him. He'll set you up.

Joe: You think that's all it is?

Pathologist: No doubt about it.

Joe: Well, I sure appreciate your help. See you later (shaking hands).

Exit Pathologist.

Slide #4- Spray operation

Joe (talking to audience): Well, I'm sure glad I consulted a professional. This spraying is expensive, but it will be worth it if I can sell decent-looking trees.

SCENE 2

Slide #1 - Title, Weather Damage

Narrator: Our second scene opens in another part of Joe's management area. Joe has had some cold weather this winter, causing a lot of frost damage in his pine plantation. Our scene begins with Joe talking to his technician.

Enter Joe and Technician.

Slide #2 - Weather damage

Technician: What are we going to do about this damage, Joe? Maybe we should consult a pathologist?

Joe: Not after the last episode I had with one. Chlorothalonil didn't work very well for root rot.

Slide #3 - Pest Alert

Joe: I received this Pest Alert in the mail last week. Maybe this is what we have. Most mortality is usually caused by some kind of root rot, anyway.

Slide #4 - Borax treatment

Joe: Borax sure is cheap and easy to apply. And it's got to be much better than treating Armillaria with chlorothalonil.

SCENE 3

Slide #1 - Title slide

Narrator: The third scene opens in a part of Joe's mixed-conifer stand where laminated root rot is causing unacceptable mortality. Joe decides to consult two pathologists this time and get independent diagnoses.

Enter Pathologist 1 and Joe

Slide #2 - Grand fir mortality

Pathologist 1: (shaking hands with Joe): Hello, Joe. Good to see you again.

Joe: Yeah, I hope we can do something about this mortality, Randy. You're not going to recommend chlorothalonil sprays again?

Pathologist 1: Sorry about that. I think we can do a better job this time. I'm more used to working in older stands, anyway.

Joe: Well, what have we got, Doc.?

Pathologist 1: Well, I'm pretty sure that what's killing your firs are engraver beetles, *Scolytus ventralis*.

Joe: How can you tell?

Slide #3 - Engraver galleries

Pathologist 1: See these galleries under the bark. They're caused by fir engraver larvae. The bug causes substantial damage, generally in weakened fir. I think if you salvage the dead and dying trees and thin the remainder to improve vigor, you should do alright.

Joe: No chlorothalonil sprays?

Pathologist 1: Not this time, Joe, just a sharp chainsaw.

Joe: Well, I hope you're right. See you soon (shaking hands).

Exit Pathologist 1

Slide #4 - Title - One Week Later

Enter Pathologist 2

Slide #5 - Grand fir mortality, same stand

Pathologist 2: (shaking hands with Joe): Hi, I'm Craig, the Pathologist. You must be Joe?

Joe: That's right. I'm sure glad you could come. Here's our problem. What do you think is happening?

Pathologist 2: The problem with the larch is the severe dwarf mistletoe infections. These really drain the life out of a tree. I'd salvage all the infected larch before they die and at the same time prevent infection in the larch regeneration.

Joe: What about the dying fir?

Pathologist 2: I wouldn't worry about the fir. If you take care of the larch and open up the stand, the fir should do alright.

Joe: Well, sounds good to me. Thanks for the advice (shaking hands).

Exit Pathologist 2

Joe (talking to audience): What am I to do? Two different opinions, and I don't like either one.

Slide # 6 - Clearcut

Joe (talking to audience): I guess I'll just harvest the sucker and start over with a new stand of fir.

SCENE 4

Slide #1 - Title, DF Root Rot

Narrator: Joe is really skeptical of pathologists now. But he still has root rot problems in another part of his forest. Reluctantly, he decides to consult two pathologists again.

Enter Pathologist 1 and Joe

Slide #2 - DF root rot

Pathologist 1: (shaking hands with Joe): Hi, Joe. Good to be back here again. Root rot problems, again?

Joe: I think so.

Pathologist 1: Let's take a look.

Slide #3 - Laminated decay

Pathologist 1: Yes, it's laminated root rot. See the typical laminated decay caused by the fungus. It's diagnostic for *Phellinus* and *Phellinus* alone.

Joe: What should I do, use a sharp chainsaw again?

Pathologist 1: You'll need to do more than that. The fungus lives in the stumps many years after stand harvest, so you'll need to treat the stumps after harvesting. I suggest you evacuate all the stumps with a bulldozer before you plant.

Joe: I don't know, Randy. That ground is pretty steep. I don't think I can get an evacuator in here.

Pathologist 1: Well, Joe, if you don't get rid of these stumps, you won't be able to grow fir again.

Joe: Well, thanks for your help. I'll see what I can do (shaking hands).

Exit Pathologist 1

Slide #4 - Title, One Week Later

Enter Pathologist 2

Slide #5 - DF root rot, same stand

Pathologist 2: (shaking hands with Joe): Hi, Joe. Good to see you again. I hope I can give you some better advice this time.

Joe: I hope so, too.

Pathologist 2: Let's see what you have.

Slide #6 - Armillaria mycelial fans

Pathologist 2: Looks like Armillaria is killing your trees, Joe. See these mycelial fans beneath the bark. They're diagnostic for Armillaria. I'd recommend harvesting the stand and planting vigorous fir stock to get the stand into a healthy condition again. Your stand has stagnated, that's all.

Joe: Well, Craig, thanks for the advice. I'll contact the Logging Department as soon as possible. See you later (shaking hands).

Exit Pathologist 2

Slide #7 - Clearcut

Joe (talking to audience): I'm sure glad I consulted two pathologists. At least, I now have a choice of what to do. Removing stumps would have been impossible on that ground.

SCENE 5

Slide #1 - Title, DM in pine

Narrator: The last scene opens in Joe's pine plantation. By now, he's convinced that all pathologists are charlatans. Joe has some dwarf mistletoe in his pine and would like to treat it. He knows he's got mistletoe. He can see the plants. However, he decides to consult a pathologist and test him a little.

Enter Pathologist 1 and Joe

Slide #2 - DM infected stand

Pathologist 1: (shaking hands with Joe): Good to see you again, Joe. What can I do to help?

Joe: What do you think about this stand, Randy?

Pathologist 1: Dwarf mistletoe, no doubt about it.

Slide #3 - DM plants

Pathologist 1: See these plants, they contain the mistletoe seeds; deadly on healthy branches.

Joe: What's your advice?

Pathologist 1: I'd destroy the stand and start over. Infections are only going to intensify.

Joe: OK, Randy. I'll get on it as soon as possible. See you later (shaking hands).

Exit Pathologist 1

Slide #4 - Destroyed stand

Joe (talking to audience): I hope he was right. I hate to destroy these stands. They're so hard to establish.

Slide #5 - Title, One Year Later

Narrator: Joe has done a lot of stand burning in the name of dwarf mistletoe. About a year later, Joe decides that there must be a better way. He consults another pathologist.

Enter Pathologist 2

Slide #6 - Similar pine stand

Pathologist 2: (shaking hands with Joe): How goes it, Joe. How can I help?

Joe: What can I do with these stands? I hate to destroy them, they're so hard to get started again. But these mistletoe infections really worry me.

Pathologist 2: I wouldn't worry about stands like these. The latest information says that light-to-moderate amounts of dwarf mistletoe in thinned pine stands cause practically no growth loss. I wouldn't be concerned at this point. It's too bad you destroyed those other stands.

THE END

TRENDS IN OZONE INJURY TO PINES IN THE SIERRA NEVADA

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Background Information

Air pollution was identified as causing a decline of pines in the San Bernardino Mountains of southern California in 1962. It was not until 1971 that symptoms of ozone injury were first detected on pines in the Sierra Nevada of northern California. The location of this first report was 50 miles east of Fresno on the Hume Lake Ranger District of Sequoia National Forest and near the Grant Grove area of Kings Canyon National Park.

The Forest Pest Management (FPM) Staff began to document additional locations of injury in the southern Sierra in 1974 and by 1977 completed an extensive ground survey on the Sierra and Sequoia National Forests. During this survey 242 plots were evaluated for air pollution injury and 52 of those plots have since been revisited to assess annual trends in injury. The rating system used to evaluate injury was based on the amount of chlorotic mottle found on ponderosa and Jeffrey pine foliage.

Also, FPM has monitored ambient ozone levels at numerous sites in the Sierra using Dasibi Ozone Analyzers. One site - Whitaker Forest - has been monitored every year since 1976. This forest is east of Fresno in the same area where chlorotic mottle symptoms on pines were first detected in 1971.

Results

Table 1 compares the amount of ozone injury in 52 trend plots evaluated between 1977 and 1980/81. Table 2 shows how the injury levels in these plots have changed during this time period.

Table 1. NUMBER OF INJURY TREND PLOTS BY RATING CLASS IN 1977 AND 1980/81

	NUMBER OF PLOTS	
RATING CLASS	1977	1980/81
Very Severe	0	0
Severe	0	7
Moderate	11	11
Slight	20	25
No Symptoms	21	9
Total:	52	52

Table 2. CHANGES IN OZONE INJURY BETWEEN 1977 AND 1980/81 TREND PLOTS

STATUS OF INJURY	NUMBER OF PLOTS 1980/81
Increased Injury	35 (15)*
No Change	10
Decreased Injury	7 (0)*
Total:	52

* Number in parentheses equals plots with significant change at P = 0.05.

The ozone monitoring results for Whitaker Forest are summarized in Table 3.

Table 3. NUMBER OF HOURS DURING WHICH THE FEDERAL OZONE STANDARD (12 PPHM) AND CALIFORNIA STATE OZONE STANDARD (10 PPHM) WERE VIOLATED AT WHITAKER FOREST, JUNE-SEPTEMBER, 1977 THRU 1981

YEAR	NUMBER OF HOURS FEDERAL STANDARD	STATE STANDARD
1977	42	561
1978	35	390
1979	16	389
1980	14	223
1981	12	343

Conclusions

FPM air pollution work in the Sierra Nevada has concluded the following:

1. Ozone symptoms are common and widespread in the areas surveyed.
2. Ozone injury on pines has increased annually while ambient concentrations of ozone have fluctuated.
3. Federal and State Ozone Standards are frequently exceeded at many sites.
4. No tree mortality to date has been attributed to ozone.
5. Sensitive pines will continue to decline as ozone moves into the Sierra Nevada each year.

OZONE INJURY AND HEIGHT GROWTH OF PLANTED PONDEROSA PINES IN THE SOUTHERN SIERRA NEVADA OF CALIFORNIA

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ABSTRACT

Ozone injury to ponderosa and Jeffrey pines was first discovered in the southern Sierra Nevada in 1970. Symptoms are now widespread on the Sierra and Sequoia National Forests, yet little is known about ozone's effects at present injury levels. To determine whether height growth of young ponderosa pines is related to severity of symptoms, I surveyed five plantations on the Hume Lake Ranger District of the Sequoia National Forest in 1978. Ozone had been monitored at nearby Whitaker Forest since 1976, and reached the highest levels to date in the year before the survey.

Nearly one-half of the trees in the plantations had ozone injury symptoms, and some 15% were injured on second-year needles; symptoms on current-year needles were very rare, however. I examined two commonly-used ozone injury rating systems, and found that the severity of needle symptoms was unrelated to height growth at present injury levels. Although the pines declined in height growth in 1977 and 1978, the 1976-77 drought, brush competition, and insect injury probably masked whatever effects ozone may have had.

At current pollutant levels in the southern Sierra, ozone does not appear to be an overriding concern in young plantations. However, these pines have been exposed to excess ozone for a few years only, and the long-term effects of this exposure are still not known. Continued monitoring of symptom development and the possible delayed effects of visible injury are essential.

SURVIVAL OF DOUGLAS-FIR INJECTED WITH FUMIGANTS CHLOROPICRIN OR METHYLISOTHIOCYANATE

by
W.G. Thies and E. E. Nelson

ABSTRACT

Douglas-fir trees in a 45-year-old stand in northwestern Oregon were classified as infected, probably infected, and noninfected according to infection by Phellinus weirii. Trees in each infection class were treated with chloropicrin or methylisothiocyanate at one of three dosage rates or left untreated. The amount applied to each tree was proportional to its estimated biomass. The highest dosage used has been shown to kill P. weirii in stumps. Five months after treatment all treated trees were alive. Chloropicrin-treated trees show the most severe symptoms of chemical toxicity. Toxicity symptoms are more severe on noninfected trees than on infected trees and are more severe with increasing dosage of the chemical. The fumigants appear to be at higher concentrations in the sapwood than in the heartwood at 1, 2, and 3 m above stump height. An unidentified volatile, fungistatic compound appears to be present in higher concentrations in the sapwood than in the heartwood in all trees sampled.

INTRODUCTION

Phellinus weirii (Murr.) Gilb, cause of lamimated root rot, infects nearly all commercially important conifer species in western U. S. and Canada. It reduces forest productivity in Western North America annually by about 4.4 million m3 (Nelson et al. 1981), with 0.9 million m3 of loss in western Oregon and Washington Douglas-fir (Childs and Shea 1967).

When infected trees die, the pathogen can continue to live saprophytically in dead roots for 50 years or more (Childs 1963, Hansen 1979). Infection in a young stand begins when developing roots of young trees contact residual, infested stumps and roots from the preceding stand. The infection spreads between living trees via root contact (Wallis and Reynolds 1965). As the fungus advances along a tree's roots, the roots distal to the fungus are killed, denying the tree water and nutrients necessary for growth. As roots decay, a tree is robbed of structural support and, eventually, can be windthrown.

Immediate replanting with Douglas-fir on a site infested with P. weirii usually results in continuation of the disease and subsequent losses in the new stand. Strategies for control of laminated root rot currently being tested include inoculum removal, high nitrogen fertilization, chemical agents, biological agents, and species manipulation (Thies 1981). Control of this disease in living infected trees has not been reported as yet.

Extensive stain on freshly cut stumps typically indicates a spongy or hollow area of advanced decay in the root collar contiguous with advanced decay and stained wood in the major roots. The presence of this "duct work" of advanced decay suggests that injection of fumigants into stumps may effectively kill P. weirii and eliminate these sources of inoculum.

Several soil fumigants have been reported to eradicate pathogenic fungi from infested wood buried in soil including chloropicrin (trichloronitromethane) (Godfrey 1936), carbon disulfide (Bliss 1951), methyl bromide (Rackham et al. 1968), and Vapam (sodium N-methyldithiocarbamate, ai 32.7%) (Houston and Eno 1969). Decay fungi were effectively eliminated from power transmission poles injected with chloropicrin, Vapam, methyl bromide, or

Vorlex (20% methylisothiocyanate, C3 chlorinated hydrocarbons) and the decay was controlled for at least 10 years (Graham 1975, Graham and Corden 1980). Armillariella mellea was effectively eradicated from infected ponderosa pine stumps injected with methyl bromide, carbon disulfide, Vorlex, chloropicrin, or Vapam (Filip and Roth 1977). Allyl alcohol, chloropicrin, Vapam, and Vorlex eradicated P. weirii from infested Douglas-fir stumps within 1 year (Thies and Nelson 1982).

In a recent study, eight live Douglas-fir trees from 47- to 73-cm diameter breast high (d.b.h.) injected with chloropicrin (125 to 500 ml/tree) were alive and appeared healthy 3 years later (personal communications B. G. Goodell and G. G. Helsing, August 1982, Oregon State University, Corvallis, Oregon).

The discoveries that chloropicrin could be used to eradicate P. weirii from infested stumps and roots and that trees can survive injection with this fumigant suggested the possibility of therapeutic application of fumigants to Douglas-fir infected by P. weirii.

In this paper we report on the survival of live Douglas-fir 5 months after injection of chloropicrin 1/ or methylisothiocyanate (MIT) above the root collar, and results of our attempt to trace the activity of the fumigants in the trees. We feel these interim results, though not statistically based, suggest some interesting possibilities worthy of discussion at this conference.

MATERIALS AND METHODS

Subject trees

The study area, in the Oregon Coast Ranges near Apiary, Oregon, supports a 45-year-old stand of predominantly Douglas-fir. Candidate Douglas-firs were selected near the periphery of a 3-ha clearcut, around openings created by P. weirii, and along roads, thus providing good opportunities to observe crowns. Each tree was examined for positive signs or symptoms of infection by P. weirii. The root collar and several major roots were examined for the presence of typical P. weirii ectotrophic mycelium; however, cutting into major roots was avoided. Increment cores were taken from each tree as needed to determine if decay typical of P. weirii was present in major roots or at the root collar. Each resulting hole was plugged with 10-cm length of hemlock dowel. Each tree examined was measured (d.b.h.), numbered, marked with two identical aluminum tags at groundline, and classified into one of three infection classes:

I--P. weirii infected;

II--Probable P. weirii infection, reduced leader and needle growth or a positive inoculum source within 5 m of the subject tree, but P. weirii not identified on the subject tree;

III--Noninfected, crowns appeared healthy, no identified inoculum source within 17 m of the subject tree.

1/ This paper reports the results of research only. Mention of a pesticide does not constitute a recommendation for use by the U.S. Department of Agriculture, nor does it imply registration under FIFRA as amended. Also, mention of a commercial or proprietary product does not constitute recommendation or endorsement by the U.S. Department of Agriculture.

Photo points were established as needed to obtain a reasonably unobstructed view of the upper crown of each candidate tree. At least one picture was taken of the crown of each candidate tree.

The trees in each infection class were separated into five groups of nine similar trees based on tree diameter, crown condition, and location. Each treatment was assigned to one tree within each group. Treatments within a group were randomly assigned.

Treatments

Two fumigants were tested in this study: chloropicrin and MIT. Both Chloropicrin and Vorlex have been shown to be effective in eradicating P. weirii from infested stumps (Thies and Nelson 1982). Vorlex is MIT (20%) and chlorinated C3 hydrocarbons including dichloropropenes, dichloropropane, and related chlorinated hydrocarbons. Pure MIT is an amorphous "solid" at room temperature. If effective, it would be more advantageous to use MIT than Vorlex: less weight to carry, safer and easier to use than a liquid, and use of toxic chlorinated hydrocarbons would be avoided.

There were nine treatments planned: chloropicrin--applied at each of four dosage rates, MIT--applied at each of four dosage rates, and check--no treatment applied.

Application

A torque converter and chuck mounted on a chainsaw motor were used to drill 3.18-cm-diameter holes in treatment trees. Holes were drilled at approximately a 45-degree angle below the horizontal plane and extended past the center of the tree. For the lowest dosage the holes were equally spaced around the tree and approximately 30 cm above the soil line. For the other dosages the holes were drilled 15 cm apart on a spiral that started approximately 15 cm above the soil line and moved approximately 30 cm up the bole with each turn around the tree. After fumigant had been placed in the holes, they were plugged with a 12.5-cm-long by 3.33-cm-diameter hemlock dowel leaving 1 cm exposed to aid in relocating the treatment holes. Plugs were prepared by beveling one end of each dowel and dipping it in resorcinol glue to form an impermeable cap.

Chloropicrin was applied as a liquid and MIT as a solid. To reduce handling, MIT was premeasured in units of 58 g each into polyethylene sacks.

Fumigant dosage

We interpreted results from our stump fumigation study to indicate that the effective dosage increased with increasing stump size (Thies and Nelson 1982). Based on fumigant movement in poles (Graham and Cordon 1980) at wood moisture contents of about 30 to 40 % which is typical of heartwood of living Douglas-fir trees, we assumed that fumigant vapors would diffuse almost 2.4 m above the root collar.

For this study we calculated dosage as though we were only treating the stump, roots and first 2.4 m of the stem; and we assumed that the effective dosage for each tree would increase linearly with an increase of the estimated treated biomass. We estimated treated biomass (2.4 m of bole and major roots to 1-cm diameter) for each 2.5-cm-d.b.h. class. Example values are listed in Table 1 and were derived from the following relationships:

1. Stem Biomass (to 2.4-m height)--$Y = 0.0007128 + 0.0002716\ X$.
   Where, Y = Stem volume in cubic meters,
   X = Basal area in square centimeters ($n = 47$, $r^2 = .993$,

Sample trees ranged from 17.0- to 61.7-cm d.b.h.). Data were from Douglas-fir in a single stand in the Coast Ranges near Apiary, Oregon, (Thies unpublished data). Wood density was assumed to be 0.44 g/cm3.

2. Below Ground Biomass--ln Y = -4.6961 + 2.6929 ln X. Where, Y = below ground biomass in kg, X = d.b.h. in cm, ln = logarithms to the base e, ($n = 26$, $r^2 = .96$. Trees ranged from 2.3- to 23.0-cm d.b.h. and from 94- to 135-cm d.b.h.). Data were from two stands on the west slope of the Cascade Range (Gholz et al. 1979).

In our stump fumigation study, 1000 ml of either chloropicrin or Vorlex poured into stumps was found to eradicate all P. weirii from stumps less than 48-cm diameter. We will assume that 1000 ml applied to a 48-cm diameter stump is the minimum effective dosage required to eradicate P. weirii. We estimated the mass of a 48-cm-diameter stump to be 156 kg. Thus, we will assume that 10 ml/1.5 kg of biomass is an approximation of the minimum effective dosage. Since 10 ml of Vorlex contains 2.3 g of MIT, a standard dose of MIT will be 2.3 g/1.5 kg of stump and root biomass. For ease of application, the dosages were rounded up to be the equivalent of the next quarter liter of chloropicrin or Vorlex (58 g of MIT). In treating living trees to 2.4-m-bole height, a standard dose (D) of chloropicrin or of MIT will mean a dose as listed on Table 1 based on the tree's d.b.h. Stated in terms of the standard dosages, the nine planned treatments are as follows:

chloropicrin at 2D, 1D, 0.5D, and 0.25D;
MIT at 2D, 1D, 0.5D, and 0.25D;
check--holes were neither drilled nor was fumigant applied.

Each of nine treatments were to be applied to five trees in each of the three infection classes. Treatments were applied in March of 1982; but for various reasons, we decided to defer the 2D treatments until after we determined if the live trees could survive lesser dosages.

Data collection

In mid-August 1982, the study trees were visited to observe toxicity symptoms and to collect samples for an exploratory study of fumigant movement within the boles of treated trees.

Each study tree crown was examined and notes were made of needle loss, stunting, yellowing, browning, or drooping of new growth. Additionally, pictures were taken from the established photo points of the crowns of trees in the first two replicates. A 10-point symptom-severity (S-S) scale was established and used to rate each tree from dead (0) to no apparent effect (10).

We used a closed-tube bioassay (Scheffer and Graham 1975) to determine the distribution of fungitoxic levels of fumigant within the bole of selected trees. Earlier reports indicate that the fumigants may remain in wood for a considerable period of time (Thies and Nelson 1982, Graham and Corden 1980). Tube slants of malt agar were prepared in 15- by 100-mm screw-top tubes and inoculated with a 3.5-mm-diameter plug cut from the margin of a young colony of Poria placenta. After 3 day's incubation at room temperature, the tubes were inverted, placed in a cooler, and taken to the field.

Samples were collected from six trees of one replicate: trees from the extreme infection classes (I--infected, III--noninfected) treated with the highest dosage (D) of each chemical, and the checks. A 4-mm-diameter increment core was taken from each tree at 1, 2, and 3m above the highest treatment hole. Two, 2.5-cm samples were cut from the increment core: sapwood--the first 2.5-cm segment beneath the bark; heartwood--the second 2.5-cm segment

into the heartwood. Each core segment was placed in a still inverted test tube, the screw-top lid replaced, and the tube numbered and marked to indicate current colony margin of the test fungus. Notes were made for each core segment as to tree number, height of collection, and type of wood. After the tubes were incubated for 5 days at 27°C, colony extension, since the core segment was put into the tube, was measured. Extension of the fungus in tubes over core segments from fumigated trees was compared with that in tubes with core segments from check trees and in tubes without core segments. Retarded growth was taken to indicate the presence of fungitoxic vapor. The magnitude of retardation was taken as a reflection of the relative amount of vapor contributed by the core segment. Previous work has shown P. placenta to be very sensitive to the presence of trace quantities of either chloropicrin or MIT and thus a useful bioassay organism (Scheffer and Graham 1975).

A variation of the above test using P. weirii as the test organism was performed using a second increment core collected 2 cm below the first.

RESULTS

Of the 105 trees included in the study, none were dead 5 months after the study was installed. The general condition of nontreated trees (checks) was unchanged. Of the 45 trees treated with MIT, 34 were given a S-S rating of 10. The average S-S rating for all MIT treated trees was 9.4. One MIT-treated tree was seen to have some branches with all brown needles. These brown branches were scattered in the area of mid-crown. Toxicity symptoms on most MIT-treated trees were limited to needle loss and stunting in the upper crown.

Of the 45 trees treated with chloropicrin, 2 were given a S-S rating of 10. The average rating of all chloropicrin treated trees was 6.5. One tree appeared near death with only a few green needles left on a few branches. Twelve trees had symptoms of severe browning with entire branches having all needles exhibiting a reddish-brown color suggesting a quick death. The brown branches typically were located in mid-crown. Such trees often had some stunting and needle loss but ample green needles in the upper crown, branches of brown needles in mid-crown, and branches appearing near normal in the lower crown.

Toxicity symptoms increased in severity with increasing dosage and with increasing apparent health of the trees (Figure 1). The noninfected trees appeared to be more adversely affected by the fumigants than were the infected trees.

In the closed-tube bioassays, colonies of P. placenta grew slightly slower (15%) than P. weirii; but their relative growths over similar sample cores were parallel. To simplify further comparisons, measurements of colony extension over comparable core segments were averaged for the two organisms. Colony extension in five tubes of each test fungus without core segments was measured in the same manner as tubes with the core segments. The mean growth in these 10 tubes was taken as the "expected" extension of the colonies. Extension of colonies over the core segments is stated as a percent of the growth of colonies without core segments (Figure 2).

With one exception, for every increment core tested colony extension was less over a core segment from the sapwood than over a core segment from the heartwood. The exception was the core collected 1 meter above the last treatment hole in the chloropicrin-treated noninfected tree in which case growth of the fungi was completely stopped over core segments from both the sapwood and the heartwood. In that particular tree, growth over sapwood core

segments was drastically reduced for samples collected at all three heights in the tree.

DISCUSSION

It is gratifying that 5 months after placing two highly toxic soil fumigants into live Douglas-firs that none of the treated trees have died although one chloropicrin-treated tree appears near death. It is important to keep in mind, however, that the primary purpose of our study is to find a control that will kill P. weirii and not kill the tree. Even though we know that the applied dosage was adequate to kill the fungus in stumps we do not know if the chemicals have affected the fungus in the trees. This is an important point since survival of the tree is only part of our objective.

Chloropicrin is causing more severe toxic symptoms in the trees than MIT. We do not have a good explanation for the apparent severe mid-crown symptoms best seen in chloropicrin-treated trees. We speculate that chloropicrin may not have migrated much past the mid-crown level and simply had a more toxic effect on the rapidly growing branches and foliage of mid-crown than on the more senescent lower crown.

The observation that higher dosages of the fumigant cause more severe toxic symptoms was expected and requires no further comment.

That infected trees show fewer toxic symptoms than noninfected trees fits a basic assumption made when we proposed the treatments--that the rot column would absorb or channel away most of the fumigant from living tree tissue.

For every increment core tested, the test fungi grew slower in the closed-tube assay over a core segment taken from the sapwood than over a similar core segment taken from the heartwood. The core segments were not in contact with the agar. We speculate that a volatile material, released in a larger quantity from the sapwood than from the heartwood, caused a reduction in the growth of the test fungi. Looking at data from the non-treated trees, we found more of a depression of colony extension in samples from noninfected trees than from infected trees.

At least two obvious (and very speculative) possible explanations immediately come to mind:

1. The disease, working on the roots and stressing the tree, caused a shift in the tree's physiology resulting in a reduction in the amount of fungistatic material produced.
2. If there is natural variability of production of the fungistatic material in sapwood perhaps the noninfected tree remains disease free because of an adequate production of the fungistatic principal. Even if a higher than normal production of the fungistatic principal does not prove to be an absolute defense against P. weirii, it may serve to slowdown the advance of the fungus and provide that tree with a survival advantage. We speculate that if such a fungistatic principal proves to be real it may be the basis for genetic selection of Douglas-fir resistant to laminated root rot.

Examination of the closed-tube assay data from the chloropicrin-treated trees indicates that the chloropicrin is moving up the tree to a greater extent in the noninfected tree than in the infected tree. These data are consistent with the development of crown symptoms and lend weight to the speculation that the fumigant is being kept low in the infected tree possibly concentrating in the ducts created by P. weirii.

We attempted to place the fumigants in the heartwood and anticipated that the fumigant would have more of a tendency to move in the heartwood than in the sapwood. Our data appear to support the conclusions that the fumigants are moving up the trees largely in the sapwood. Data from the

chloropicrin-treated noninfected trees seem to show that the chloropicrin is causing a reduction in colony extension over samples of sapwood collected 3 meters above the highest treatment hole. There was, at time of sampling, apparently some fumigant in the heartwood samples but clearly not to the same extent or concentration as in the sapwood samples. A similar conclusion could be drawn from the 1-meter sample from both the chloropicrin and MIT-treated infected trees.

Once again, let us stress that our conclusions regarding the presence of a volatile fungistatic principal should be taken as preliminary and speculative. At this time our data base is small and needs to be augmented by additional tests.

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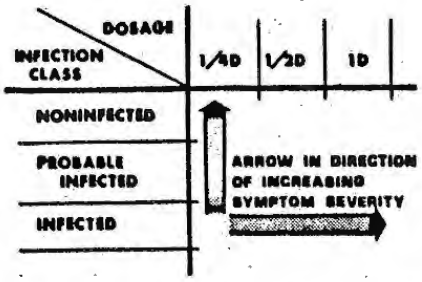

Figure 1. Crown symptom severity in relation to fumigant dosage rate and infection class.

D.B.H. (cm)	BIO-MASS (kg)	STANDARD DOSE (D) CHLOROPICRIN (l)	STANDARD DOSE (D) MIT (g)
15.0	24	.25	58
30.0	170	1.25	290
45.0	448	3.00	696
60.0	898	6.00	1392

Table 1. Example dosages for fumigants applied to live trees.

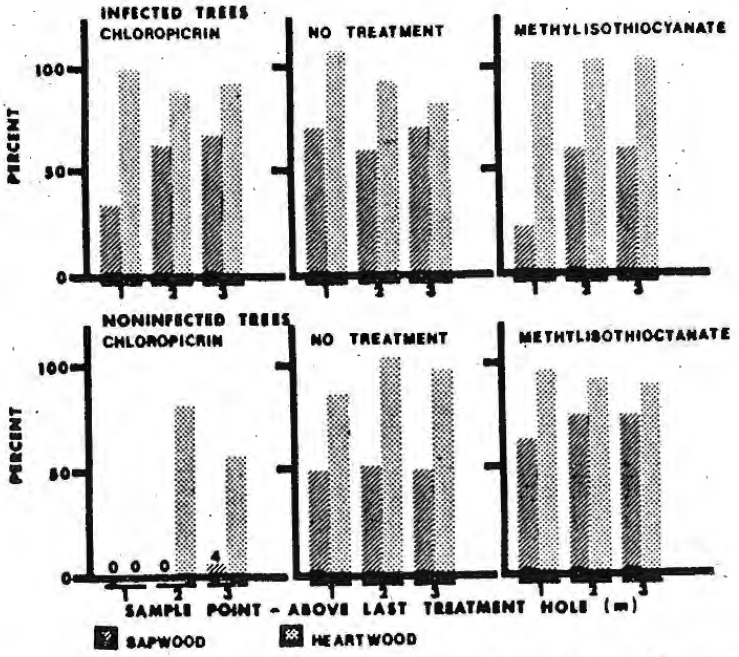

Figure 2. Colony extension in presence of sample core as a percent of extension in absence of a sample core.

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The pine wood nematode, Bursaphelenchus xylophilus in Minnesota and Wisconsin: Insect associates and transmission studies.

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ABSTRACT

The pine wood nematode, Bursaphelenchus xylophilus, was most commonly extracted from Cerambycidae emerging from nematode - infested pines in Minnesota and Wisconsin during 1981 and 1982. The highest number of nematodes were extracted from Monochamus scutellatus and M. carolinensis. Low numbers of B. xylophilus were found in some buprestid but not in curculionid and scolytid beetles examined. Two species of Cerambycidae, M. marmorator and M. scutellatus were associated with B. xylophilus from balsam fir in Minnesota. B. xylophilus from insects associated with balsam fir were morphologically different to those from insects associated with pine. Dauer larvae of B. xylophilus were concentrated in the thoracic segment of M. scutellatus and M. mutator examined. In preliminary transmission studies, B. xylophilus was transmitted to twigs during maturation feeding and to logs during oviposition of M. carolinensis.

The pine wood nematode, Bursaphelenchus xylophilus Steiner and Buhrer (Nickle) is the primary cause of a severe wilt disease of native pines (Pinus thunberghii Parl. and P. densiflora Sieb et Zucc.) in Japan (Mamiya, 1976; Mamiya and Enda, 1972). In Japan, B. xylophilus is vectored primarily by cerambycid beetles (Coleoptera:Cerambycidae) (Mamiya and Enda, 1972; Morimoto and Iwasaki, 1972). The dauer larvae (transmission stage larvae) of B. xylophilus enter the spiracles of the vectors before they emerge and are carried to young shoots of healthy trees on which the Cerambycidae undergo maturation feeding. Nematodes moult to the adult stage and enter the resin canals of host trees which exhibit rapid wilt symptoms and die within three months (Ishibashi and Kondo 1977, Mamiya, 1976). Symptoms and development of pine wilt disease in Japan have been discussed in detail in a number of review articles (Dropkin et al., 1981, Mamiya 1972, Mamiya 1976, Wingfield et al., 1982a).

Bursaphelenchus xylophilus was first found in the United States in 1929 (Steiner and Buhrer, 1934) at which time it was not recognized as a pathogen (Nickle et al., 1980). The nematode was more recently found in this country in 1979 (Dropkin and Foudin, 1979) and has subsequently been reported widespread throughout the United States on a wide range of conifer hosts, primarily Pinus spp. (Dropkin et al., 1981; Wingfield et al., 1982a). The association of B. xylophilus with dying trees in the United States has led to speculation that it may be the primary cause of mortality of native conifer species in this country (Dropkin et al., 1981).

In Japan, Monochamus alternatus Hops. is the most important vector of B. xylophilus (Mamiya and Enda, 1972; Morimoto and Iwasaki, 1972). Preliminary investigations identifying insect associates of B. xylophilus in

the United States have been reported from Missouri (Dropkin et al., 1981; Dropkin et al., 1982; Linit et al., 1982), and Florida (Luzzi and Tarjan, 1982). This investigation was intended to identify the insect species most commonly carrying B. xylophilus in Minnesota and Wisconsin. The results of preliminary transmission studies with Monochanus carolinensis are also discussed.

MATERIALS AND METHODS

Insect associates of B. xylophilus. Insects were collected from Austrian pine (P. nigra Arnold), jack pine (P. banksiana Lamb), ponderosa pine (P. ponderosa Laws.), red pine (P. resinosa Ait.), white pine (P. strobus L.) and balsam fir (Abies balsamea (L.) Mill.) wood using emergence traps; during the spring and summer of 1981 and from the wood of Austrian pine, jack pine and red pine during the spring and summer of 1982. Wood bolts placed in emergence traps were collected from recently killed trees from various locations in Minnesota and Wisconsin (Table 1). Subsamples of wood (70 gm fresh weight) from bolts were placed in Baermann funnels (Dropkin, 1980) to ascertain that B. xylophilus was present in the trees.

Wood bolts placed in emergence traps included branches (1.5 - 8.0 cm diam.) and sections from the main bole of trees (8 - 23 cm diam.). Insects emerging from red pine collected in Spring Green, Wisconsin were supplied by D. Hall, Forest Entomologist, Wisconsin Department of Natural Resources. In addition to wood samples from dead trees, bolts of Austrian pine cut from healthy, nematode free trees and subsequently infested with cerambycid beetles and B. xylophilus as reported in a previous investigation (Wingfield, 1982) were also placed in emergence traps.

Insects emerging from caged wood were collected daily. Sub-samples of Cerambycidae of unknown identification were retained and sent to Dr. D.C.L. Gosling, White Pigeon, Michigan, for identification. During 1981, insects collected were individually placed in 100 ml of water in a Waring blender and macerated for 1 minute. Macerated insects were placed in Baerman funnels and nematodes were extracted after 24 hrs. Insects collected during 1982 were lightly macerated in 100 ml of water using a mortar and pestle and nematodes were extracted by placing the macerated insects in a double layer of Kimwipe tissue in plastic weighing boats. Weighing boats were inverted over the boats containing the macerated insects to reduce evaporation.

Field collected Hylobius pales, Pachylobius picivorus and Pissodes approximatus were trapped associated with B. xylophilus infested Austrian pine in Zimmerman, Minnesota by placing freshly cut discs of white pine under dying trees. In addition, Chalcophora spp. resting on B. xylophilus infested red pine in Black River Falls, Wisconsin were hand caught. These insects were macerated and examined for the presence of B. xylophilus in the same way as insects caught in emergence traps during the 1982 investigation.

Nematodes extracted from insects were concentrated in 8 ml of water, placed in line-marked plastic culture dishes and counted under a dissection microscope. When nematode numbers were high, samples were diluted in water and two 8 ml subsamples were counted after thorough mixing. After counting, dauer larvae of nematodes extracted from the first 10 insects of any insect species collected were concentrated in a swing bucket, clinical centrifuge

using 10 ml conical centrifuge tubes. These dauer larvae were then placed on the mycelium of Botrytis cinerea Pers. ex Fr. growing on Difco potato dextrose agar in 125 ml erlenmeyer flasks. After three weeks, nematodes were extracted from cultures using Baerman funnels and adult male and female nematodes examined microscopically and confirmed to be those of B. xylophilus.

During 1982, 30 Monochamus scutellatus from Austrian pine wood and eight M. mutator from red pine wood were divided into head (including antennae), thorax (including legs and wings) and abdominal segments. These parts were macerated separately using a mortar and pestle and nematodes were extracted in plastic weighing boats as described above.

Nematode transmission. A single M. scutellatus beetle from Austrian pine was placed in each of four screen covered cages containing a two-year-old red pine, white pine, balsam fir and black spruce (Picea mariana Mill. B.S.P.) seedling to test whether this beetle species feeds preferentially on any of these trees. After one week, beetles were removed and seedlings observed for two months for symptom development. In addition, three M. carolinensis beetles were placed in single screened cages containing 3-year-old red pine seedlings and allowed to maturation feed. After 15 days beetles were removed, macerated and placed in plactic weighing boats to extract nematodes which were counted. Red pine seedlings were observed for symptom development.

Adult male and female M. carolinensis emerging from Austrian pine logs from Zimmerman, Minnesota were placed in pairs in screen cages with white pine twigs in water and with a 12 cm long pine bolt (3 -9 cm diam.) taken from healthy, nematode free Austrian pine. Beetles were allowed to maturation feed on twigs and oviposit in the logs. Logs and twigs were replaced at weekly intervals for between five and eight weeks. At weekly intervals, parts of the white pine twigs damaged by beetle maturation feeding and one cm pieces of bark and cambium surrounding all oviposition niches were removed and nematodes were extracted in plactic weighing boats. After eight weeks or when mortality of either beetle was observed, beetles were removed, macerated and nematodes were extracted using plastic weighing boats and counted.

RESULTS

Insect associates of B. xylophilus. Insects collected emerging from pine and balsam fir bolts during 1981 and 1982 and the number of nematodes extracted from these insects are presented in Tables 1 and 2. Dauer larvae extracted from Cerambycidae and Buprestidae were confirmed as those of B. xylophilus after moulting to the adult stage on Botrytis cinerea. Nematodes extracted from Ips grandicollis, Hylobius pales and Pachylobius picivorus were not B. xylophilus and were not identified. Species of Cerambycidae were the most frequently collected insects from emergence traps and most commonly contained B. xylophilus. Monochamus spp. were the most commonly collected cerambycid species emerging from pine bolts and with the exception of Xylotrechus saggitatus, Monochamus spp. were the largest insects collected and contained the highest number of nematodes. Xylotrechus saggitatus and the smaller Cerambycidae such as Amniscus sexguttatus and Neocanthocinus pusilus, contained few B. xylophilus (Tables 1 and 2).

The maximum number of nematodes extracted from a single beetle during the two collection seasons was 65039 from M. scutellatus from Austrian pine. Monochamus carolinensis from red pine in Wisconsin also contained relatively high numbers of B. xylophilus (Tables 1 and 2).

Tails of female B. xylophilus extracted from M. marmorator and M. scutellatus emerging from balsam fir logs were mucronate and unlike the more rounded tails of female B. xylophilus associated with insects from pine. The maximum number of nematodes recorded from a single M. scutellatus and M. marmorator from balsam fir were approximately the same. These maximum numbers of nematodes were lower than the maximum number of nematodes extracted from any single insect from pine.

B. xylophilus were present in head, thorax and abdominal segments of M. scutellatus and M. mutator (Table 3). The highest number of nematodes were always present in the thoracic region of insects examined.

Nematode transmission. M. scutellatus showed no preference for maturation feeding on red pine, white pine, balsam fir and spruce seedlings in cages. No mortality of seedlings on which beetles fed for one week was observed and maturation feeding wounds were overgrown by callus tissue. Four M. carolinensis allowed to maturation feed on red pine seedlings all contained B. xylophilus after 15 days. The average number of nematodes extracted from these beetles was 5872. The four red pine seedlings on which these beetles had fed were all girdled by maturation feeding damage and died.

B. xylophilus was transmitted by M. carolinesis during maturation feeding and oviposition (Table 4). Nematodes were more commonly transmitted to logs during oviposition than to twigs during maturation feeding. Transmission of nematodes did not occur during the first two weeks of feeding and oviposition. Only 7.3 percent of 316 oviposition niches examined during the eight week period contained nematodes and the number of nematodes extracted from a single oviposition niche did not exceed 64. B. xylophilus was transmitted during maturation feeding by only one of the three pairs of insects examined. This transmission occurred during the fourth and fifth weeks of feeding while oviposition transmission occurred during the fourth, fifth and sixth week of oviposition by the same beetles. At the end of the eight week period two of the three female beetles contained B. xylophilus while male beetles did not contain nematodes (Table 5).

DISCUSSION

Results of this investigation confirm that insects in the family Cerambycidae are most commonly associated with B. xylophilus in Minnesota and Wisconsin. This observation is consistent with reports from other regions of the United States (Linit et al., 1982) and Japan (Mamiya and Enda, 1972; Morimoto and Iwasaki, 1972). In Japan, B. xylophilus was reported only associated with Cerambycidae. The nematode has been reported, associated with Hylobius pales and Pissodes approximatus (Coleoptera:-Curculionidae) in the United States (Linit et al., 1982) but was not extracted from these species and the related Pachylobius picivorus here. Although certain species of weevils such as H. pales undergo maturation feeding (Baker, 1972) and would have the capacity to transmit B. xylophilus,

they apparently seldom carry the nematode (Linit et al., 1982; Mamiya and Enda, 1972) and probably do not serve as common vectors of B. xylophilus. Of the Buprestidae examined in this study, only a single insect contained B. xylophilus. Buprestidae have been reported as associates of B. xylophilus in the United States (Linit et al., 1982) but we are not aware of a report of the nematode carried by Buprestidae in Japan.

This is the first report of Monochamus marmorator, M. mutator, Neocanthocinus pusilus and Xylotrechus saggitatus carrying B. xylophilus. The absence of B. xylophilus from Ips grandicollis is consistent with reports that B. xylophilus is not carried by Scolytidae (Mamiya and Enda, 1972; Dropkin et al., 1982).

Monochamus alternatus is the primary vector of B. xylophilus in Japan and a single beetle can carry up to 175,000 dauer larvae of the nematode (Mamiya and Enda, 1972). Monochamus scutellatus and M. carolinensis from various pine species in this study were found to carry the highest number of nematodes, although these maxima were considerably lower than those reported from M. alternatus in Japan. The maximum number of nematodes and the mean number per insect for M. scutellatus and M. carolinensis in this study are similar to those from M. carolinensis in Missouri (Linit et al., 1982). The lower numbers of nematodes extracted from these species in 1982 may have been due to lower numbers of nematodes in the wood from which beetles were collected. B. xylophilus feeds on fungi (Dozono and Yoshida, 1974; Kobayashi et al., 1974; Kiyohara, 1976; Kiyohara and Tokushigi, 1971) and probably survives on fungi in dead wood after tree death. Various factors including the amount of blue stain due to certain fungi in dead pines, would influence the number of nematodes in logs and consequently the numbers of dauer larvae in beetles emerging from these logs. The third Monochamus sp., M. mutator trapped from pine in this investigation carried less nematodes than did M. scutellatus and M. carolinensis. The number of beetles of this species trapped were low. However, the number of nematodes extracted from this species when comparing nematode numbers in various body parts (Table 3) was comparable to those extracted from M. scutellatus in the same study. For this reason, we would expect that M. mutator would be an equally good vector of B. xylophilus as M. carolinensis and M. scutellatus in Minnesota and Wisconsin.

The concentration of B. xylophilus dauer larvae in the throacic region of M. scutellatus and M. mutator in this study is similar to observations on M. altermatus in Japan (Mamiya and Enda, 1982) and M. carolinensis in Missouri (Linit et al., 1982). Linit et al., 1982 have shown that B. xylophilus is most highly concentrated in the metathoracic region and that they are also carried in beetle appendages of M. carolinensis. Although results presented here are similar to those of previous investigators, it should be noted that parts of the tracheae associated with the third thoracic spiracle are located in the anterior portion of the first abdominal segment and that nematodes extracted from abdominal parts may have originated from the metathoracic tracheal system.

This paper represents the first report of insects associated with B. xylophilus from a non-pine host. Balsam fir wood sampled in Minnesota has yielded low numbers (less than 10 adults/60 gm fresh weight of wood) of B. xylophilus in comparison to the numbers of this nematode commonly extracted

from pines (Authors, unpublished). This may explain the fact that nematode numbers extracted from M. scutellatus from balsam fir were comparatively lower than those extracted from pine. The lower numbers of B. xylophilus in balsam fir is thought to be related to the limited occurrence of blue stain fungi in fir as compared with pine. In addition, balsam fir does not have resin canals in which B. xylophilus are reported to develop and move in pine (Mamiya 1972; Mamiya, 1976).

The mucronate tail of female B. xylophilus from insects emerging from balsam fir wood was similar to those observed on adult females extracted from balsam fir wood in Minnesota. An isolate of these nematodes was pathogenic on balsam fir seedlings but not on pine seedlings and was less well adapted to feed on certain fungi common on pine than B. xylophilus from pine (Wingfield, et al., 1982c). Balsam fir is the exclusive host of M. marmorator (Baker, 1972) whereas M. scutellatus is found on many conifer species including Larix spp., Picea spp., Abies spp. and Pinus spp. (Wilson, 1962). The strain of B. xylophilus from balsam fir may therefore not be restricted to balsam fir by means of vector and specialization may rather be based on other host factors. It is, however, possible that M. scutellatus from balsam fir is not able to oviposit and develop successfully in pine and vice versa.

The presence of B. xylophilus in beetles emerging from bolts used in a previous study to show that B. xylophilus is transmitted to cut timber (Wingfield, 1982) proves that B. xylophilus can survive in cut timber for a season. The nematodes apparently feed on stain fungi and emerge from these bolts with cerambycid beetles. By this means, cut timber would serve as a reservoir for the pine wood nematode as well as the associated cerambycid vectors and illustrates a life cycle for the pine wood nematode in the absence of apparently susceptible host species. Forestry practices reducing the amount of cut timber and dying trees to reduce bark beetle populations should also serve to reduce populations of Cerambycidae and B. xylophilus.

Monochamus scutellatus did not exhibit selective feeding on pine, spruce and balsam fir in this study. This may have been due to unnatural circumstances where beetles were caged and confronted with seedlings on which they may not feed in nature. Dropkin et al., 1982 report that M. carolinensis does not feed selectively on various pine species although the studies were also conducted using seedlings in cages. Mamiya and Enda, 1972 present evidence showing a dramatic decrease in numbers of B. xylophilus in Monochamus alternatus from time of emergence to the onset of egg laying. In this investigation, M. carolinensis allowed to feed on red pine seedlings for 15 days (a time period sufficiently long to include the first oviposition activity) contained a higher average number of nematodes than M. alternatus at the time of egg laying in Japan. This data is however based on a small number of beetles and may be misleading.

Transmission of B. xylophilus to cut timber and girdled trees has been discussed previously (Wingfield, 1982). Although the means of transmission was not known, it was suggested that vectors transmit B. xylophilus during oviposition on the logs and stressed trees. Preliminary transmission studies presented here confirm that B. xylophilus is commonly transmitted during vector oviposition and is consistent with similar results from Florida

(Luzzi and Tarjan, 1982). In this investigation, transmission of B. xylophilus was more common during oviposition than during maturation feeding. The fact that two out of three female beetles contained some nematodes at the end of the eight week period and that no transmission of any kind occurred during the last two weeks in any one cage suggests that transmission of B. xylophilus by its vectors is innefficient. However, once nematodes are established on a log or susceptible tree, they are able to reproduce extremely fast (Kiyohara and Suzuki, 1978; Mamiya 1972; Mamiya, 1976) and their numbers would increase dramatically.

The number of nematodes in beetles would undoubtedly affect the success of transmission during oviposition and maturation feeding. Although this investigation has shown that between 79 and 90 percent of all M. carolinensis emerging from Austrian pine logs are infested with B. xylophilus and that beetles carry a relatively high number of nematodes, it is impossible to know whether beetles used in transmission studies are carrying nematodes or how many nematodes they might contain. For this reason, additional transmission studies with higher numbers of beetles are required to better understand the patterns of transmission of B. xylophilus. In addition, transmission studies using seedlings and trees under field conditions are required to ascertain whether B. xylophilus can be transmitted and kill seedlings and trees. Preliminary studies of this nature have been reported from other parts of the United States (Linit et al., 1982; Luzzi and Tarjan, 1982).

Cerambycidae are attracted to, and oviposit in dead and dying trees and cut timber (Baker, 1972; Ikeda et al., 1980; Ikeda et al., 1981). Transmission of B. xylophilus at this stage in the life cycle of the vector will result in the pine wood nematode being present in conifers dying as a result of any biotic or abiotic cause. This may explain the association of B. xylophilus with trees in Minnesota, Iowa and Wisconsin which were stressed by various forest pathogens and insects and in which B. xylophilus appeared to be a secondary component of the disease complex (Wingfield et al., 1982b).

The role of B. xylophilus as a primary pathogen of conifers in the United States is unknown. Serious losses of pines primarily attributable to infection by B. xylophilus have been reported from Illinois (Green 1982, Malek and McClary, 1982). In addition, Scots pine in Missouri (Dropkin et al., 1981; Dropkin et al., 1982), Japanese black pine in Delaware (Adams and Morehart, 1981) and Japanese black pine and Scots pine in Maryland (Nickle, 1982) are thought to be dying as a result of B. xylophilus infection. Although B. xylophilus has been shown to kill many species of pine seedling in greenhouse inoculations (Dropkin et al., 1981; Dropkin et al., 1982) inoculation and subsequent death of trees in the field have not been reported. Luzzi and Tarjan (1982), reported unsuccessful inoculations on branches of 10-yr-old Pinus taeda L. yet obtained branch mortality by allowing nematode-infested Monochamus titillator to maturation feed on similar branches. The pathogenicity of B. xylophilus on pine seedlings and on mature trees in Japan present us with sufficient reason to carefully study B. xylophilus as a potentially important forest pathogen in the United States. Careful observations ensuring the absence of all biotic and abiotic factors capable of stressing trees are, however, necessary to ascertain that B. xylophilus was not transmitted to stressed trees during

vector oviposition and that the nematode was the primary cause of tree death.

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Table 1. Insects associated with Bursaphelenchus xylophilus-infected pine and balsam fir from Minnesota and Wisconsin and the number of nematodes extracted from insects during 1981.[a/]

Host	Source	Insect families and species	Number of Insects		Number of Nematodes / Insect	
			Examined	with B. xylophilus	Maximum	Average
Austrian Pine	Zimmerman, Minnesota	CERAMBYCIDAE Monochamus scutellatus (Say)	21	20	65,039	12415.4
		M. carolinesis (Olivier)	48	47	39,781	8094.3
		Amniscus sexguttatus (Say)	15	14	43	8.9
		Neocanthocinus pusilus	2	0	-	-
		Xylotrechus saggitatus (Germar)	10	1	3	0.3
		Chrysobothris sp.	7	2	7	1.6
		CURCULIONIDAE Hylobius pales (Herbst.)[b/]	20	0	-	-
		Pachylobius picivorus (Germ.)[b/]	20	0	-	-
		Pissodes approximatus (Hopkins)[b/]	20	0	-	-
		SCOLYTIDAE Ips pini (Say)[b/]	20	0	-	-
Austrian Pine	Durand, WI	CERAMBYCIDAE M. carolinensis	2	1	9	4.5
Jack Pine	Black River Falls, WI	M. carolinensis	2	1	48	24.0
		M. mutator Lec.	3	3	25	15.3
Ponderosa Pine	LaCrosse, WI	M. carolinensis	2	2	11,662	5834.0
Red Pine	Spring Green, WI	M. carolinensis	52	52	49,875	10515.6
		N. pusilus	1	1	14	14.0
White Pine	Independence, WI	M. carolinensis	9	7	920	138
Balsam Fir	Cloquet, MN	M. scutellatus	102	91	7,963	543.1
		M. marmorator (Kiby.)	10	9	8,652	1686.4
		BUPRESTIDAE Chrysorothris sp.	1	0	-	-

[a/] Insects collected from emergence traps containing B. xylophilus infected wood.

[b/] Insects trapped associated with B. xylophilus infected trees in the field.

Table 2. Insects associated with *Bursaphelenchus xylophilus* infected pine from Minnesota and Wisconsin and the number of nematodes extracted from insects during 1982[a/]

Host	Source	Insect families and species	Number of Insects Examined	Number of Insects with *B. xylophilus*	Number of Nematodes / Insect Maximum	Number of Nematodes / Insect Average
Austrian Pine	Zimmerman, MN	CERAMBYCIDAE *Monochamus scutellatus*	8	7	22570	4871
		M. carolinensis	49	39	10872	4538
		Amniscus sexguttatus	104	25	7793	236
		Neocanthocinus pusilus	22	6	101	8
		Xylotrechus saggitatus	10	0	-	-
		BUPRESTIDAE *Chrysobothris* sp.	1	1	4068	4068
Austrian Pine[b/]	Zimmerman, MN	CERAMBYCIDAE *M. scutellatus*	5	5	23827	9149
		M. carolinensis	1	0	-	-
Red Pine	Black River Falls, WI	*M. mutator*	2	2	325	195
		BUPRESTIDAE *Chalchophora* spp.[c/]	29	0	-	-
		SCOLYTIDAE *Ips grandicollis* (Eichh.)[c/]	20	0	-	-

[a/] Insects collected from emergence traps containing *B. xylophilus* infected wood.

[b/] Wood bolts from previously cut, nematode free trees previously described (Wingfield, 1982).

[c/] Insects trapped associated with *B. xylophilus* infected trees in the field.

Table 3. Distribution of *Bursaphelenchus xylophilus* in three body sections (head, thorax and abdomen) of *Monochamus scutellatus* and *Monochamus mutator*.[a/]

Insect Species	Source	Number of Insects		Mean *B. xylophilus*		
		Examined	With Nematodes	Head	Thorax	Abdomen
M. scutellatus	Austrian Pine Zimmerman, MN	30	26	69	2460	383
M. mutator	Red Pine Black River Falls, WI	8	7	0	4615	96

[a/] Insects collected from emergence traps containing infected wood. Head section includes antennae and thorax includes wings and legs.

Table 4. Transmission of Bursaphelenchus xylophilus by three pairs of Monochamus carolinensis during maturation feeding and oviposition over an eight week period.[a/]

Cage Number	Week Number	Number of Oviposition Niches Examined	Number of Oviposition Niches With B. xylophilus	Number of B. xylophilus in Oviposition Niches[b/]	Number of B. xylophilus in Maturation Feeding Sites (Combined)
I	1	0	-	-	0
	2	0	-	-	0
	3	0	-	-	0
	4	13	7	5.1 (2-11)	53
	5	24	5	3.0 (1-7)	2
	6	20	6	4.8 (1-12)	0
	7	20	0	-	0
	8	29	0	-	0
II	1	0	0	-	0
	2	15	0	-	0
	3	3	0	-	0
	4	9	3	27.3 (8-64)	0
	5	21	0	-	0
	6	23	0	-	0
	7	29	0	-	0
	8	25	0	-	0
III	1	0	-	-	0
	2	0	-	-	0
	3	4	2	29 (22-36)	0
	4	21	-	-	0
	5	31	-	-	0
	6	19	-	-	0
	7	5	-	-	0
	8	0	0	-	0

[a/] Beetles from Austrian pine, Zimmerman, MN allowed to maturation feed on white pine twigs and oviposit in Austrian pine logs.

[b/] Number of nematodes in oviposition niches represent a mean followed by range in parentheses.

Table 5. Numbers of *Bursaphelenchus xylophilus* extracted from *Monochamus carolinensis* after maturation feeding and oviposition for eight weeks.[a/]

Cage Number	Number of *B. xylophilus*	
	Male Beetle	Female Beetle
Cage I	0	33
Cage II	0	14
Cage III	0	0

[a/] Beetles examined were those used in transmission studies presented in Table 4.

Ethephon, a plant growth regulator, stimulates abscission of eastern dwarf mistletoe (*Arceuthobium pusillum*) aerial shoots on black spruce (*Picea mariana*)

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ABSTRACT

Ethephon, an ethylene releasing agent, was sprayed at 1250, 2500, and 5000 ppm on black spruce branches infected with eastern dwarf mistletoe. At the two highest concentrations, the number of seeds and aerial shoots on infected branches were reduced 90-100%. Ethephon treatment resulted in partial dieback of dwarf mistletoe brooms.

Control of dwarf mistletoe (*Arceuthobium* spp.) with chemicals has been restricted to tests using herbicides (F.A. Baker, unpublished PhD Thesis, Univ. of Minn., St. Paul; 7,8). Ethylene releasing agents have not been tested. Ethylene is a growth regulator that promotes abscission of plant parts (6). If ethylene could stimulate dwarf mistletoe seed capsules or shoots to abscise before maturity when the seeds are not viable, a new method of dwarf mistletoe control would be realized. Ethephon ((2-chloroethyl)phosphonic acid, also cited as 2-chloroethane phosphonic acid, CEPA, ETHREL™) is a chemical that releases ethylene within plant tissues and is routinely used in agricultural sciences to promote abscission of leaves and fruit (2). This paper reports the results of ethephon sprayed on eastern dwarf mistletoe (*A. pusillum* Peck) infecting black spruce (*Picea mariana* (Mill.) B.S.P.).

MATERIALS AND METHODS

Three black spruce, 6-20 cm dia 1.4 m above ground, located in Carlton Co., MN, were used for testing ethephon. Four separate brooms bearing dwarf mistletoe seeds were located on each tree. Within each broom, three branch

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segments having the largest number of seed-bearing dwarf mistletoe shoots were tagged and the number of dwarf mistletoe shoots counted. All newly formed dwarf mistletoe shoots (in contrast to the seed bearing shoots which were 1 yr old) were counted on branch segments arising from the tagged branch segments. On 28 August 1981, distilled water (used as a control) and ethephon (ETHREL™, AMCHEM Prod. Inc., Ambler, PA) in distilled water at three concentrations (1250, 2500, 5000 ppm) were sprayed on tagged brooms selected at random on each tree. A hand-held aspirator was used to spray the dwarf mistletoe plants to the point of run-off. After 11 days, the number of 1 yr old and newly formed dwarf mistletoe shoots were counted on each tagged branch. Temperature at the time of treatment was 20°C.

RESULTS AND DISCUSSION

Within 11 days of treatment, ethephon sprayed at 2500 and 5000 ppm stimulated the abscission of most (90-100%) eastern dwarf mistletoe shoots, newly formed and 1 year old (Table 1). Basal cups remained on the stem while dwarf mistletoe shoots and seed capsules had shrivelled and fallen from the branch. Ethephon prevented seed dispersal of eastern dwarf mistletoe on black spruce.

No visible effects of ethephon on black spruce were observed until 1 mo. after spraying. At this time, 2-4 year old needles began to yellow on branches sprayed with ethephon. By June 1982, some of the infected tissue treated with ethephon had died. The percentage of dead buds on the tagged branches were 3.2 - 27% for the control, 11 - 25% for tissue treated with 1250 ppm ethephon, 35 - 60% for 2500 ppm, and 74 - 75% for 5000 ppm. In previous tests (1,3,4,10), similar ethephon treatments did not cause stem death on healthy conifer seedlings grown in the greenhouse nor on seed orchard trees. The treatments induced bud dormancy, decreased shoot elongation, and reduced the number of lateral branches on seedlings. These ethephon mediated effects differ from the effects of dwarf mistletoe infections on conifers, i.e. proliferation and elongation of tree shoots (5,9). The effects of ethephon spraying on dwarf mistletoe infections involves more than abscission of aerial shoots. Additional studies are underway to ascertain how ethephon mediates changes on the growth habit of infected and uninfected host tissue.

Ethephon treatment has potential for preventing seed dispersal of dwarf mistletoe. This would be useful in protecting conifer seedlings from dwarf mistletoe infection that spreads from a seed tree or shelterwood overstory.

ACKNOWLEDGEMENTS

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Table 1. Percent of abscising eastern dwarf mistletoe shoots on black spruce branches sprayed with ethephon.

Tree*	Control		1250 ppm Ethephon		2500 ppm Ethephon		5000 ppm Ethephon	
	1 yr shoots	New shoots	1 yr shoots	New shoots	1 yr shoots	New shoots	1 yr shoots	New shoots
1	44	4	100	75	100	100	100	100
2	0	0	100	0	100	90	100	100
3	9	0	42	5	100	96	100	100

* Counts from three branch segments were pooled for each broom treated and varied from 15 to 274.

Host Specialization in Heterobasidion annosum

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In many root disease centers caused by Heterobasidion annosum (Fomes annosus) in true fir in California, pines are present but apparently unaffected. Possible host specialization among isolates from fir and pine was tested by cross inoculations in the greenhouse.

Some isolates were from the collection of Bob James, and others were freshly collected. Most were from diseased trees, but some were from stumps and may represent stump colonization after felling. In two experiments, a total of 30 isolates were grown on wood wedges and inserted into incisions on the lower stems of 2-0 seedlings (28-30 seedlings of each host per isolate) of white fir and ponderosa pine. Seedlings were harvested when advanced symptoms developed, and experiments were terminated when any isolates killed all of one host. Three controls died in the lst experiment and one in the 2nd.

The ANOVA shows highly significant isolate group-host interaction for both experiments, indicating that the two isolate groups differ significantly in relative virulence on the two hosts. Isolates from one host were more virulent to that host than isolates from the other host. In Experiment 1,fir isolates were almost as virulent as pine isolates to pine, but were significantly more virulent to fir than pine isolates. In Experiment 2, fir isolates were much less virulent than pine isolates to pine and were only slightly more virulent than pine isolates to fir. The higher ambient temperatures of Experiment 2 may have selectively reduced virulence of fir isolates, which in nature cause disease at sites characterized by lower temperatures.

In view of these results, it may be desirable to test replanting of severely infested stands with several species where feasible.

ANOVA

	Expt 1		Expt 2	
	F	P	F	P
Host	4.1	0.01	15.8	0.01
Isolate	4.5	0.05	7.7	0.01
Host x Iso.	9.3	0.01	11.1	0.01

% MORTALITY

	Expt 1		Expt 2	
	Pine	Fir	Pine	Fir
Pine Isolates	80	12	77	17
Fir Isolates	71	48	27	23

Michaels, E. and G. A Chastagner. 1982. Conditions which influence release of Phaeocryptopus gaeumannii ascospores. Western Washington Research and Extension Center, Washington State University, Puyallup, WA 98371.

Swiss needle-cast, caused by Phaeocryptopus gaeumannii (Rohde) Petrak, is common in Douglas-fir Christmas tree plantations throughout western Washington and Oregon. To effectively develop chemical control measures a complete understanding of the infection process is necessary. Investigations were conducted to determine the period of ascospore availability and conditions which influence release of ascospores from pseudothecia. Using infected needles collected from the field, it was found that pseudothecia have the potential to release ascospores from late April through September. Maximum numbers of ascospores were released during June and July. This would agree with work previously done in other parts of the U.S., rather than a 1978 study conducted in western Washington which indicated peak spore release in May and October. Fewer ascospores are available from two-year-old infected needles than from one-year-old infected needles. With a single misting, pseudothecia release all available ascospores within four hours, 75% of these within the first 20 minutes. Ascospore release occurred at temperatures ranging from 5-25°C with an optimum at 20°C. No spores were released at 35°C. Ascospore release was greater when needles were incubated under fluorescent or ultra-violet lighting than under dark incubation. Environmental conditions comparable to laboratory conditions favoring ascospore release would be expected to be present throughout the year in western Washington. Fungicidal control experiments have shown that infection takes place only after bud break during shoot elongation. These findings apparently indicate that changes in host susceptibility rather than inoculum availability determine the infection period.

AN OVERVIEW OF RECENT *FOMES ANNOSUS* (*HETEROBASIDION ANNOSUM*) RESEARCH AT THE UNIVERSITY OF WASHINGTON

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Recent research has focussed on (1) chemical stump treatment control, (2) patterns of deposition and release of airborne spores, (3) effects of *H. annosum* on physical, chemical and pulping properties of western hemlock wood, and (4) variation in decay rates of western U.S. isolates of *H. annosum*.

We have determined that; (1) typical agricultural fungicides (Ridomil, Benlate, Captan, Ferbam, Thiram, and Daconil) are ineffective against *H. annosum* and are not as good as $ZnCl_2$ or borax. We are continuing to test other chemicals including wood preservatives and more soluble forms of borax; (2) spore deposition peaks do not occur at any specific hour of the day or night. Seasonally greatest numbers of spores are deposited in the fall with lowest numbers in late spring or summer. Highest values were 38,074 spores m^{-2} h^{-1} at a Cascade Mountain site in Washington and 36,260 spores m^{-2} h^{-1} at a coastal Washington site. Using a Gelman filter sampler and a scanning electron microscope we determined that 20% of the spores deposited at the coastal Washington site in October were conidia and 80% were basidiospores. Regional spore deposition patterns in coastal Oregon and Washington indicate that more spores were deposited in precommercially thinned stands than commercially thinned stands; (3) there is little effect at the incipient decay stage on wood physical properties (Modulus of Rupture and Modulus of Elasticity), but there is an effect at the advanced decay stage. However, total pulp yield is reduced by 2.5% using incipiently decayed wood and is reduced by 8% by using advanced decayed wood; and (4) soil block decay tests have indicated variability of western U. S. isolates of *H. annosum* to decay western hemlock wood.

WHITE-POCKET ROT BY PHELLINUS PINI IN CONIFERS

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Phellinus (Fomes) pini (Thore ex Fr.) A. Ames. is one of the most destructive decay causing organisms of living trees. Decay caused by P. pini is typically a white-pocket rot. This type of decay is characterized by spindle-shaped zones of white fibers surrounded by apparently sound wood. A three dimensional view of decayed wood, obtained from a composite of micrographs using scanning electron microscopy and histological techniques, is present to demonstrate why P. pini causes a white-pocket rot.

White-pocket material from black spruce, Douglas-fir, jack pine, Western larch, and Western white pine indicate:

1. White pockets contain delignified tracheids (3.1% lignin and 78.0% total sugars whereas wood surrounding the white pockets contained 29.6% lignin and 66.9% total sugars).
2. Latewood tracheids are preferentially degraded.
3. The middle lamella between tracheids is completely decomposed.
4. Cell walls are delignified revealing the macrofibrillar structure of cellulose in the remaining cell wall.
5. Ray parenchyma cells are completely removed.

Wood between the white pockets was nondecayed. The morphological and chemical barriers to delignification consist of:

1. Earlywood tracheids
2. Resin ducts
3. Resin-filled ray parenchyma and tracheids

Investigations involving P. pini, one of the most common and devastating heart rot organisms, will not only help to elucidate the decay process within living trees but will also provide needed information concerning biological delignification which could be of great economic importance to man.

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FORMATION OF WHITE POCKETS IN OAK CAUSED BY *Inonotus* (*Polyporus*) *dryophilus*

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INTRODUCTION

Inonotus (*Polyporus*) *dryophilus* causes a white pocket rot in the heartwood of living oaks. White pocket decay is characterized by the presence of apparently sound wood existing between localized areas or pockets of degradation. Within the pockets *I. dryophilus* was found to selectively remove lignin leaving only the white cellulosic macrofibrils of the cell walls. Chemical analyses have shown these delignified tissues to be composed of 93.5% total sugars and 2.6% lignin, whereas sound oak heartwood had 64.5% total sugars and 25.0% lignin (Otjen, 1982). Organisms with the ability to selectively remove lignin from wood are of potential importance in application to biopulping processes. Other potential uses include the cleanup of pulping mill effluent, biobleaching of wood pulp, or releasing wood sugars for ethanol production, or use as a carbohydrate for ruminant animals. Before white pocket rot fungi could ever be used for such purposes it is important to determine what restricts them from delignifying all of the wood uniformly. The major objective of this study was to elucidate the factors responsible for the formation of white pockets by *I. dryophilus* in the heartwood of living oaks.

MATERIALS AND METHODS

Four bur oaks (*Quercus macrocarpa* Michx.) and one white oak (*Quercus alba* L.) approximately 50-75 years old with conks of *I. dryophilus* were cut in Anoka and Dakota counties, Minnesota. Trees were cut into 30 cm bolts above and below the sporophore beyond any evident discoloration or decay.

Radial and tangential sections of wood were cut from all representative areas of affected wood. Unfixed specimens were dried in a dissicator, and coated with 40% gold and 60% paladium in a Kinney KSE-2A-M vacuum evaporator. Specimens were observed and photographed with a Philips scanning electron microscope at 12 kV.

Radial, tangential, and transverse sections 13-15 µm thick, were obtained using a Tissue Tek II Cryostat cooled to -20°C. Sections were stained with safranin-fast green.

RESULTS

Selective delignification occurred in axial parenchyma cells surrounding vessels of earlywood and latewood. Flame-shaped tracts of vessels with accompanying axial parenchyma, present throughout the latewood, provided avenues for radial movement of *I. dryophilus*. Dense groups of latewood fibers were not degraded. *Inonotus dryophilus* did not delignify ray parenchyma, instead, a typical white rot occurred, differentiated microscopically by a shot-hole appearance. Tyloses did not restrict *I. dryophilus* movement in heartwood vessels of living oaks. Occluded latewood fibers and medullary rays were often left intact forming borders between white pockets. The reason *I. dryophilus* causes a white pocket rot in oak is clearly related to the anatomy of its host.

REFERENCE

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Histopathology of *Verticicladiella wageneriі* in Douglas-fir

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INTRODUCTION

Verticicladiella (*Ceratocystis*) *wageneriі* Kendrick causes a vascular wilt disease of Douglas-fir *Pseudotsuga menziesii* and other western conifers. Foliar symptoms are similar to those caused by other root pathogens of Douglas-fir. There is a progressive reduction in the length of needles and internodes, followed by chlorosis, loss of older needles, and distress cone crops (Fig. 1). Finally the needles turn orange-red and fall to the ground (Fig. 2). New infections are presumably initiated in healthy roots either by insect vectors or by root to root transmission, but infection courts have not been experimentally demonstrated. Colonization of the host is accomplished solely by hyphal growth, and branching is restricted to sapwood tracheids (Phytopathology 57: 935-938, PDR 45: 831-835). This work was designed to examine histologically the colonization biology of *V. wageneriі* in Douglas-fir roots to aid in characterizing infection sites and the infection process.

BASIC PATTERN OF SECONDARY XYLEM COLONIZATION

V. wageneriі imparts a dark amber to olive-brown stain to infected Douglas-fir sapwood. Factors contributing to this "black-stain" include:

1. melanized hyphal walls,
2. amber-brown hyphal sheath,
3. discolored tracheid walls adjacent to hyphae,
4. gum-plugging of ray tracheids,
5. infrequent tyloses,
6. resin soaking of colonized xylem

Hyphae in longitudinal (axial) tracheids are serpentine to helicoid and contact many border pit-pairs on radial walls. Sequential invasion of tracheids longitudinally is through bordered pit-pairs on their tapered ends. Hyphae reduce their diameter when passing through bordered pits. The torus is pushed out of position by the hyphae and is left partially attached to the margo. Bordered pits are completely occluded by passage of hyphae. Earlywood tends to be preferentially invaded over latewood probably due to the larger lumen and pit-aperture sizes. When colonized, latewood tracheids tend to be more intensively ramified by comparison since the last formed axial tracheids of the latewood have bordered pits on both radial and tangential walls.

RADIAL AND CIRCUMFERENTIAL COLONIZATION

Lateral (circumferential) colonization occurs via bordered pits on radial walls of longitudinal tracheids. Radial colonization happens in one of three ways:

1. Along ray tracheids,

2. Through bordered pit-pairs on tangential walls of the 4-10 last formed longitudinal tracheids, or

3. During circumferential growth between laterally adjacent pairs of axial tracheids.

The vertical declination of a radial file of longitudinal tracheids produced by the same fusiform initial is roughly constant. The axes of two laterally adjacent tracheids produced by adjacent fusiform initials may be different. It is this difference that accounts for most of the radial growth of V. wagenerii in Douglas-fir.

Hyphal development in ray tracheids is infrequent and appears to be limited by small lumen and pit-aperture sizes. Frequently uniseriate and fusiform (those containing resin canals) rays act as an impediment to circumferential development of the fungus. Three reasons are important:

1. Most ray cells are parenchymatous,

2. V. wagenerii hyphae do not penetrate the piceoid simple pit-pairs or half bordered pits of the ray cross-fields, and

3. Most ray tracheids occupy terminal or near terminal positions in the ray.

IMPLICATIONS OF COLONIZATION BIOLOGY

The rate of radial colonization in vivo is slow and is determined by the rate of longitudinal colonization. Observations of growth rate of the fungus within the optimum range of temperature for growth indicate that in seedlings, axial colonization occurs at the rate of 2.0-2.5 mm/day. Circumferential and radial colonization proceeds at rates of 0.15-0.25 mm/day and 0.02-0.03 mm/day, respectively. These factors help to account for the characterisitc crescent-shaped pattern of staining that so contrasts V. wagenerii with other sapstaining members of its telomorphic genus Ceratocystis.

Field Trip Notes

September 15, 1982

A fine, cool, rainy day was used to entice members to observe pathological problems in the Lake Tahoe area. Several fine examples of sick trees were viewed and grand explanations of past and future problems given. Great examples of Fomes root rot, western gall rust, stalactiform rust, Elytroderma needle cast, salt damage, bug damage and dwarf mistletoes were found.

30th WIFDWC Dwarf Mistletoe field Trip--
Or how to eat your way across the State.

Bob Scharpf

Seventeen enthusiastic WIFDWC members and guests gathered at the Institute of Forest Genetics at noon on Saturday, September 11 for the beginning of the dwarf Mistletoe field trip. Our first order of business was to consume our bag lunches including various pot luck dishes and a freshly baked chocolate cake provided by Carol Alosi--all washed down with an abundant supply of beer and soft drinks provided by local arrangements.

After our multicourse feast, those who were able to get on their feet walked to the southeast corner of the institute grounds to look at 40 year old Jeffrey pines naturally infected by western dwarf mistletoe. Differences in resistance among several altitudinal collections of Jeffrey pines were observed and discussed. By this time, lunch was partially digested, we had finished our discussion, and all participants managed to make it back up the hill and to our vehicles for the next stop on the trip.

At Camino, a few miles east of the institute, we stopped to look at a series of recent inoculations in a young plantation to test the resistance of different seed sources of Jeffrey pines to dwarf mistletoe. These controlled inoculations were made as a follow up to the naturally infected Jeffrey pines observed at the institute. Lunch was nearly digested so we headed east on highway 50 toward the pass (Echo summit) to see mature red fir stands infested with dwarf mistletoe and Cytospora canker. At Sierra Ski Ranch near the summit, Dick Parmeter showed the group large trunk swellings caused by dwarf mistletoe and set up a demonstration with dissected portions of trunk swellings. Enthusiasm reined for almost an hour until the group suddenly realized the sun was low in the sky and they were getting hungry, and most of all thirsty again. With little dissention in the ranks, we headed for our prearranged lodging in South Lake Tahoe.

On Sunday morning (I will dispense with any descriptions of the nights activities) we gathered our forces, including our wounded-like (Terry Shaw), and proceeded on to Nevada Beach Campground where we were met by several more WIFDWC ers who were either too poor (or were not on expenses) to stay in our Motel. At Nevada Beach, Det Vogler discussed the project he had underway there, involving the problems of dwarf mistletoe in a high use recreational area. By mid morning, Terry was feeling much better so we headed toward our next stop, Truckee, via the beautiful east shore of Lake Tahoe. By the time we arrived at Truckee the group was hungry again, so we stormed the towns' restaurants for lunches that ranged from dining in an elegant old Victorian house to grabbing a beer and pizza in one of the local saloons.

With our energy and enthusiasm back to par we headed north of Truckee on hwy. 89 to see and discuss control of dwarf mistletoe in young true firs by thinning. A dusty 7 mile side road off 89 did not deter our determination because we knew that this was the last stop of the trip. After we absorbed all knowledge about fir dwarf mistletoe, the group proceeded back along the west shore of Lake Tahoe to the WIFDWC Conference Center at Fallen Leaf Lake just in time for the evening happy hour and dinner. A good time was had by all.

WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE
MISTLETOE COMMITTEE REPORT
1982 HIGHLIGHTS

John G. Laut, Chairman

I Taxonomy, Hosts and Distribution

A. A certain dwarf mistletoe monograph listed corkbark fir as a secondary host for Arceuthobium douglasii. However, more detailed examinations of many mixed Douglas-fir/corkbark fir stands in Arizona and New Mexico have shown that corkbark fir is less susceptible and should more appropriately be classed as an "occasional" host. Not only are fewer corkbark firs infected in mixed stands but infection intensity is consistently lower in corkbark than in associated Douglas-fir. A manuscript has been prepared on the infection of southwestern true firs (white fir, corkbark fir, subalpine fir) to dwarf mistletoe for publication as a research note by Northern Arizona University. (R. L. Mathiasen, NAU; F. G. Hawksworth, RM Sta.)

B. Arceuthobium divaricatum was collected for the first time (on Pinus edulis) in the Guadalupe Mountains in southern New Mexico. This locality fills a gap of about 100 miles in the previously known range of pinyon dwarf mistletoe. Additional pinyon hosts for A. divaricatum not listed in our 1972 monograph are Pinus discolor in New Mexico and P. edulis var. fallax in Arizona. (F. G. Hawksworth, RM Sta.)

II Physiology and Anatomy

A. Studies of the isozymes of Arceuthobium were continued. Preliminary work with several species collected in 1981 identified some 100 enzyme systems, of which about 12 will be used for comparison among taxa. Objectives of the study are (1) to determine correlations between "niche width" among taxa (e.g., comparison of dwarf mistletoes that have brood host ranges with those that have narrow host ranges) and (2) to determine the potential of isozyme analyses in defining classification and evolution of the genus Arceuthobium. Most Mexican and United States taxa will be studied. (Don Nickrent, Miami Univ., Ohio and F. G. Hawksworth, RM Sta.)

B. Electrical resistance (ER) between steel needle electrodes forced through bark into outermost xylem was measured by a Shigometer in lodgepole pines (Pinus contorta) infected by dwarf mistletoe (Arceuthobium americanum) in the greenhouse and field. In the greenhouse, seedlings with bole infections were measured. ER was lowest at the infection, intermediate below, and highest above the infection. In the field, ER of 100-year-old trees of different dwarf mistletoe intensity and vigor ratings was measured at breast height. Trees of poor vigor or those with high mistletoe ratings (Class 6) had significantly elevated ER. From manuscript accepted for Forest Science. (B. Schaffer, Colo. State Univ.; F. G. Hawksworth, RM Sta.)

C. Cytokinins were extracted and assayed from aerial shoots of the dwarf mistletoe Arceuthobium vaginatum subsp. cryptopodum (Engelm.) Hawksworth ahd Wiens, which induces witches broom formation on infected trees. Cytokinins were also extracted from ponderosa pine tissue infected with the parasite, and healthy ponderosa pine tissue. Dwarf mistletoe shoots contained the highest concentration of cytokinins. The same cytokinins were present in the infected ponderosa pine tissue but in much lower concentrations. Cytokinins were not detected in healthy ponderosa pine bark and wood tissues. Cytokinins were also extracted and assayed from aerial shoots of Arceuthobium occidentale Engelm., a parasite of digger pine which does not induce witches broom formation. Although cytokinins were present in the shoots of this dwarf mistletoe species, they were different from those found in Arceuthobium vaginatum. These results suggest that cytokinins may be involved in the induction of witches brooms by dwarf mistletoes. From manuscript accepted by Forest Science. (B. Schaffer, Colo. State Univ.; F. G. Hawksworth, RM Sta.)

D. Seeds of dwarf mistletoe parasites, A. americanum and A. cyanocarpum, avoid freezing by deep undercooling all freezable tissue water to near -35C. Seeds nucleated with ice at -3C and cooled to -40C were killed. Seeds cooled to -30C had germination levels equivalent to nonfrozen seeds. Differential Thermal Analysis studies showed that the position and size of the exotherms of the seeds were not significantly changed by the following treatments: absence or presence of external ice-nucleation at -3C, ice-nucleation at -3C followed by holding at -17C for 48h prior to subsequent cooling, and changes in cooling rate between 0.1 and 1.0C/min. Thus, the deep undercooling mechanism is persistent and the imposed hardiness limit of the mistletoe seeds overwintering on the host may be a factor in restricting the distribution of the mistletoe parasite relative to the distribution of the host tree. (M. R. Becwar, F. G. Hawksworth, M. J. Burke, Colo. State Univ. and RM Sta.)

III Life Cycle Studies

A. The manuscript entitled "Population dynamics of dwarf mistletoes in young true firs in the central Sierra Nevada" reported as in press at the last WIFDWC is still in press. The USDA moratorium on publications has recently been lifted and we anticipate being in print soon. (R. F. Scharpf, PSW; J. R. Parmeter Jr., UC)

B. A preliminary study is being made to identify possible bird and mammal vectors of lodgepole pine dwarf mistletoe seeds. The study is being conducted at the Fraser Experimental Forest, Colorado. (T. Nicholls, NC Station; F. G. Hawksworth and L. M. Merrill, RM Sta.)

IV Host-Parasite Relations

A. Initial intensification and lateral spread of Arceuthobium laricis were measured for three levels of stocking, 1680, 549, and 272 stems/ha (2.4x2.4, 4.3x4.3, and 6.0x6.0 m spacing) of young, single-layer, western larch monoculture, following artificial inoculation.

Intensification was slow for the first six years following inoculation, but showed fivefold to tenfold increases in treatment means for the next three-year period. Lateral spread from the inoculated trees to neighboring suscepts was not observed during the first six years. It was observed in all three treatments between years seven to nine and averaged 3.5 to 4.9 m for the three spacing treatments. Maximum lateral spread during the period was 6.4 m. We conclude that A. laricis can intensify rapidly on young western larch once an inoculum source is established. The lowest stocking level (highest spacing level, 6x6 m) used in this study did not prevent lateral spread of the pathogen. These data attest to the value of prevention or early detection and removal of infections for the protection of western larch stands from subsequent losses. From manuscript being revised for submission to Can. J. For. Res. (E. F. Wicker and J. M. Wells, RM Sta.)

V Effects on Host

A. Dwarf mistletoe-related mortality of ponderosa and Jeffrey pines is being followed at five campgrounds in California and Nevada. A report of mortality after eight years was published in 1981, and showed that the rate of mortality increased exponentially as the infection rating increased. Further data on mortality and mistletoe intensification will be collected through 1984. The final report will use these data to predict losses in mistletoe-infected campgrounds. (D. R. Vogler, FPM, Region 5; R. F. Scharpf, PSW)

B. A manuscript entitled "Stem infection by dwarf mistletoe in California firs" is in press. The manuscript summarizes the importance and frequency of "trunk" infection by dwarf mistletoe in firs. (J. R. Parmeter Jr., UC; R. F. Scharpf, PSW)

C. Stem analysis data from approximately 200 western hemlock 40-150 years old are being analyzed to determine effects of dwarf mistletoe infection on volume increment. In addition, 26 25-year-old trees have been dissected to examine effects of size and distribution of infections and tree competition on tree growth rate. (A. J. Thomson, R. B. Smith and R. Alfaro, PFRC Victoria)

D. Ten-year readings are being taken in summer, 82 on the effect of dwarf mistletoe on released red firs in California. A summary will be prepared on growth after release, mortality as related to dwarf mistletoe, and buildup of the parasite over time. (R. F. Scharpf, PSW)

E. Fieldwork has been completed and data analyses are well underway to develop a yield simulation program for mistletoe-infested, mixed conifer stands in the Southwest. Data from 70,000 trees from 387 plots in Arizona and New Mexico will provide the basis for the new program. The model will be able to project yields in uninfested stands and those infested by A. douglasii, A. microcarpum and A. vaginatum in various combinations. (R. L. Mathiasen, Nor. Ariz. Univ.; C. B. Edminster and F. G. Hawksworth, RM Sta.; E. Wood, FPM, Region 3)

VI Ecology

A. A study was continued to determine correlations between dwarf mistletoe (A. vaginatum and A. douglasii) frequency and abundance with habitat types in Colorado. Some 940 plots have been established. The results will be analyzed this winter and presented in a M.S. thesis at Colorado State Univeristy. (L. M. Merrill and F. G. Hawksworth, RM Sta.; D. Johnson, FPM, Region 2)

VII Control - Chemical

A. Chemical control tests on A. vaginatum on ponderosa pine were continued near Estes Park, Colorado. Several chemicals look promising as they prevent seed formation and selectively kill mistletoe shoots but the effects on the endophytic system are not yet certain. Spring spraying is most effective. Chemicals that look best to date are: Butyrac ester and Emulsamine (Union Carbide); D-40 and Esteron 99C (Dow); RoundUp (Monsanto); Dacamine (Diamond Shamrock); RP 2,4-D and MCPA (Rhone Poulene); and Acti-Aide (Upjohn). (A. Moinat, Estes Park, Colo.)

VIII Control - Biological

A. Spittlebugs (presumably Clasdoptera distincta) were very common on Arceuthobium vaginatum on the Mescalero Apache Reservation in southern New Mexico in 1982. (F. G. Hawksworth, RM Sta.)

B. A manuscript on the "Mistletoe Rust" Peridermium bethelii associated with Arceuthobium americanum on lodgepole pine has been submitted to Plant Disease. The life cycle of this rust is still not known although it is apparently autoecious. It resembles Cronartium comandrae but has distinct spore morphology, sporulation period, and will not infect Comandra. (F. G. Hawksworth and C. Dixon, RM Sta.; R. G. Krebill, Int. Sta.)

IX Control - Silvicultural

A. Treated over 6,900 acres of A. americanum infested lodgepole pine stands on the Arapaho and Roosevelt, Grand Mesa, Uncompahgre, and Gunnison, Pike and San Isabel, Shoshone, and White River National Forests and State-owned sections in Wyoming. (D. W. Johnson, USFS, Region 2)

B. Overstory removal of A. americanum from 330 acres on the Caribou National Forest and 2,967 acres on the Targhee National Forest. Control treatment of A. douglasii on 486 infected acres on the Sawtooth National Forest and 364 acres on the Boise National Forest. (Hoffman, USFS, Region 4)

C. Using the western hemlock dwarf mistletoe model, DWARF, infection severity is being estimated over a range of stand and site conditions and various silvicultural regimes. The results will form the basis for silvicultural guidelines. (W. J. Bloomberg, PFRC Victoria)

D. J. Laut under contract to the B.C. Ministry of Forests Protection Branch is directing Ministry field crews to collect data for testing and adopting the LPMIST mistletoe evaluation technique to B.C. conditions. Approximately 150 plots were established, and data should be analyzed by December 1982. Hopefully, a B.C. version of the technique to make site-specific prescriptions and regional loss assessment surveys will be available in 1983. (Muir, B.C. M. of F.)

E. Field establishment and data collection for the California Region-PSW Cooperative Project to test the efficacy of controlling dwarf mistletoe in true firs by pre-commercial thinning was completed in 1982, following an early winter which prevented completion in 1981. Data verification and analysis will be undertaken in 1982-83 and a progress summary prepared. (R. F. Scharpf, PSW; R. S. Smith and D. Vogler, Region 5)

F. Reexamination of long-term (30- to 37-year-old) ponderosa pine dwarf mistletoe control plots were made on the Mescalero Apache Reservation in New Mexico and at Grand Canyon National Park in Arizona. The results, which will be analyzed this winter, will be used to help evaluate the RMYLD growth simulation model for even-aged stands and also to provide data for a new yield program for uneven-aged stands. (F. G. Hawksworth, L. M. Merrill and T. E. Hinds, RM Sta.; E. Wood, FPM, Region 3)

X Surveys

A. In 1981, a survey of dwarf mistletoes (A. laricis, A. americanum) was undertaken in five districts of the Nelson Region in southeastern British Columbia. Ministry of Forests personnel selected a problem site in each district. Each site was inspected with M.O.F. personnel including A. Renwick, Pest Management Coordinator for the Nelson Region. A survey procedure was selected and conducted at each site.

In three areas we found both mistletoe species; in one, A. americanum and in one A. laricis. Two young stands and three older stands were surveyed. A workshop to discuss mistletoe identification and control options was held at each site with local government and industry personnel who are concerned with mistletoe control. Field data and a technical report were prepared for the Ministry. (Muir, B.C. M. of F.)

B. Biological evaluation of 16,013 acres of proposed dwarf mistletoe control projects on the Payette National Forest. The disease-causing organisms include A. americanum, A. douglasii, A. campylopodum, and A. laricis. (Hoffman, USFS, Region 4)

C. Data from approximately 400 western hemlock 1/50 acre plots in seven Vancouver Island and Gulf Island locations previously cruised for dwarf mistletoe severity ratings are being analyzed to obtain estimates of ratings on a stand basis by tree size class. Rating data will be correlated with volume impact data obtained from felled sample trees in each plot in order to estimate volume impact on a stand basis. (R. Alfaro and A. J. Thomson, PFRC Victoria)

D. Presuppression surveys for A. americanum were conducted on 52,000 acres on the Arapaho and Roosevelt, Medicine Bow, Pike and San Isabel, Shoshone and White River National Forests and State-owned sections in Wyoming. (D. W. Johnson, USFS, Region 2)

E. Dwarf mistletoe loss assessment survey -- ponderosa pine and Douglas-fir -- Colorado National Forests. As part of a continuing effort to assess growth loss and mortality caused by Arceuthobium species in the Rocky Mountain Region, a road-plot survey initiated during the 1981 field season was continued in 1982. A report will be issued in 1983. (D. W. Johnson and F. G. Hawksworth, USFS, Region 2 and RM Sta.)

F. An evaluation of the roadside survey technique has been made for ponderosa pine dwarf mistletoe on three National Forests in Colorado. Comparisons were made between stands with various intensities of dwarf mistletoe along 36 one-mile strips of roadside and transects parallel to the roads. Results are not yet available. (L. M. Merrill and F. G. Hawksworth, RM Sta.; D. W. Johnson, FPM, Region 2)

XI Miscellaneous

A. Ministry policy statements, and guidelines for Regional and Provincial use have been drafted, circulated for comment, and, in the Cariboo, implemented.

Additional guidelines for mistletoe treatments after logging and in stand tending (thinning) were drafted for the Provincial Silviculture Manual.

A workshop in cooperation with the Canadian Forestry Service, P.F.R.C. is planned to outline impact data, survey methods, and control options for coastal B.C. hemlock mistletoe. (Muir, B.C. M. of F.)

B. The study that was begun at the Institute of Forest Genetics in 1980 to test the resistance of Jeffrey pines to infection by A. campylopodum was expanded in 1981. Because of heavy seed loss from branches in spring of 1981, an additional 4,500 inoculations were made in the fall. Bird and bug exclusion bags were tested to reduce seed loss. Data will be collected in 1982. (R. F. Scharpf, Bro Kinloch and Jim Jenkinson, PSW)

C. Two manuscripts have been submitted for publication that are indirectly related to dwarf mistletoe. They involve cytospora abietis, a common associate of dwarf mistletoe-infected branches of firs. The manuscripts are (a) Temperature-influenced growth and pothogenicity of C. abietis on white fir, and (b) Growth of C. abietis on dwarf mistletoe-infected and uninfected branch tissues of red fir. (R. F. Scharpf, PSW)

D. Bruce Schaffer completed his Masters thesis on "Effects of dwarf mistletoe and Comandra rust on cone and seed production of Lodgepole pine" at Colorado State University. A manuscript summarizing the results has been accepted for publication in Plant Disease.

XII Late Reports (Continued)

V. Effects on Hosts

F. We are attempting to simulate effects of A. americanum in stands of jack pine in Manitoba, based on information from long term plot studies and from transect studies. An abstract of these studies appeared in Phytopathology 72:979. (F. Baker and D. W. French, University of Minnesota.)

G. The manuscript on our black spruce dwarf mistletoe simulator has appeared in Forest Science 28:590-598. (F. Baker and D. W. French, University of Minnesota.)

H. Height and radial growth data were collected from 103 felled sawtimber size grand fir in three stands severely infected with Arceuthobium abietinum f. sp. concoloris in central Oregon. Trees with high live crown ratio (LCR) and low DMR grew fastest over the last 25 years, while trees with low LCR and high DMR grew the slowest; LCR was the more important factor. From a sample of 673 branches with dwarf mistletoe, it was determined that mean age when a branch became infected was 8 years and it took an average of 11 years of parasitism for infections to kill a branch. About 9 percent of the sampled branches also were infected by Cytospora abietis. This information was presented in a Forest Pest Management office report and will be submitted to a journal for publication. (G. Filip, FPM-Portland.)

VII Control-Chemical

A. An ethylene releasing agent, ethephon, promoted the abscission of all Arceuthobium pusillum shoots on black spruce. Refer to the special paper in these proceedings for additional information. (W. H. Livingston, M. L. Brenner, University of Minnesota.)

X Surveys

Black and white aerial photography (scale 1:15, 840) for one township in the Koochiching State Forest was interpreted to locate mortality centers and determine basal area losses caused by eastern dwarf mistletoe (Arceuthobium pusillum Peck). Due to difficult access, suspected mortality centers were checked by helicopter. Seven of the 32 stands checked were infested with A. pusillum, of which five were correctly identified and two were not identified from the aerial photos. Of the total area checked, 89 acres were out of production, and 85 of the 89 acres were in one stand. Basal area lost was 5% of the total basal area. (P. Scherman, W. H. Livingston, University of Minnesota; M. Albers, G. Hecht, Minnesota DNR.)

XII Late Reports

I. Taxonomy, Hosts and Distribution

C. Efforts are underway to eradicate A. americanum from the southeastern part of its range in Canada. The infestation near Milner Ridge, Manitoba was harvested during the winter of 1981-82, and operations were started near Wallace Lake, Manitoba. In August we did an aerial survey of these areas and an area in Ontario where A. americanum was eradicated by wild fire in 1976. Arceutbobium americanum was not observed in Ontario, but the parasite was found on three trees approximately 0.5 K from the eradicated center at Milner Ridge. These trees will be killed. (F. Baker and D. W. French, University of Minnesota.)

II. Physiology and Anatomy

E. High performance liquid chromatography is being used to purify black spruce (Picea mariana) tissue infected with eastern dwarf mistletoe (Arceuthobium pusillum) for plant growth substance analysis. Infected and uninfected tissue is being analyzed for abscisic acid (ABA), indole-3-acetic acid (IAA), zeatin, and zeatin ribozide. For tissue collected in April and May, 1982, major differences in compound amounts are associated only with ABA. Infected tissue has only 1/2 the level of ABA (350 ng/gdw) that uninfected tissue has (600-880 ng/gdw) - (W. H. Livingston, M. L. Brenner, University of Minnesota).

F. Arceuthobium pusillum seed was collected in September from naturally infected black spruce by wrapping cheese cloth around the brooms. Seed caught by the cheese cloth was removed, sterilized in 3% hydrogren peroxide, and suspended on discs in jars over a saturated NaCl solution. Jars were stored at -10°C and +4°C. Starting two weeks after collection and continuing at biweekly and monthly intervals, 100 seeds were removed from jars at each storage temperature and placed in a growth chamber at 15°C, 18 hr light period. Seeds were moistened three times a week. Maximum germination (the appearance of the red radicle) was ca. 60% and did not differ between storage periods but was slightly larger for seeds stored at -10°C. However, radicle growth did not exceed 1 mm unless seeds received at least 10 weeks of cold storage. Arceuthobium pusillum seeds treated as described above successfully infected black spruce seedlings in the greenhouse. (W. H. Livingston, R. A. Blanchette, University of Minnesota.)

G. We continue to follow the development of A. pusillum on black spruce and larch branches inoculated in 1975, 1976, and 1977. To date, 8 infections have appeared, all on spruce. (F. Baker and D. W. French, University of Minnesota.)

Highlights of the 1982 meeting

Greg Filip, Chairman

The following summaries of work concerning root diseases were submitted:

1. Armillaria Root Rot

   a. Paired, 1/4-acre plots have been established in five different areas in Oregon and Washington to determine the long-term effects of precommercial thinning on crop tree mortality caused by *A. mellea*.

3 plots in Douglas-fir:	two are 4 years old. one is a year old.
1 plot in Shasta red fir:	2 years old.
1 plot in ponderosa pine:	14 years old.

   As yet, only the 14-year-old plot shows a significant decrease in mortality of crop trees in thinned plots.

   (Filip, FPM. R-6)

   b. Several limited field inspections in mixed spruce-subalpine fir stands in British Columbia indicated that Armillaria root disease and balsam bark beetle were associated with most dying trees.

   (Muir, BCFS)

   c. Intensive survey of 590-600 acres on Santa Fe NF found *A. mellea* had caused loss through mortality of 10 percent (1,000 BF) of residual overstory stand since the area was logged 8 years ago. Damage to the understory was less.

   (Wood, FPM, R-3)

   d. A Regionwide root disease survey of 2.8 million acres found that root diseases (mostly Armillaria) were found in 30 percent of trees and 30 percent of volume, usually in complex with bark beetles and/or dwarf mistletoe. Root disease caused more damage in mixed conifer and spruce-fir types than in pine types. All mortality in spruce-fir type was root rot associated.

   (Wood, FPM, R-3)

   e. Several Forests have found Armillaria causing serious problems in regeneration areas, usually associated with poor planting technique.

   (Wood, FPM, R-3)

   f. Five intersterile groups were found among 40 collections from the southern half of British Columbia.

   (Morrison, PFRC)

g. In 1982, 363,206 acres of spruce-fir type on the San Juan National Forest were surveyed for root disease-caused tree mortality. Approximately 23 percent of the area was occupied by root disease (not area out of production).

Root disease (mostly *A. mellea*) alone accounted for only 1.3 percent of the mortality, bark beetles (mostly western balsam bark beetle) alone caused 35.1 percent, and the combination of root disease and bark beetles caused 63.5 percent. Mortality was almost exclusively in subalpine fir.

(Fuller, FPM, R-2)

h. The influence of *A. mellea* on *H. annosum* is being examined.

(Edmonds, UW)

2. Annosus Root Rot

a. *F. annosus* was found killing trees throughout the central Oregon Plateau in young lodgepole pine plantations. Of 19 stands surveyed, one has had 3 percent of its total area in production reduced by *F. annosus*. Ten other stands had 0.66 percent to less than 0.001 percent of their area affected by *F. annosus*.

(Schmitt, FPM, R-6)

b. Substantial amounts of white fir mortality associated with *F. annosus*, fir engravers, and drought have been found on the Fremont National Forest. A Forest-wide survey of the white fir type is proposed for 1983.

(Goheen, FPM, R-6)

c. Stumps in 20 stands were assayed for incidence and area of *F. annosus* infection. Equal numbers of stumps were sampled in each diameter class. Incidence and area were greatest in large stumps. Decreasing species susceptibility was *Abies*, hemlock, spruce (Sitka), and Douglas-fir. No *F. annosus* was found in lodgepole pine.

(Morrison, PFRC)

d. Data collection for calculation of *F. annosus* impacts in California firs has been completed. Loss estimates whill be converted to impact. Impact survey in campground firs is underway.

(Parmeter, UC Berkeley)

e. Greenhouse cross-inoculation of *F. annosus* isolates from pine to fir indicate host preferences in the two isolate groups.

(Worrall, UC Berkeley)

f. A study of conditions in wetwood and their effects on *F. annosus* indicate that wetwood may be inhibitory to fungal growth and decay.

(Parmeter, UC Berkeley)

g. Airborne spore loads of *H. annosum* in precommercially and commercially thinned stands of western hemlock in Oregon and Washington are being studied. Also, diurnal and seasonal spore deposition and release patterns as well as proportions of conidia to basidiospores in the air are being investigated. Genetic diversity of *H. annosum* with respect to decay rates of hemlock wood is being tested with soil blocks.

(Edmonds, UW)

h. The following chemicals are being tested to control stump infection by *H. annosum*: Ridomil, benlate, $ZnCl_2$, dithane-M45, captan, ferbam, thiram, borax, daconil, lauricidin, pentachlorophenol, ACA, CCA, PQ-10, permatox, sodium metaborate, borateem, and grandstand 9E.

(Edmonds, UW)

i. The impact of *H. annosum* on physical (modulus of rupture and elasticity and specific gravity) and chemical (soda solubility, pulp yield, and paper quality) properties of western hemlock wood are being studied.

(Edmonds, UW)

j. Methods to prevent introduction into and to remove existing *F. annosus* inoculum from ponderosa pine stands in selected areas is being evaluated on the Payette National Forest.

(Williams, FPM, R-4)

3. Black Stain Root Disease

a. Bloomberg survey technique was successfully adapted and tested in lodgepole pine stands with black stain and black stain plus Armillaria.

(Morrison, PFRC)

b. Results are being analyzed from several thinned and unthinned Douglas-fir plantations for incidence of black stain on the Siskiyou National Forest and the Coos Bay District, BLM.

(Goheen, FPM, R-6)

4. Laminated Root Rot

a. Survey of over 1,300 acres of grand fir-Douglas-fir type on the Mt. Hood National Forest revealed over 35 percent of the area to be infected by *P. weirii*. A modified Bloomberg technique was used.

(Filip, FPM, R-6)

b. Field observations of *P. weirii* in 120- 140-year-old Douglas-fir stands near Merritt, B.C. indicated that low-level aerial surveys should be feasible. Stands or infested areas could be sketch-mapped or designated on aerial photographs as light, medium, or heavy incidence. Field work and plans to develop the technique are underway. The Ministry supported a cooperative study with G. Wallis to determine the suitability of 70 mm aerial photography for root disease surveys.

(Muir, BCFS)

c. Management guidelines called for favoring or planting tolerant or resistant species in 120- to 140-year-old-stands of Douglas-fir with *P. weirii* in interior B.C. Several areas in B.C. have been "stumped" to reduce or prevent disease. A project was proposed to collate and compile field data on previous work and suggest further trials, demonstrations, and development.

(Muir, BCFS)

5. Root Diseases In General

a. Impact surveys for root diseases (area out of production) have been completed for all northern Idaho Forests and the Lolo and Kootenai National Forests in northwestern Montana.

(James, Dubreuil, Byler, Dooling, FPM, R-1)

b. In cooperation with the Intermountain Forest and Range Experiment Station, a demonstration of silvicultural management of root diseases has been established on the Flathead IR north of Missoula.

(FPM, R-1)

c. Disease spread and progress of symptom expression in infected trees is being monitored in several 10- to 20-year-old stands.

(FPM, R-1)

d. Through excavation of entire root systems, the role of root disease complexes in the northern Rocky Mountains is being evaluated.

(FPM, R-1)

6. Survey Technology

a. The analysis program for root disease ground surveys can now accommodate up to nine different disease types recorded on the same survey form. Results including survey maps can be obtained for each type or combination of types.

(Bloomberg, PFRC)

b. A sorting program has been developed for root disease survey data which permits subdivision of the surveyed area into compartments and recalculation of root disease estimates for each compartment separately.

(Bloomberg, PFRC)

Following the meeting, Bill Bloomberg gave a short slide presentation on the use of radio-controlled model airplanes to take low-level aerial photographs of root-diseased areas.

Report of the Black Stain Subgroup of the Root Disease Committee

At the invitation of Fields Cobb, U.C. Berkeley, some 20 pathologists and entomologists working on Black Stain Root Disease met 10-12 September 1982 at the Blodgett Experimental Forest.

Representatives from the Canadian Forestry Service, Oregon State University, U.C. Berkeley, California Dept. of Forestry, Forest Pest Management in Regions 3, 4 and 5, Minnesota and Michigan were present. The session included reports on work in progress, comparison of isolates, discussion of the etiology of this unique disease, field observations, Hearts, and a marathon effort to keep Fields and his snoring somewhere else.

Two points were deemed worthy of the attention of WIFDWC as a whole. The specific epithet is properly spelled wageneri, with a single i. Dr. Kendrick erred grammatically in his original description; future papers should use the correct spelling.

Verticidadiella spp. are associated with wood staining of conifers around the world. Often they are secondary fungi introduced by insects. In a few cases they act as primary pathogens capable of killing trees. Taxonomy of the group is confusing and common names for the diseases are not standardized.

In an attempt to clarify the situation, our group agreed by consensus that Black Stain Root Disease is unique. The name should be confined to the disease caused by Ceratocystis (Verticicladiella) wageneri. The disease is characterized by a crescent-shaped pattern of xylem stain, caused by pigmented hyphae that are confined to xylem tracheids. Unlike other Verticicladiella species, V. wageneri does not colonize parenchyma cells. This unique behavior results in a unique symptomology. Futhermore, most other Verticicladiella species are not pathogenic or cause only localized infections upon inoculation. The several host-specialized forms of V. wageneri grow vertically in xylem tracheids of appropriate hosts after inoculation, often leading to tree death.

DISEASE CONTROL COMMITTEE

1982 INVESTIGATIONS

Kenelm Russell, Chairman

In a year of short money your 1982 disease control reports reflect a definite downward trend. Here is the score:

Number of Control Projects

Category	1981	1982
Seedling Diseases	13	9
Foliage Diseases	3	2
Root Rots	8	3
Rusts	2	1
Wilts	1	-
TOTAL	27	15

Nursery disease control tests lead the list. Root rots were strong last year but dropped dramatically this year. Is this real or just a lack of reporting? What about foliage diseases? Pacific Northwest Christmas tree growers need controls for Rhabdocline and several unknown maladies of both grand fir and noble fir. Some are fungus caused. How are the rest of you doing on foliage disease? Tell me your thoughts on where we should go with disease control for next year. I'll use this column as a forum for your comments. The 1982 list by disease category follows.

I. Seedling Diseases

A. Nursery Root Pathogens
Host: Conifer Seedlings
Causal Organisms: Fusarium, Pythium, nematodes
Control: Solarization
Development Stage: Field Trial

Solarization of Bend Nursery soil for control of pathogens and weeds. Comparison with fumigation and no treatment. Seedling survival in solarization plots comparable to no treatment. Fumigation most effective (Cooley).

B. Phytophthora Root Rot
Host: Douglas-fir
Causal Organisms: Phytophthora
Control: Chemical
Development Stage: Field Trial

Measurement of Subdue efficacy in preventing rootlet infection by Phytophthora. Applied as soil drenches (Cooley, P. Hamm).

C. Bird Predation
Host: Conifer Seeds
Causal Organisms: Birds
Control: Chemical
Development Stage: Field Trial

Evaluating the chemical Mesurol(R) as a bird repellant for conifer seeds in bare-root nurseries (Fuller).

D. Nursery Damping-off
Host: Conifers
Causal Organisms: Fusarium sp
Control: Biological
Development Stage: Field Trial

Use of solar pasteurization is being evaluated at Colorado State Nursery, Ft. Collins, CO. Trested summer 1982 to be planted spring 1983 (Hildebrand, Fuller).

E. Fusarium Hypocotyl Rot
Host: Sugar pine, red & white firs
Causal Organisms: Fusarium oxysporum
Control: Chemical, Biological, Silvicultural
Development Stage: Pilot Operational

Disease incidence has been reduced through a combination of control methods: soil fumigation in the fall previous to planting, 50% shade over the beds after seedling emergence, careful timing of irrigation, solar pasteurization and fungicide seed coating are being looked at through trials (Adams, Krelle, Brownell).

F. Fusarium Disease
Host: Sugar pine and Douglas-fir
Causal Organisms: Fusarium oxysporum
Control: Chemical and Biological
Development Stage: Field Trial

Captafol provided some control of the disease in 1981. 1982 trial includes seed treatments. Solarization controlled Fusarium but not macrophomena; trial is being repeated (McCain, Bega).

G. Phytophthora Root Rot
Host: Douglas-fir
Causal Organisms: Phytophthora spp.
Control: Chemical
Development Stage: Field Trial

We are testing the efficacy of the fungistat Ridomil or Subdue. So far, tree appearance has improved but the fungus is still present (Hansen).

H. General Seedling Foliage Diseases
Host: Conifers
Causal Organisms: Several (mainly Botrytis)
Control: Biological
Development Stage: Greenhouse

Preliminary test showed that certain seaweeds (dried) applied to foliage as a spray will reduce top height and crowding. This will mean lower humidity and foliage disease (Russell).

I. Sirococcus Tip Blight
Host: Pond pine
Causal Organisms: Sirococcus strobilinus
Control: Chemical
Development Stage: Field Trial

Bravo 500 applications were inconclusive. Several other fungicides were tried. Bayleton looks best so far. Results will be evaluated spring 1983 (Schwandt).

II. Foliage Diseases

A. Swiss Needlecast
Host: Douglas-fir
Causal Organisms: Phaeocryptopus gaeumannii
Control: Chemical
Development Stage: Full Operational and Field Trial

Studies have shown that a single timely aerial application of chlorothalonil will provide effective control of this disease. The impact of this disease on growth of plantation trees is being evaluated (Chastagner).

B. Swiss Needlecast
Host: Douglas-fir
Causal Organisms: Phaeocryptopus gaeumannii
Control: Chemical
Development Stage: Pilot Operational

Bravo 500 and Dithane M-45 were applied June 1, 1982 by helicopter with standard spray boom. Results will be evaluated in April 1985 (Russell).

III. Root Rot

A. Annosus Root Rot
Host: Ponderosa pine
Causal Organisms: Fomes annosus
Control: Biological and Silvicultural
Development Stage: Field Trial

Initiating studies to evaluate:
(1) Spread rate and persistence
(2) Methods to retard spread on margins
(3) Methods to reclaim disease centers
(4) Methods to reduce infection via spores (Williams, Marshall).

B. Fomes Annosus Root Rot
Host: Western hemlock
Causal Organisms: Fomes annosus
Control: Chemical
Developemnt Stage: Field Trial

Agricultural fungicides, captan ferbam, Daconil, etc., are ineffective in controlling stump infection by Fomes annosus. Wood preservative chemicals show more promise. (Edmonds, Driver).

IV. Rusts

A. White Pine Blister Rust
Host: Western white pine
Causal Organisms: Tuberculina maxima
Control: Biological
Development Stage: Field Trial

Sprayed Tuberculina maxima spores onto sporulating white pine blister rust cankers in a plantation to determine if Tuberculina maxima would become established, stop canker development, and spread to other untreated cankers (Hadfield, Harvey).

Business Meeting Minutes

The business meeting was called to order by Chairperson Bill Bloomberg at 9:30 a.m. on September 16. A summary of important points are presented here.

REPORTS

Disease Control Committee. No verbal report given since a written report is included in the proceedings.

Mistletoe Committee. No verbal report given since a written report is included in the proceedings.

Root Disease Committee. No verbal report given since a written report is included in the proceedings.

Interim Program. Walt Thies reported that he had received quite a few good suggestions. The written report is included in the proceedings.

Treasurer's Report. The treasurer's report shows a balance after the twenty-ninth meeting at $1032.78 and after the thirtieth meeting at an estimated $612.58. The difference was utilized to print the proceedings. This is about $350.00 more than was collected for the proceedings. Future secretaries should overestimate costs so this deficit is not repeated. The extra cost was due to the 32 honorary members and reduced rates for graduate students.

OLD BUSINESS

The minutes of last year's meeting and treasurer's report were approved as printed in the Proceedings of the twenty-ninth conference.

Cost of Proceedings. The membership agreed that life members should receive a free copy of the proceedings. The cost of these can be covered by higher charges to others or hopefully from interest on our balance.

Proceeding Disclaimer. There was general agreement among members that the proceeding is a publication in the eyes of libraries and others. Thus, to insure that the proceedings can remain a vehicle for reporting research progress and informal results the members voted to have Bill Jacobi and Bob Scharpf rewrite the present disclaimer to take into account the fact that the proceedings is a publication and is cited. The new version of the disclaimer can be found on page 1.

1983 Meeting Place. John Schwandt announced that the meeting would probably be held between August 29 and September 2, 1983, at Coeur d'Alene. There was a spirited discussion about dates for WIFDWC. Early dates such as the end of August were supported by members who teach and those with school age children. Later dates, i.e., September, were supported by members whose field season ran into September. The best suggestion made was to alternate "early" and "late" dates on a yearly basis.

NEW BUSINESS

1984 Meeting Location. Ken Russell invited us to the Seattle area with a Mt. St. Helen field trip. Greg Filip invited us once again to Bend, Oregon. Ed Wood invited us to the Santa Fe area. Members voted to meet in the Santa Fe area.

Length of Meetings. There was a discussion on extending meetings for one day so there is more time for workshops and committees. Some members felt the meetings were long enough and the added cost would be prohibitive. No decision was reached on this topic.

Special Papers. A member suggested that special papers should be given a specific length of time instead of the present variable system. This would allow members presenting papers and program chairman to plan better. No decision was made on this suggestion.

Field Trip. A member suggested that field trips should be on the first or last day so those that want to attend can and others don't have to. No decision was made on this suggestion.

Business Meeting. A member suggested that the business meeting should be held before the last hour of the meetings. No decision was made on this suggestion.

Election of Officers. The nominating committee of Bob Scharpf, Frank Hawksworth and Tom Laurent nominated John Laut for Chairman and Suzanne Dubreuil as Secretary-Treasurer. The nominations were accepted and unanimously voted into office.

Common Names. Frank Hawksworth and Bob Gilbertson were asked to prepare a list of common names of forest diseases of the West. They will present their results at the 1983 WIFDWC.

Balance on hand following twenty-ninth meeting		$ 946.13
(Extra deposit, October 1981 (Offord)		30.00
Interest paid July 1, 1980 through June 30, 1982		56.65
Total		$1032.78

Thirtieth WIFDWC Statement:

Receipts: Registration (76 people)	$ 962.00	
Expenses - meeting	674.00	
Balance		287.80
Balance September 30, 1982		$1320.58
Expenses - proceedings (140 copies) Estimated *		708.00
Balance February 28, 1983 (Estimated)*		$ 612.58

Deposit held: Washington State Employees Credit Union
P.O. Box WSECU
Olympia, WA 98507
Account No. 936258

* This estimate will be updated at 1983 meeting.

Duties Of WIFDWC Officers

In an attempt to help future officers in their positions the officers of 1982 have explained their responsibilities.

DUTIES OF WIFDWC SECRETARY-TREASURER

The secretary is responsible for taking notes during the meeting and especially the business meeting. The secretary is also responsible for informing the members how and when the special papers, panel reports, etc., should be turned in so they can be printed. All printing, editing and other activities related to producing the proceedings are the responsibilities of the secretary. Cost estimates of the proceedings are made before the meeting so the appropriate amount can be charged at registration.

DUTIES OF WIFDWC INTERIM PROGRAM CHAIRMAN

The interim program chairman is appointed by the current officers at the initiation of the conference. The interim chairman's responsibilities include soliciting suggestions for meeting improvements, ideas for papers, panels and workshops for the next year. The chairman should report his findings at the business meeting and provide the secretary a written copy to be placed in the proceedings.

DUTIES OF WIFDWC PROGRAM CHAIRMAN

The program chairman is responsible for the main portion of the WIFDWC program, including panels, discussions, workshops, special papers, etc. He is not responsible for project reports, banquet entertainment or the business meeting, and shares responsibility with the local arrangements committee and the general chairman for keynote address and field trip.

The program chairman, in consultation with the interim chairman, determines the major program events. He may delegate responsibility for a particular session to a moderator who will in turn select speakers.

The program chairman solicits special papers and posters, coordinating his mailing with the meeting announcement released by the local arrangements group.

The program chairman, in consultation with the general chairman and local arrangements committee, develops a preliminary program and a final program to be mailed to the members prior to the meeting.

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A. Forest Disease Surveys--General

71-A-4 Appraisal of damage caused by forest pests in British Columbia. (R. Alfaro and G. Van Sickle).

71-A-5 Forest insect and disease survey (G. Van Sickle).

71-A-7 Disease sampling in Douglas-fir plantations (G. Wallis).

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73-A-4 Forest disease: diagnostic and taxonomic services and research (J. Hopkins and R. Hunt).

74-A-1 Disease (and insect) detection surveys in Colorado forests (J. Laut and M. Schomaker).

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78-A-1 Evaluation of jack pine mortality in the Nebraska National Forest (E. Sharon).

79-A-1 DISACC: a computerized access and analysis system for forest tree problems (A. Partridge).

79-A-2 Standardizing damage estimation procedures for inventory foresters: a pictorial system (A. Partridge).

80-A-1 Standard damage estimating systems for major disease and insect problems in the inland Northwest (A. Partridge).

81-A-1 Pilot testing in R-3 of the pest damage inventory procedures developed in R-5 to provide estimates on disease losses for FIDIS (E. Wood).

81-A-2 Region-wide surveys to establish impacts of root rots for FIDIS (L. Fuller and D. Johnson).

81-A-3 Appraisal of damage caused by forest pests in the prairie Provinces (B. Moody).

81-A-4 Disease-caused growth impact in southern Utah commercial aspen stands (B. Tkacz).

81-A-5 Evaluation of disease occurrence and conifer family/disease occurrence associations in intermountain tree improvement plantations (J. Hoffman and J. Marshall).

81-A-6 Mortality of Chamaecyparis nootkatensis in southeast Alaska (T. Shaw and T. Laurent).

82-A-1 Root disease impact surveys of National Forests of northern Idaho and western Montana (R. James).

82-A-2 Pest damage inventory to evaluate one-year mortality on the San Bernardino NF (G. DeNitto).

82-A-3 Disease and insect impact on young-growth mixed conifer stands (J. Pronos and L. Dolph).

B. Non-Infectious Diseases

68-B-1 Detection of chronic photochemical oxident injury to conifers by remote sensing (P. Miller, R. Bega, and R. Heller).

68-B-2 Physiological impact on ponderosa pine growing under natural conditions of chronic exposure to oxidant air pollution (P. Miller).

71-B-1 Influence of the forest canopy on total oxident concentrations (P. Miller).

71-B-2 The effect of atmospheric effluents on the forest (D. Hocking and S. Malhotra).

72-B-1 Effects of smoke on forest disease fungi (J. Parmeter).

72-B-2 Chronic effect of photochemical oxident air pollution on the composition of the ponderosa pine-sugar pine-fir forest cover type (P. Miller).

78-B-2 Evaluation of air pollution effects on ponderosa pine in the Colorado Front Range (E. Sharon and J. Staley).

80-B-2 Trend of ozone injury to conifers in the southern Sierra Nevada (J. Pronos and D. Vogler).

81-B-1 Establishment of a network of air pollution (ozone injury) trend plots in the central Sierra Nevada National Forests (J. Allison).

82-B-1 Evaluation of ozone injury to conifers in the central Sierra Nevada (J. Allison).

82-B-2 Monitoring of ozone levels in the central and southern Sierra Nevada (J. Allison and J. Pronos).

82-B-3 Evaluation of sulfur dioxide levels on the Inyo N.F. (R. S. Smith).

C. Cone, Seed, and Seedling Diseases

71-C-1 Occurrence of endophytic fungi in conifer seedlings (W. Bloomberg).

76-C-1 Diseases of seeds and cones. PC-14-246 (J. Sutherland).

76-C-2 Simulation of forest nursery diseases. PC-40-157 (W. Bloomberg).

76-C-3 Potential of several species of Phytophthora for damage to coniferous forests and forest nurseries (E. Hansen and P. Hamm).

77-C-1 Nursery disease problems at the Albuquerque Tree Nursery (E. Wood and J. Riffle).

78-C-2 Greenhouse and nursery pathogenicity and symptomatology of four soil-borne fungi on five commercial species of conifers at various ages of growth (R. Bega).

79-C-1 Chemical & Biological control of soil-borne fungi on several conifer species at the Institute of Forest Genetics nursery. (R. Bega & A. H. McCain).

79-C-4 Identification of fungi on Northern Region conifer seed, their detrimental effects, and methods to reduce detrimental effects (J. Woo and R. James).

80-C-1 Fungicide efficacy tests to evaluate control of Botrytis blight at the Coeur d'Alene Nursery, Idaho (R. James and J. Woo).

80-C-2 Sugar pine hypocotyl rot in California forest nurseries. Etiology, inoculum sources, and host-parasite physiology (K. Brownell).

80-C-3 Effects of herbicides on mycorrhizae development of conifer seedlings in Rocky Mountain-Great Basin tree nurseries (A. Harvey and R. Ryker).

80-C-4 Pathogenesis of Fusarium on sugar pine at the Medford Nursery (C. Li, W. Thies, and E. Nelson).

80-C-5 Detection, identification, and quantification of impact of fungi on developing cones and seeds of Douglas-fir and western white pine (S. Cooley).

80-C-7 Parameters to describe normal and disease tree seedlings (A. Partridge).

80-C-8 Effect of sowing date on root disease and seedling growth in sugar pine (R. Bega, A. H. McCain, and J. Jenkinson).

81-C-2 Sirococcus shoot blight on ponderosa pine seedlings (J. Schwandt).

81-C-3 Seedcoat sterilization of conifer seeds using hydrogen peroxide (L. Fuller).

81-C-4 Mesurol[R] as a seed treatment to reduce bird predation (L. Fuller).

81-C-5 Reduction of pathogenic soil fungi in a forest nursery using solar radiation (L. Fuller).

81-C-6 Control of Fusarium cortical root in containerized conifers (L. Fuller).

81-C-7 Control of nursery pathogens and weeds by solarization (S. Cooley).

81-C-8 Control of Meria laricis on larch seedlings with fungicides (S. Cooley).

81-C-9 Control of Phytophthora root rot of true fir with Subdue (Ridomil) (S. Cooley).

81-C-10 Outplanting success of larch seedlings infected with Meria laricis (S. Cooley).

81-C-11 Outplanting success of noble fir seedlings grown in Phytopthora-infested soil (S. Cooley).

81-C-12 Benomyl and captan residues and biological activities in forest nursery soils (C. Li and E. Nelson).

81-C-13 Efficacy of inoculating Pisolithus tinctorius into conifer seedbeds at Lucky Peak Nursery (J. Marshall).

81-C-14 Survival and growth of seedlings in root disease centers (M. Militante and A. Partridge).

81-C-15 Pathogenicities and modes of infection of some fungi isolated from seeds and symptomatic seedlings of conifers (B. Advincola and A. Partridge).

81-C-16 Influence of pH and temperature on growth of and infection by a Cylindrocarpon sp. (B. Advincola and A. Partridge).

81-C-17 Root disease fungi of conifer seedlings not previously reported in the inland northwestern United States (B. Advincola and A. Partridge).

81-C-18 Effect of soil solarization on Fusarium & Macrophomina (A.H. McCain and R. Bega).

81-C-19 Effect of rhizobacters on root disease & seedling growth (A. H. McCain & R. Bega).

82-C-1 Endomycorrhiza & seedling responses of Incense Cedar (R. Bega).

82-C-2 Evaluation of seaweed applications to container seedlings for control of top height and root growth (K. Russell).

82-C-3 Fungicide efficacy tests to evaluate control of Botrytis blight at the Champion International Nursery, Plains, Montana (R. James).

82-C-4 Evaluation of fungicides to control damping-off and root disease at the champion International Nursery, Plains, Montana (R. James).

82-C-5 Mycofloral populations of ponderosa pine seed (R. James).

82-C-6 Tolerance of Botrytis cinerea to selected fungicides - BIA Nursery, Ronan, Montana (R. James).

82-C-7 Ponderosa pine needlecast at the Couer d'Alene Nursery, Idaho (R. James).

82-C-8 Conditioning, winter storage and initial field performance of containerized conifer seedling (H. Zalasky).

82-C-9 Fungicide trial to evaluate to evaluate efficacy against Phoma sp. on white fir Sirococcus strobilinus (J. Kliejunas, J. Allison, and A. McCain).

D. Root and Soil Diseases or Relationships (Including Mycorrhizae)

66-D-1 Investigations on the occurrence and control of Fomes annosus (C. Driver).

66-D-2 Studies on the cytology and genetics of Fomes annosus (C. Driver).

81-C-6 Control of Fusarium cortical root in containerized conifers (L. Fuller).

81-C-7 Control of nursery pathogens and weeds by solarization (S. Cooley).

81-C-8 Control of Meria laricis on larch seedlings with fungicides (S. Cooley).

81-C-9 Control of Phytophthora root rot of true fir with Subdue (Ridomil) (S. Cooley).

81-C-10 Outplanting success of larch seedlings infected with Meria laricis (S. Cooley).

81-C-11 Outplanting success of noble fir seedlings grown in Phytopthora-infested soil (S. Cooley).

81-C-12 Benomyl and captan residues and biological activities in forest nursery soils (C. Li and E. Nelson).

81-C-13 Efficacy of inoculating Pisolithus tinctorius into conifer seedbeds at Lucky Peak Nursery (J. Marshall).

81-C-14 Survival and growth of seedlings in root disease centers (M. Militante and A. Partridge).

81-C-15 Pathogenicities and modes of infection of some fungi isolated from seeds and symptomatic seedlings of conifers (B. Advincola and A. Partridge).

81-C-16 Influence of pH and temperature on growth of and infection by a Cylindrocarpon sp. (B. Advincola and A. Partridge).

81-C-17 Root disease fungi of conifer seedlings not previously reported in the inland northwestern United States (B. Advincola and A. Partridge).

81-C-18 Effect of soil solarization on Fusarium & Macrophomina (A.H. McCain and R. Bega).

81-C-19 Effect of rhizobacters on root disease & seedling growth (A. H. McCain & R. Bega).

82-C-1 Endomycorrhiza & seedling responses of Incense Cedar (R. Bega).

82-C-2 Evaluation of seaweed applications to container seedlings for control of top height and root growth (K. Russell).

82-C-3 Fungicide efficacy tests to evaluate control of Botrytis blight at the Champion International Nursery, Plains, Montana (R. James).

82-C-4 Evaluation of fungicides to control damping-off and root disease at the champion International Nursery, Plains, Montana (R. James).

82-C-5 Mycofloral populations of ponderosa pine seed (R. James).

82-C-6 Tolerance of Botrytis cinerea to selected fungicides - BIA Nursery, Ronan, Montana (R. James).

82-C-7 Ponderosa pine needlecast at the Couer d'Alene Nursery, Idaho (R. James).

82-C-8 Conditioning, winter storage and initial field performance of containerized conifer seedling (H. Zalasky).

82-C-9 Fungicide trial to evaluate to evaluate efficacy against Phoma sp. on white fir Sirococcus strobilinus (J. Kliejunas, J. Allison, and A. McCain).

D. Root and Soil Diseases or Relationships (Including Mycorrhizae)

66-D-1 Investigations on the occurrence and control of Fomes annosus (C. Driver).

66-D-2 Studies on the cytology and genetics of Fomes annosus (C. Driver).

66-D-3 Studies on the effects of site treatments (slash burning, fertilization, mechanical soil disturbance, etc.) on limiting the abilities of Phellinus weirii to infect the regenerating stand (C. Driver).

69-D-3 Relative species susceptibility to Phellinus weirii infection (E. Nelson).

71-D-2 Phellinus weirii root rot: biology and control (G. Wallis and D. Morrison).

71-D-3 Fomes annosus root and butt rot: biology and control (D. Morrison).

72-D-2 Armillaria mellea root disease: development and testing of stand management guidelines (D. Morrison).

72-D-3 Identification, distribution and intensity of root rots in western Montana and northern Idaho (R. James and S. Dubreuil).

73-D-1 Testing native conifer plantings for resistance to Phellinus weirii (K. Russell).

73-D-2 Testing red alder plantings to reduce Phellinus weirii development (K. Russell).

73-D-3 Alnus rubra as a biological control agent for Phellinus weirii (E. Hansen, E. Nelson, and J. Trappe).

73-D-4 Taxonomy and distribution of the endomycorrhizal fungi of the family Endogonaceae (J. Trappe).

74-D-6 Silvicultural prescriptions for management of stands affected by root diseases (N. Martin, R. James, and S. Dubreuil).

74-D-7 The role of ectomycorrhizas in conversion of nitrogen from inorganic to organic forms (C. Reid and R. France).

74-D-8 Selection and induction of drought resistance in trees from ecotypes of the Colorado Front Range: interaction of tree ecotype with its mycorrhizal symbiant (C. Reid and M. Cline).

75-D-1 Stump pushing in eastern Washington to control Phellinus weirii and subsequent performance of six planted conifers (K. Russell).

76-D-4 Simulation of root rot impact in second-growth coastal Douglas-fir stands (W. Bloomberg and G. Wallis).

76-D-5 Fertilization and root disruption to control laminated root rot of Douglas-fir (W. Thies and E. Nelson).

76-D-8 Evaluation of the rate of spread of black stain root disease, Verticicladiella wagnerii, in plantations (D. Goheen).

77-D-1 Characterization of zone lines formed on artificial media and in wood by Phellinus weirii (C. Li).

77-D-13 Inoculation of ponderosa pine seedlings with Pisolithus tinctorius (J. Riffle).

77-D-14 Evaluation of Pisolithus tinctorius inoculum produced by Abbott Laboratories for ectomycorrhizal development on pine species in container and bare-root nurseries in the Great Plains (J. Riffle).

77-D-15 Stump pushing in western Washington to control Phellinus weirii and subsequent performance of planted Douglas-fir and western hemlock (K. Russell).

78-D-1 Lab, greenhouse, and nursery tests on effect of six mycorrhizal fungi on five species of conifers (R. Bega).

78-D-5 Survival of Phellinus weirii in residual roots following stump removal and nitrogen fertilization (W. Thies).

78-D-6 Occurrence of Phellinus weirii beyond visible limits of infection (W. Thies).

78-D-7 Growth loss of Douglas-fir infected by Phellinus weirii (W. Thies).

78-D-8 Chemical control of Armillaria root rot near Glenwood, Washington (K. Russell).

78-D-9 Fomes annosus in thinned and chemically treated hemlock stands in Olympic Peninsula, Washington (D. Chavez, C. Driver, R. Edmonds, and K. Russell).

79-D-1 Surveys of root diseases in managed conifer stands in R-2 (R. Fuller and D. Johnson).

79-D-2 Fomes annosus on true firs in Colorado: distribution and impact (R. Fuller).

79-D-3 Verticicladiella wagnerii on pinyon pine at Mesa Verde National Park: disease spread characteristics and vector relationships (R. Fuller and C. Lister).

79-D-4 Interactions between root diseases and insects on true firs (R. Fuller).

79-D-5 Spread of Armillaria mellea disease centers in managed pine stands (R. Fuller).

79-D-9 Evaluation of effects of precommercial thinning in 10- to 20-year-old Douglas-fir plantations infected with Armillaria root rot in Oregon and Washington (G. Filip).

79-D-11 Evaluation of timber loss due to root disease in the Wagon Sale area, Sisters Ranger District, Deschutes National Forest, Oregon (G. Filip).

79-D-13 Comparison of root disease incidence in plantations of local versus nonlocal seed source stock (D. Goheen).

79-D-14 Occurrence of airborne spores of Fomes annosus at forest sites in southeast Alaska (T. Shaw).

79-D-15 Infection of Sitka spruce and western hemlock thinning stumps by Fomes annosus in southeast Alaska (T. Shaw).

79-D-16 Relative abundance of conidia and basidiospores of Fomes annosus in airborne inoculum (T. Shaw and E. Florance).

79-D-17 Evaluation of the incidence and impact of Fomes annosus in California fir stands (G. Slaughter, J. Mihaill, and J. Parmeter).

79-D-18 Evaluation of borax stump treatment for control of Fomes annosus in California fir stands (M. Schultz and J. Parmeter).

79-D-21 Displacement of Phellinus weirii from stumps by the antagonist, Trichoderma viride (E. Nelson and W. Thies).

79-D-22 Chemical control of Phellinus weirii (W. Thies and E. Nelson).

79-D-23 Susceptibility of Pacific Northwest conifers to laminated root rot (W. Thies and E. Nelson).

79-D-24 Conifer culture with roots in nutrient mist (A. Harvey).

79-D-25 Spatial relations of tree species in root disease areas (N. Martin).

79-D-26 Fungi and insects associated with and causing black stain root disease in Idaho (A. Partridge).

79-D-29 Evaluation of selected mycorrhizal fungi for improving the survival and growth of container grown Sitka spruce in southeast Alaska (T. Shaw).

79-D-30 Effect of red alder, cottonwood, and Douglas-fir on nitrogen and microbiological activity in soil (C. Li).

80-D-2 Black stain root disease of western North American conifers. Epidemiology and taxonomy of Verticicladiella wagnerii (T. Harrington and F. Cobb).

80-D-3 Distribution and activity of conifer mycorrhizae in Rocky Mountain forest ecosystems: impacts of disturbance, species, and age (A. Harvey).

80-D-4 Effects of fire management and intensive forest utilization on soil nitrogen status in northern Rocky Mountain timber types (M. Jurgensen and A. Harvey).

80-D-5 Evaluation of effects of precommercial thinning in 10- to 20-year-old red fir plantations infected with Armillaria root rot in southern Oregon (G. Filip).

80-D-7 Losses caused by black stain root disease in intensively managed Douglas-fir stands, Coos Bay District, BLM (D. Goheen).

80-D-9 Biology and management of Phellinus weirii (E. Hansen).

80-D-10 Identification and characterization of high and low laminated root rot hazard sites in the coastal Douglas-fir region (E. Hansen).

80-D-11 Insect-fungus interactions in the development of black stain root disease in Douglas-fir (E. Hansen).

80-D-12 Occurrence of Phytophthora lateralis in the forests of California (J. Klienjunes and D. Adams).

80-D-13 Systems of organisms causing black stain in pine roots (A. Partridge).

81-D-1 Black stain root disease: biology and control (R. Hunt and D. Morrison).

81-D-2 Growth loss of Douglas-fir caused by Phellinus weirii (W. Bloomberg).

81-D-3 Effects of Armillaria/bark beetle complexes on residual stocking (R. Fuller and E. Lessard).

81-D-4 Logging effects on root disease areas (J. Schwandt).

81-D-5 Impacts of root disease control measures by silvicultural means on soil and site productivity (R. Smith and E. Noss).

81-D-6 Evaluation of factors contributing to Armillaria root disease risk to conifer regeneration on potential stand conversion sites in the upper peninsula of Michigan (J. Bruhn).

81-D-7 Mortality caused by Fomes annosus in 10- to 20-year-old lodgepole pine plantations in central Oregon (C. Schmitt).

81-D-10 Evaluation of selected silvicultural treatments on root disease development in the Northern Region (S. Dubreuil, N. Martin, and R. James).

81-D-11 Odontia bicolor in coniferous root wood (C. Bertagnole and A. Partridge).

81-D-12 Hylurgops porosus as a possible carrier of Verticicladiella spp. (C. Bertagnole and A. Partridge).

81-D-13 Some conditions affecting the growth of Perenniporia subacida in culture and in wood (M. Chang and A. Partridge).

81-D-14 Phellinus weirii and Phellinus furrugineo-fuscus in wood: penetration and modes of action (E. Militante and A. Partridge).

81-D-15 Insect attractants produced by some Verticicladiella spp. and pine hosts (C. Bertagnole and A. Partridge).

81-D-16 Root disease agents associated with subalpine fir mortality in central and southern Utah (B. Tkacz).

81-D-17 Identification of root pathogens and development of root disease management strategies in southern Utah spruce forests (B. Tkacz).

81-D-18 Fomes annosus: longevity and rate of spread in a young ponderosa pine plantation (J. Hoffman and J. Marshall).

81-D-19 Fomes annosus: evaluation of methods to prevent introduction into and to remove existing inoculum from ponderosa pine stands in southwestern Idaho (R. Williams and J. Marshall).

81-D-20 Infection, development, and survival of Fomes annosus in large hemlock stumps created by clearcutting (B. Van der Kamp).

82-D-1 The application of chloropicrin or methyl isothiocyanate to live trees to control laminated root rot (caused by Phellinus weirii) (W.G. Theis).

82-D-2 The application of chloropicrin or Vorlex to infected stumps to eradicate Phellinus weirii (W. G. Thies and E. E. Nelson).

82-D-3 Endemic ectomycorrhizal fungi of ponderosa pine in central Great Plains plantings: identification of fungi and synthesis of ectomycorrhizae (J. W. Riffle).

82-D-4 Demonstration of Armillaria root disease control methods (S. Dubreuil, O. Dooling, and N. Martin).

82-D-5 Assessment of root disease development in young managed stands and plantations (R. James).

82-D-6 Assessment of Armillaria root disease in commercially thinned natural stands (S. Dubreuil).

82-D-7 Armillaria root rot of young intensively managed lodgepole pine stands of Alberta (Y. Hiratsuka).

82-D-8 Resistance screening of Port-Orford cedar to Phytophthora lateralis root rot (E. Hansen and P. Hamm).

82-D-9 Effect of precommercial thinning on development of black stain in root disease (E. Hansen, W. Thies, J. Witcorky).

82-D-10 Evaluation of the association of Mountain pine beetle and Armillaria in ponderosa pine in the Black Hills, South Dakota (L. R. Fuller, D. W. Washington, T. E. Hinds).

E. Foliage Diseases

74-E-1 Inheritance of resistance to Rhabdocline pseudotsugae in Douglas-fir (G. McDonald and G. Rehfeldt).

76-E-2 Evaluation of the growth impact of Rhabdocline pseudotsugae on sapling Douglas-fir in western Oregon (D. Goheen).

77-E-1 Dothistroma pini resistance in ponderosa pine (G. Peterson).

77-E-2 Inheritance of resistance to Dothistroma pini in Austrian pine (G. Peterson and D. Van Haverbeke).

77-E-4 Resistance to Phomopsis juniperovora in geographic sources of Juniperus virginiana and J. scopulorum (G. Peterson).

81-E-1 Needle casts on Christmas trees (J. Schwandt).

81-E-2 Impact (growth loss and mortality) of Hypodermella laricis and larch casebearer on western larch in northeastern Washington (D. Goheen).

81-E-3 Impact (growth loss and mortality) of Meria laricis and larch casebearer on western larch in eastern Oregon (J. Hadfield).

81-E-4 Fungicidal control of Swiss needle cast in Douglas-fir Christmas tree plantations in northwestern Oregon (J. Hadfield).

81-E-6 Identification of needle fungi associated with the "grey beard" needle disease of pines (R. Williams and J. Staley).

81-E-7 Growth of germ tubes positively-directed twoard stomates--is this a common phenomenon of fungi infecting plant foliage? (G. W. Peterson).

82-E-1 Helicopter fungicide applications to control Swiss needle cast in 8-12 year old Douglas-fir forest plantings (K. Russell).

82-E-2 Needlecasts and blights of western larch in the Northern Region (S. Dubreuil)

82-E-3 Dothistroma pini of ponderosa pine in northern Idaho (R. James).

F. Stem Diseases, Malformations, Witches-Brooms, Dwarf Mistletoes, etc.

62-F-1 Life tables for lodgepole pine and ponderosa pine dwarf mistletoe (F. Hawksworth and T. Hinds).

62-F-2 Ecology of lodgepole and ponderosa pine dwarf mistletoes (F. Hawksworth).

62-F-4 Taxonomy, hosts, and distribution of Arceuthobium (F. Hawksworth and D. Wiens).

62-F-5 Silvicultural control of ponderosa pine dwarf mistletoe in the Southwest (F. Hawksworth).

63-F-1 Spread and intensification of dwarf mistletoe in ponderosa and Jeffrey pines in California (R. Scharpf and J. Parmeter).

65-F-1 The effect of dwarf mistletoe on growth of western hemlock (K. Russell).

68-F-4 Spread and intensification of dwarf mistletoe in young unistoried stands of western larch, Douglas-fir, and lodgepole pine with controlled stocking (N. Martin).

69-F-1 Effectiveness of dwarf mistletoe control following special DM - precommercial thinnings in ponderosa pine and Douglas-fir (K. Russell).

71-F-1 Growth impact, associated mortality, and spread and intensification of dwarf mistletoe in stands of Douglas-fir, lodgepole pine, and western larch (O. Dooling and N. Martin).

71-F-2 Dwarf mistletoe control in rural and suburban residential developments (J. Laut and F. Hawksworth).

72-F-1 Simulation of the effects of dwarf mistletoe in ponderosa pine and lodgepole pine stands (F. Hawksworth, T. Hinds, and C. Edminster).

76-F-4 Inoculation studies to determine the host ranges of Arceuthobium campylopodum and A. occidentale in California (W. Mark, R. Scharpf, F. Hawksworth).

76-F-5 Biology and epidemiology of a Peridermium associated with lodgepole pine dwarf mistletoe (F. Hawksworth).

78-F-1 Expanded field plot study (into southwest Oregon) of Douglas-fir dwarf mistletoe development in thinned precommercial stands (D. Knutson).

78-F-2 Control of dwarf mistletoe-caused losses in young true fir stands by thinning (R. Smith, R. Scharpf, and D. Vogler).

78-F-3 Population dynamics of dwarf mistletoe on true firs in California (R. Scharpf and J. Parmeter).

78-F-4 The effect of dwarf mistletoe on mortality and volume loss in released true fir stands (R. Scharpf).

78-F-5 Reduction of dwarf mistletoe-caused mortality of Jeffrey pines by broom pruning (R. Smith and R. Scharpf).

78-F-6 Simulation of hemlock dwarf mistletoe infection and spread (W. Bloomberg, R. Smith, A. Thomson).

79-F-1 Lodgepole pine dwarf mistletoe surveys in the Gunnison National Forest (D. Johnson).

79-F-3 Dwarf mistletoe loss assessment in Douglas-fir, lodgepole pine and western larch in Montana and north Idaho National Forests (O. Dooling).

79-F-4 Dwarf mistletoe infection in young-growth western hemlock beneath infected old-growth residuals in southeast Alaska (T. Shaw).

79-F-5 Genetics of resistance of western hemlock to dwarf mistletoe (B. van der Kamp).

79-F-6 Relationship between spread of dwarf mistletoe and stand development in western hemlock (B. van der Kamp).

79-F-7 Growth loss in managed, even-aged, dwarf mistletoe-infested stands of ponderosa pine in the Pacific Northwest (E. Nelson).

79-F-8 Impact of dwarf mistletoe in the Intermountain Region (J. Hoffman).

79-F-9 Evaluation of dwarf mistletoe effects and development of a yield program for mixed conifer stands in the Southwest (R. Mathiasen, R. Gilbertson, F. Hawksworth, C. Edminister, and R. Wood).

80-F-1 Dwarf mistletoe loss assessment surveys (D. Johnson and F. Hawksworth).

80-F-2 Seed production and viability loss assessment of dwarf mistletoe of lodgepole and ponderosa pines (D. Johnson, F. Hawksworth, J. Laut, and B. Schaffer).

80-F-4 Changes in plant growth regulators in black spruce associated with infection by eastern dwarf mistletoe (W. Livingston, M. Brenner, F. Baker, R. Blanchette, and D. French).

80-F-5 Seed collection, storage, and inoculation of eastern dwarf mistletoe on black spruce and white spruce (W. Livingston, R. Blanchette, and D. French).

80-F-6 Root disease fungi found on black spruce infected with eastern dwarf mistletoe (W. Livingston).

80-F-7 Evaluation of effects of dwarf mistletoe on the growth and release of understory grand fir in central Oregon (G. Filip).

80-F-8 Adaptation of RMYLD to predict yields in dwarf mistletoe-infected lodgepole pine stands in the Pacific Northwest (C. Schmitt).

81-F-1 Resistance of Jeffrey pine to dwarf mistletoe, Arceuthobium campylopodium (R. Scharpf, B. Kinlock, and J. Jenkinson).

81-F-2 Correlation of ponderosa pine and Douglas-fir dwarf mistletoes with ecological factors (L. Merrill, F. Hawksworth and W. Jacobi).

81-F-3 Interactions of dwarf mistletoe and fire in lodgepole pine forests of the central Rocky Mountains (T. Zimmerman and F. Hawksworth).

81-F-4 Development of hemlock dwarf mistletoe following precommercial thinning of infected young stands in southeast Alaska (T. Shaw and T. Laurent).

82-F-1 Evaluation of the animal vectors lodgepole pine dwarf mistletoes in Colorado (T. H. Nicholls, F. G. Hawksworth).

82-F-2 Development of a framework for a yield simulation model in uneven-aged, mistletoe infected ponderosa pine stands (H. Maffei, W. R. Jacobi, F. G. Hawksworth).

82-F-3 Evaluation of timber growth productivity of southwestern mixed conifer stands in relation to habitat types and dwarf mistletoe (R. L. Mathiasen).

82-F-4 Dwarf mistletoe-related mortality of ponderosa and Jeffrey pines in campgrounds in California (Vogler and Scharpf).

G. Stem Diseases: Stains, and Decays

63-G-1 A study of Ophiostomaceae wood staining fungi in North America (R. Davidson).

72-G-2 Characterization and development of heartwood stain in Populus trichocarpa (A. Gokhele).

73-G-1 Decay associated with logging-damaged conifers in Oregon and Washington (P. Aho).

73-G-2 Tests of wound dressings on artificial injuries on western hemlock and Sitka spruce (P. Aho)

73-G-3 Decay hazard in advanced regeneration of tolerant conifers in Oregon and Washington (P. Aho)

73-G-4 The role of microorganisms in bark beetle epidemiology (H. Whitney).

77-G-1 Survey for Fomes fraxinophilus heart rot of green ash in natural stands in Nebraska (J. Riffle and E. Sharon).

79-G-1 Evaluation of decay in released stands of advanced grand and white fir regeneration in eastern Oregon and Washington (G. Filip and P. Aho).

79-G-3 Phellinus robineae stem decay of black locust: distribution, damage, and biology (J. Riffle).

79-G-4 Decay associated with logging wounds in young-growth white fir and red fir in northern California (P. Aho, R. Smith, and G. Fiddler).

79-G-5 Decays and cavity-nesting birds in the Pacific Northwest (A. Partridge).

79-G-7 Improved methods for identifying cultures of common wood-inhabiting fungi (A. Partridge).

80-G-1 Decay and height growth losses associated with Douglas-fir and grand fir tops killed by the spruce budworm in the Wenatchee and Okanogan National Forests (P. Aho).

80-G-2 The role of Actinomycetes in the discoloration and decay process of living trees (R. Blanchette).

80-G-3 Inonotus andersonii and decay of oaks in Arizona (K. Yohem and R. Gilbertson).

80-G-4 Rate of decay in mature grand fir and western hemlock infected by Echinodontium tinctorium in northern Idaho (J. Schwandt).

82-G-1 Bioactive metabolites of forest tree pathogens - Gremmeniella abietina, blue stain fungi associated with mountain pine beetle, Condrostereum purpureum, Verticicladiella spp. (Y. Hiratsuka, W. A. Ayer).

82-G-2 Incidence and damage caused by heart rots, primarily Hericium abietis, in old-growth Pacific silver fir-western hemlock stands on the Olympic National Forest (G. Filip).

H. Stem Diseases: Rusts and Cankers

53-H-1 Testing progeny of resistant pines for susceptibility to white pine blister rust in the Inland Empire (R. Bingham).

61-H-1 Streamlining pollination and progeny test methods in breeding for blister rust resistance in western white pine (R. Bingham).

61-H-2 Breeding and selection for climatic adaption in interspecies hybrids, toward accumulation of a pool of rust-resistance genes from other white pines of the world (R. Bingham).

66-H-1 Comparative physiology of varieties of western white pine with respect to their reaction to the blister rust fungus (R. Hoff).

66-H-4 Numbers and kinds of resistance genes and their relation to rust symptomatology (G. McDonald and R. Hoff).

66-H-5 Precise estimates of heritability and combining ability of rust resistance (G. McDonald).

66-H-6 Development and pathogenicity of Hypoxylon fuscum on northwestern species of alder (J. Rogers).

67-H-1 Etiology of aspen cankers (T. Hinds).

67-H-2 Field level of blister rust infection in early generation, partially resistant, western white pine stock (R. Hoff).

69-H-1 Thinning and pruning western white pine to control the blister rust disease (J. Byler and N. Martin).

71-H-3 Forest tree rusts of western North America (Y. Hiratsuka).

71-H-4 Computer simulation of white pine blister rust disease (G. McDonald and R. Hoff).

74-H-1 Rust fungi of Cupressaceae and Taxadeae: taxonomy and life histories (R. Peterson).

74-H-4 Biology, development, and systematics of Hypoxylon and its allies (J. Rogers).

74-H-6 Seed production areas for obtaining western white pine that is genetically improved for resistance to blister rust (R. Hoff and G. McDonald).

77-H-1 Characterization of Champion Mine race of Cronartium ribicola (G. McDonald and E. Hansen).

77-H-2 White pine blister rust (R. Hunt).

79-H-1 Diplodia tip blight in the Black Hills of South Dakota (G. Peterson and D. Johnson).

79-H-4 Ecological studies of spruce rust diseases in subarctic taiga forests. Coop with USFS and Univ. Alaska (J. McBeath).

80-H-1 Evaluation of aspen harvesting practices in Colorado and New Mexico (D. Johnson, T. Hinds, and J. Beatty).

80-H-2 A survey of the incidence and impact of stem rusts and Atropellis canker on immature lodgepole pine in British Columbia (B. van der Kamp).

80-H-4 Genetic variation of gall frequency in lodgepole and ponderosa pine seedlings inoculated with western gall rust (R. Hoff).

80-H-5 Inheritance of horizontal resistance mechanisms (R. Hoff).

80-H-6 Verification of white pine blister rust simulation (G. McDonald).

80-H-7 Pruning white pine for blister rust control (K. Russell).

81-H-1 Biology, cytology, and systematics of Xylaria (J. Rogers).

81-H-2 The effects of comandra blister rust on lodgepole pine: predicting the consequences of silvicultural treatments in rust-infected stands (B. Geils and W. Jacobi).

81-H-3 The etiology of Thyronectria canker on Colorado honeylocusts (W. Jacobi).

81-H-4 Mode of penetration and tissue invasion by Endocronartium harknessii (M. Chang and A. Partridge).

81-H-5 Biology and control of stem rusts of hard pines (R. Blanchette and D. French).

81-H-6 Wood deterioration by canker-rot fungi (R. Blanchette).

82-H-1 Hazard-rating and ecology of comandra blister rust in the Rocky Mt. Region (W. Jacobi).

82-H-2 Canker diseases of honeylocust: etiology, infection, and disease development. (J. W. Riffle and G. W. Peterson).

82-H-3 Guidelines for management of western white pine in the Northern Region (S. Dubreuil, G. McDonald, and G. Norby).

82-H-4 Western gall rust studies in relation to the genetic improvement program of lodgepole pinel (Y. Hiratsuka).

82-H-5 Incidence and intensification of blister rust on the Sierra N.F. (J. Kliejunas).

I. Wilt and Blight Disease

71-I-1 Dutch elm disease detection surveys in all municipalities in Colorado (J. Laut).

74-I-1 Control of Dutch elm disease using vector pheromones. Coop with USFS, NEFES, and CSFS. (C. Helburg, D. Leatherman, and J. Laut).

77-I-1 Distribution of Dutch elm disease and its principal vector, the smaller European elm bark beetle, in Montana urban areas (O. Dooling and S. Kohler).

77-I-3 Diplodia pinea tip blight on pines: etiology of stem infections (G. Peterson).

77-I-4 Herpobasidium deformans blight of honeysuckle: infection and control (J. Riffle).

79-I-1 Dutch elm disease control demonstration project in Colorado (D. Johnson and J. Laut).

79-I-2 Resistance to Cerospora sequoiae var. juniperi in geographic sources of Juniperus virginiana and J. scopulorum (G. Peterson).

80-I-1 Microbial antagonists as a biological control for Dutch elm disease (R. Blanchette).

80-I-2 Methyl bromide fumigation of oak wilt-infected oak logs (D. French).

81-I-1 Diplodia tip blight in the Black Hills of South Dakota (D. Johnson and G. Peterson).

J. Defects and Decays of Forest Products

58-J-1 Deterioration of beetle-killed Engelmann spruce in Colorado (T. Hinds).

68-J-2 Role of heartwood microflora in the breakdown of thujaplicin in western redcedar heartwood (B. van der Kamp).

71-J-1 Evaluation of potential wood preservatives: Thiram and Thiram-Oxathiin mixtures (R. Smith and C. Johansen).

71-J-2 Analysis of aspen chip deterioration during outside storage (R. Smith and C. Johansen).

72-J-2 Utilization of decayed wood in pulp manufacture (K. Hunt).

72-J-3 Degradation and preservative treatments of western redcedar shingles and shakes (A. Cserjesi, R. Smith, and T. Littleford).

73-J-1 Interaction of fungi and chemicals--pentachloraphenol (A. Cserjesi).

76-J-1 Microdistribution and efficacy of preservatives in treated wood and their effects on microorganisms (W. Wilcox).

79-J-1 Diagnosis of wood decay (W. Wilcox).

80-J-1 Deterioration of timber following the Mt. St. Helens eruption (K. Russell).

82-J-1 Deterioration of wind thrown timber in the Olympic Peninsula from the February 12, 1979, Lincoln Day storm (K. Russell).

K. Miscellaneous Studies

71-K-4 Species of Mycospaerella on Salicaceae in western interior of Canada (H. Zalasky).

71-K-5 Winter injury in poplar: a histological study (H. Zalasky).

71-K-6 Prevention of winter injury to conifers and other hardwoods (H. Zalasky).

72-K-1 The pathology of Ohia decline in Hawaii (C. Hodges).

73-K-2 Forest disease simulation model (W. Bloomberg).

73-K-3 Fungi of Washington State (J. Rogers).

76-K-2 Determination of cause of "drooping malady" in Colorado aspen (V. Scarpa, T. Hinds, and C. Livingston).

77-K-5 Development of operational use of biological control of forest pests in British Columbia. PC-45 (H. Whitney).

78-K-1 Effect of thinning on the incidence and impact of cytospora canker, fir engraver beetle, and Fomes annosus in white fir stands on the east-side Sierra Nevada (G. Ferrell, R. Scharpf, and J. Parmeter).

78-K-2 Reduction in stem volume of grand fir defoliated by western spruce budworm outbreaks in th Payette National Forest, Idaho (G. Ferrell and R. Scharpf).

79-K-1 Use of the Shigometer for assessment of tree vigor and growth in 25- to 100-year-old Sitka spruce and western hemlock (T. Shaw).

79-K-2 Mortality of Douglas-fir: biotic systems and impacts (A. Partridge).

79-K-3 Management alternatives in forests with Douglas-fir mortality centers (A. Partridge).

79-K-4 Revision and update of "Keys to major disease and insects..." in color (A. Partridge).

80-K-1 Evaluation of hazardous trees in forested recreation sites and ski areas (D. Johnson and E. Sharon).

80-K-2 Evaluation of diseases and their impact on Minnesota's shade trees (R. Blanchette).

80-K-3 Interactions among the pine wilt nematode, fungi, and bark beetles in the Midwest (M. Wingfield and R. Blanchette).

80-K-4 Evaluation of the Mount St. Helens eruptions on insect and disease activity in the blast area (J. Hadfield).

80-K-6 Computer programs to analyze street tree inventory data in urban areas of Idaho (J. Schwandt).

81-K-1 Comparative roles for saprophytic and pathogenic decays in Rocky Mountain forest soils: impacts of disturbance on regeneration and growth (A. Harvey and M. Larsen).

81-K-2 Life histories and anamorphs of lignicolous Pyrenomycetes (J. Rogers).

81-K-4 Reestablishment of vegetation on Mount St. Helens-created debris flow: an unusual "pathological" event (K. Russell).

82-K-1 Comprehensive pest management plan for Washington State (within the Forest Land Management Plan) (K. Russell).

82-K-2 Etiology and Epidemiology of Alaska yellow cedar in S.E. Alaska (E. Hansen, P. Hennon and T. Shaw).

Interim Program Chairman's Report

Walter G. Thies

A total of 30 participants of the 1982 WIFDWC offered multiple suggestions for the 1983 meeting. The following are the suggested items edited only to reduce duplication:

Topics for Panels or Special Invited Papers:

1. One panel each year dealing with basic biology.
2. Electronic data recording for lab or field.
3. Seed borne problems and nursery diseases. (IPC Note: This topic was mentioned often).
4. Status of research on Armillaria mellea.
5. Assessment of losses caused by forest diseases: techniques, philosophies, impact vs. loss.
6. Disease hazard rating systems for various diseases.
7. Technology transfer systems and techniques.
8. Role of forest pathology in forest planning.
9. Genetic engineering. (There seems to be some potential for this in ponderosa pine. Dallice I. Mills from OSU was suggested as a possible speaker.)
10. Plant growth promoting bacteria (siderophores).
11. Synthesis of ideas for management of important diseases:
    a. Thinning vs. pruning vs. clearcutting for control of mistletoe in various hosts.
    b. Possible stump treatments for control of Fomes annosus in various hosts.
    c. Clearcut vs. stump injection vs. stump removal for control of Phellinus weirii.
12. Relative roles of Forest Pest Management (FPM) and Forest Disease Research (FDR):
    a. Differences and similarities between FDR and FPM in mission and organization.
    b. Does FPM do research? Why?
    c. Is it permissible for FPM to do quasi research to answer short-term questions to fill gaps left by current research programs?
    d. Is FDR responsive (enough) to the needs of FPM or land managers? And vice versa? If not, how can this gap be bridged?

Suggested Workshops

1. Isolation and identification of various nursery disease pathogens: Fusarium spp., Pythium spp., and Phytophthora spp. (Ev Hansen, OSU, has volunteered to present a workshop on identification of Phytophthora spp.).
2. Armillaria mellea biotype identification.
3. Isolation and identification of Verticladiella spp.

Procedural Suggestions:

1. Hold the meeting before the beginning of September to avoid conflicts with University schedules.
2. Extend meeting by a day to allow more time for small group interaction, keep the evenings open (except for the banquet).
3. Hold the business meeting before the last day.
4. Use a self-contained meeting location whenever possible.
5. Keep costs to a minimum. Provide lower cost options for grad students and others on tight budgets.
6. Arrange for better name tags (easier to read).
7. Establish handout tables.
8. Encourage members to bring copies of recent publications.
9. Formally encourage posters. Continuing the quality of this year's posters will establish a valuable quality tradition.
10. Set a specific time for poster presentations.
11. Schedule more open time.
12. Encourage greater participation/attendance by more students, foresters, industry representatives.
13. Suggestions concerning special papers:
    a. Adequate time should be allowed for discussion of special papers.
    b. Set a time limit for presentation.
    c. Schedule special papers in a block with known starting times for each.
14. Limit extraterritorial presentations (with the exception of panel members) to posters or handouts.
15. Structure panels to be true panels. Emphasize discussion rather than presentation of a paper.
16. Hold concurrent panels. This has an advantage of covering more topics and smaller groups for discussion.
17. Continue the biological workshops along the lines of the polypore presentation (IPC Note: This comment was made often.)
18. Schedule the workshop as a daytime meeting, the late hour detracted from the usefullness of the polypore workshop. (IPC Note: This comment was made several times.)
19. Suggestions concerning presentation of new, modified or terminated projects:
    a. Limit oral reports to members from western states and invite others to provide written summaries.
    b. Limit presentation to one member from an institution.
    c. Report repetitious material or material of very limited interest (i.e., routine surveys) in written form to be made available as a handout.
    d. Encourage written summaries of progress, limit oral presentations to a few key points, not a review of all activities for the year.
    e. Schedule status reports so that they do not run against a time limit such as lunch.
    f. Split the status reports so that each group does not present an oral report each year.
20. Incorporate special field trips (i.e., the DM trip) into the week of WIFDWC.
21. Keep the date of the field trip flexible to take advantage of good weather.

22. Schedule the field trip for the first or the last day of the meeting.
23. Consider tactful entertainment after "limited" festivities at the banquet. This will minimize "unfortunate incidents."

Past Meeting Locations And Executive Committees

Conf. No.	Year	Meeting	Chairman	Secretary Treasurer	Program Chairman	Local Arrangements
1	1953	Victoria, B. C.	R. E. Foster	--	--	--
2	1954	Berkeley, California	W. W. Wagener	P. C. Lightle	--	--
3	1955	Spokane, Washington	L. J. Nordin	C. D. Lepahart	G. P. Thomas	--
4	1956	El Paso, Texas	L. S. Gill	R. W. Davidson	V. J. Nordin	--
5	1957	Salem, Oregon	G. P. Thomas	T. W. Childs	R. L. Gilbertson	--
6	1958	Vancouver, B. C.	J. W. Kimmey	H. R. Offord	A. K. Parker	--
7	1959	Pullman, Washington	H. R. Offord	R. E. Foster	C. G. Shaw	--
8	1960	Centralia, Washington	A. K. Parker	F. G. Hawksworth	J. R. Parmeter	K. R. Shea
9	1961	Banff, Alberta	F. G. Hawksworth	J. R. Parmeter	A. C. Molnar	G. P. Thomas
10	1962	Victoria, B. C.	J. R. Parmeter	C. G. Shaw	K. R. Shea	R. G. McMinn
11	1963	Jackson, Wyoming	C. G. Shaw	J. E. Bier	R. F. Scharpf	L. Farmer
12	1964	Berkeley, California	K. R. Shea	R. F. Scharpf	C. D. Leaphart	H. R. Offord
13	1965	Kelowna, B. C.	J. E. Bier	H. S. Whitney	R. V. Bega	A. C. Molnar
14	1966	Bend, Oregon	C. D. Leaphart	D. P. Graham	G. C. Pentland	D. P. Graham
15	1967	Santa Fe, New Mexico	A. C. Molnar	E. F. Wicker	L. C. Weir	P. C. Lightle
16	1968	Coeur d'Alene, Idaho	S. R. Andrews	R. G. McMinn	J. L. Stewart	C. D. Leaphart
17	1969	Olympia, Washington	G. W. Wallis	R. L. Gilbertson	F. G. Hawksworth	K. W. Russell
18	1970	Harrison Hot Springs, B.C.	R. F. Scharpf	H. V. Toko	A. E. Harvey	J. Roff
19	1971	Medford, Oregon	J. A. Baranyay	D. A. Graham	R. B. Smith	H. H. Bynum
20	1972	Victoria, B. C.	P. C. Lightle	A. H. McCain	L. C. Weir	D. Morrison
21	1973	Estes Park, Colorado	E. F. Wicker	R. C. Loomis	R. L. Gilbertson	J. G. Laut
22	1974	Monterey, California	R. V. Bega	D. Hocking	J. R. Parmeter	--
23	1975	Missoula, Montana	H. S. Whitney	J. W. Byler	E. F. Wicker	O. J. Dooling
24	1976	Coos Bay, Oregon	L. F. Roth	K. W. Russel	L. C. Weir	J. Hadfield
25	1977	Victoria, B. C.	D. P. Graham	J. G. Laut	E. E. Nelson	J. Bloomberg
26	1978	Tucson, Arizona	R. S. Smith	D. B. Drummond	L. C. Weir	R. L. Gilbertson
27	1979	Salem, Oregon	T. H. Laurent	T. E. Hinds	B. Van Der Kamp	L. C. Weir
28	1980	Pingree Park, Colorado	R. L. Gilbertson	O. J. Dooling	J. G. Laut	M. Schomaker
29	1981	Vernon, B. C.	L. C. Weir	C. G. Shaw III	J. Schwandt	D. J. Morrison/R. S. Hunt
30	1982	Fallen Leaf Lake, CA	W. J. Bloomberg	W. R. Jacobi	E. Hansen	F. Cobb/J. Parmeter/R. Scharpf

Social Achievement Award Winners

Conference	Location	Winner
5	Salem	Stuie Andrews
6	Vancouver	Stuie Andrews
7	Pullman	Don Leaphart
8	Centralia	Keith Shea
9	Banff	Phil Thomas
10	Victoria	Toby Childs
11	Jackson	Alex Molnar
12	Berkeley	Reed Miller
13	Kelowna	Art Parker
14	Bend	C. Gardner Shaw
15	Santa Fe	Larry Weir
16	Coeur d'Alene	Bob Scharpf
17	Olympia	Dick Parmeter
18	Harrison	Jim Kimmey
19	Medford	Ed Wicker
20	Victoria	Vivian Muir
21	Estes Park	Tom Laurent
22	Monterey	Bob Bega
23	Missoula	Art McCain
24	Coos Bay	--
25	Victoria	Ray (Founder) Foster
26	Tucson	John Hopkins
27	Salem	Oscar Dooling
28	Pingree Park	Tommy Hinds
29	Vernon	Fields Cobb
30	Fallen Leaf Lake	John Laut

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