Proceedings of the 28th Annual Western International Forest Disease Work Conference

Pingree Park, Colorado
September 1980

Compiled by:
Oscar J. Dooling
Forest Insect and Disease Management
USDA Forest Service
Missoula, Montana.

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The twenty-eighth annual Western International Forest Disease Work Conference met at Pingree Park, Colorado, on September 15-19, 1980.

Chairman Bob Gilbertson opened the Conference at 9 a.m. Tuesday. Bob's brief comments were unimportant and not included in these proceedings.

Colorado State Forester Tom Borden welcomed WIFDWC members to Colorado and Pingree Park. Tom didn't have notes, so his remarks are not included in the proceedings. He told several Standard Borden Jokes and warned us not to get hung up with the "mine" syndrome.

Two WIFDWC members received honors during the past year:

Tom Harrington won a Mycological Society of America $1,000 scholarship, and Lew Roth was elected a Fellow of the American Photopathological Society. Congratulations, guys.

The theme of the 28th Conference was FOREST PATHOLOGY--WHO CARES? Ken Russell's advice? "Keep your ear to the grindstone and listen to the wind."

Fifty-one persons registered for the Conference. Several wives (and one husband) joined the group for a steak fry and social Thursday evening. An old mountain man (Larry Helberg) visited the group after dinner.

Officers for the 28th Conference were:

Bob Gilbertson Chairman
Oscar Dooling Secretary-Treasurer
John Laut Program Chairman
Mike Shoemaker Local Arrangements

Thanks to everyone who helped make the Conference a success, especially Sherry Pommering, Pingree Park Director of Conferences.
FIELDS COBB MEMORIAL WALKING TOUR

W.I.F.D.W.C. - Pingree Park  September 1980

Stop

1  **Cronartium comandrae** in pole-sized lodgepole pine.

2  Recent clearcut in overmature lodgepole pine. Stand was very heavily infested with dwarf mistletoe. Size of clearcut is approximately 5 acres, much smaller than is used for commercial sales in this area (20-40 acres). Gross volume removed was approximately 3600 cubic feet per acre.

3  Various treatments are being applied to this approximately 10-acre area (see attached map). Five stand types are represented in this area.

   A. **Overstory.** This mature stand of lodgepole pine was very heavily infested by dwarf mistletoe and approximately 1/3 of the trees had already been killed by the parasite. Average dwarf mistletoe rating (DMR) was 5.1. Average tree size was 7 inches d.b.h. and 33 feet tall. Basal area 70 square feet/acre.

       Treatment:  Complete overstory removal--natural regeneration from cones in slash.

   B. **Scattered overstory with 30-year-old understory.** Stand data:

<table>
<thead>
<tr>
<th></th>
<th>Overstory</th>
<th>Understory</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Trees/acre</td>
<td>120</td>
<td>-</td>
</tr>
<tr>
<td>Ave. d.b.h.</td>
<td>7.8</td>
<td>-</td>
</tr>
<tr>
<td>Ave. height</td>
<td>31</td>
<td>-</td>
</tr>
<tr>
<td>BA/acre</td>
<td>40</td>
<td>-</td>
</tr>
<tr>
<td>DMR</td>
<td>5.8</td>
<td>-</td>
</tr>
</tbody>
</table>

       Treatment:  Remove all overstory trees; thin understory from growing stock level 110 to 60.

   C. **30-year-old stand, essentially no overstory.** Stand data:

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trees/acre</td>
<td>690</td>
<td>210</td>
</tr>
<tr>
<td>Ave. d.b.h.</td>
<td>3.6</td>
<td>4.8</td>
</tr>
<tr>
<td>Ave. height</td>
<td>17</td>
<td>.22</td>
</tr>
<tr>
<td>BA/acre</td>
<td>50</td>
<td>26</td>
</tr>
<tr>
<td>DMR</td>
<td>1.0</td>
<td>0.8</td>
</tr>
</tbody>
</table>

       Treatment:  Thin from growing stock level 130 to GSL 100.
D. 70-year-old pole stand previously thinned. Stand data:

<table>
<thead>
<tr>
<th>Trees/acre</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ave. d.b.h.</td>
<td>570</td>
<td>340</td>
</tr>
<tr>
<td>Ave. height</td>
<td>6.2</td>
<td>7.0</td>
</tr>
<tr>
<td>BA/acre</td>
<td>31</td>
<td>33</td>
</tr>
<tr>
<td>DMR</td>
<td>127</td>
<td>90</td>
</tr>
<tr>
<td>B/Acre</td>
<td>2.7</td>
<td>2.3</td>
</tr>
</tbody>
</table>

Treatment: Thin from growing stock level 175 to GSL 110.

E. 70-year-old unthinned pole stand.

Predominantly lodgepole pine with patches of aspen and Engelmann spruce. Thinning data:

<table>
<thead>
<tr>
<th>Trees/acre</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ave. d.b.h.</td>
<td>755</td>
<td>190</td>
</tr>
<tr>
<td>Ave. height</td>
<td>4.4</td>
<td>6.3</td>
</tr>
<tr>
<td>BA/acre</td>
<td>28</td>
<td>36</td>
</tr>
<tr>
<td>DMR</td>
<td>80</td>
<td>40</td>
</tr>
</tbody>
</table>

Treatment: Thin from growing stock level 160 to GSL 60.

Optional visit to discuss Shigometer vigor rating in relation to dwarf mistletoe infection intensity and thinnings in lodgepole pine. The thinnings were made approximately 40 years ago in a stand now about 100 years old. Four plots: 1 unthinned, and others thinned to spacing of about 8 x 8, 10 x 10 and 12 x 12 feet were established:

<table>
<thead>
<tr>
<th>Plot</th>
<th>1941</th>
<th>Merch. cu.</th>
<th>1980</th>
<th>Merch. cu.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Trees/acre</td>
<td>ft./acre</td>
<td>Trees/acre</td>
<td>ft./acre</td>
</tr>
<tr>
<td>Unthinned</td>
<td>6060</td>
<td>-</td>
<td>2860</td>
<td>-</td>
</tr>
<tr>
<td>8 x 8</td>
<td>680</td>
<td>-</td>
<td>664</td>
<td>1540</td>
</tr>
<tr>
<td>10 x 10</td>
<td>464</td>
<td>-</td>
<td>392</td>
<td>1410</td>
</tr>
<tr>
<td>12 x 12</td>
<td>320</td>
<td>-</td>
<td>288</td>
<td>1980</td>
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<tbody>
<tr>
<td></td>
<td>BA</td>
<td>GSL</td>
<td>BA</td>
<td>GSL</td>
</tr>
<tr>
<td>Unthinned</td>
<td>200</td>
<td>1030</td>
<td>200</td>
<td>500</td>
</tr>
<tr>
<td>8 x 8</td>
<td>63</td>
<td>138</td>
<td>138</td>
<td>188</td>
</tr>
<tr>
<td>10 x 10</td>
<td>36</td>
<td>88</td>
<td>93</td>
<td>120</td>
</tr>
<tr>
<td>12 x 12</td>
<td>36</td>
<td>69</td>
<td>91</td>
<td>121</td>
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CAUSAL AGENTS OF COMMON TREE DISEASES
IN PINGREE PARK AND VICINITY

LODGEPOLE PINE

*Arceuthobium americanum*
*Cronartium comandrae*
*Endocronartium harknessii* (orange spored)
*Cronartium stalactiforme*
*Peridermium bethelii*
*Necpeckia coulteri* ("Bear wipe")
*Lophodermella concolor*
*Lophodermella montevaga*
*Armillaria mellea*
*Phellinus pini*
*Polyporus circinatus*
*Coniophora puteana*

PONDEROSA PINE

*Arceuthobium vaginatum*
*Arceuthobium americanum*
*Peridermium filamentosum* (2 races)
*Cronartium comandrae*
*Endocronartium harknessii* (white spored)
*Elytroderma deformans*
*Davisonymycella ponderosae*
*Davisonymycella medusae*
*Phellinus pini*
*Polyporus aniceps*
"Buckhorn Blight" (Hail?)

LIMBER PINE

*Arceuthobium cyanocarpum*
*Phellinus pini*
*Lophodermium arcuata*
*Bifusella linearis*
*Bifusella saccata*

ENDELMANN SPRUCE

*Chrysomyxa arctostaphyli*
*Herpotricha nigra*
*Phellinus pini*
*Phellinus nigrolimitatus*
"Genetic" brooms

BLUE SPRUCE

*Chrysomyxa arctostaphyli*
*Lirula macrospora*

DOUGLAS-FIR

*Rhabdocline pseudotsugae*
*Rhabdocline weirii*
*Phellinus pini*

SUBALPINE FIR

*Mellampsorella caryophyllaceous*
*Lirula abietis-concoloris*
*Ceratocystis-Dryocetes complex*
*Armillaria mellea*
*Herpotricha nigra*
*Grovesiella abieticola*

ASPEN

*Cenangium singulare*
*Ceratocystis fimbriata*
*Cryptosphaeria populina*
*Cytospora chrysosperma*
*Marssonia populii*
*Pollacia radiosa*
*Ciborinia whetzelii*
*Phellinus igniarius*
*Ganoderma applanatum*
NEEDLE CAST IDENTIFICATION WORKSHOP

John M. Staley

An identification workshop dealing with needle cast fungi and related organisms was presented. Attendees were encouraged to take specimens from those on display for use in their herbaria, or to form the nucleus of a personal collection.

The taxonomic framework for needle cast fungi was discussed and certain arbitrary aspects pointed out. Techniques of collection, examination, culture, illustration, and preservation were described. Stroma tissue type, embeddment, color, variation in size of ascocarp, symptoms, phenology, and cultural characteristics were discussed as useful taxonomic criteria. Their use in effecting recent changes in Lophodermium was presented as an example. The use of host and geographical distribution, and the use of known specimens for comparative purposes were stressed as practical means of achieving accurate diagnoses.

Below is a more detailed listing of specimens displayed by name and (usually) by herbarium (FFP) number. By reference to this list, additional information may be found concerning the specimens given you at the workshop.

*Bifusella linearis* (Peck) v. Hohn. FPF 3249. *Pinus flexilis*
James. Larimer Co., Colorado 7/19/63.

*Bifusella pini* (Dearn.) Darker. FPF 4438. *Pinus monophylla*
Torr. & Fremont. Mono Co., California. 7/10/64.

*Bifusella saccata* (Darker) Darker. FPF 3248. *Pinus flexilis*
James. Larimer Co., Colorado. 7/19/63.

San Juan Co., Utah. 5/4/65.


*Davisomyces medusa* (Dearn.) Darker. FPF 3223. *Pinus ponderosa*

*Davisomyces montana* (Darker) Darker. FPF 4439. *Pinus contorta*
Dougl. Calaveras Co., California. 7/10/64.

*Davisomyces ponderosae* (Staley) Dubin. FPF 3198. *Pinus*


Lophodermium decorum Darker. FPF 3423. Abies concolor (Gord. & Glend.) Lindl. ex Hildebr. Pueblo Co., Colorado. 8/11/64.


Lophodermium pini-excelsae Ahmad. FPF 4497. Pinus sylvestris L. Lincoln Co., Oregon. 2/12/76.


Rhabdocline weirii Parker & Reid. Pseudotsuga menziesii (Mirb.) Franco. Bayfield Co., Wisconsin. 6/7/79.


I wish to take this opportunity to thank members of the WIFDWC and scientists at the North Central and Southern Forest and Range Experiment Stations for sending specimens included in the display.
OPEN DISCUSSION: PUBLIC PERCEPTIONS OF FOREST PATHOLOGY

Discussion Leaders
Ken Russell                Mike Finnis

Special editing help by Valerie Scarpa

This was an open discussion on public perceptions of forest pathology. Are there any? Should there be any? How can they be improved? Who is our public, etc.

WIFDWCers have always been known for their vocal and other contributions and this "nonpanel" was a superb example of the beginning of a good thing.

The best arrangement for complete group participation was having the chairs in a circle and encouraging everyone to fill the front two rows. This psychological plus provided participants with the feeling they were talking to one another, not the leaders. We have summarized your written comments and added our own to round out the discussion. Some of you signed your name which we printed at the end of your contribution. Others who didn't will be listed Anon. Thanks for your "mini papers" and statements.

First, we identified our "public". This list is very inclusive as we soon found out (no special order):

1. Special interest groups - environmentalists, developers, etc.
2. Pathologists
3. Administrators
4. Students
5. Scientists in other disciplines
6. Managers
7. Woods Workers
8. Politicians
9. Media
10. Private landowners
11. Forest industry
12. Public utilities
13. Pollution producers
14. Local government
15. City slickers
16. Suburb dwellers
17. Native Americans
18. Neighborhood planning associations
19. Other agencies
20. Farm forestry organizations

By now you can see that the list could be much longer.
Next we listed how we contact our public (again, no special order):

1. Outdoor school
2. Service clubs
3. Media
4. User oriented publications
5. Silviculture certification program
6. Team teaching
7. Pathology hotline
8. Training seminar
9. Environmental impact statements
10. Bring outsider (boss, manager or other) to conference
11. Timing information releases to coincide with attention getting disease

How can we improve public perceptions? We found in discussing this subject that it was very difficult to avoid becoming enmeshed in a communications discussion. When you think about it, perceptions of what we do and the communication process are closely related.

Various pathologists deal with different segments of the public according to their jobs. Each person must seriously consider which segment, then deal with that public appropriately. The way to make a pathological problem familiar to a public is to sell it in an attractive package. Consider the very professional (artistic and pathological combined) Shigo CODIT package. Look how it sold and how popular it became. We may find faults with it in our specific expertise, but look how much good CODIT principles have done for foresters, teachers and numerous other segments of the public. Those that study the attractive material actually understand some of the things that go on inside a tree when it is injured or begins to decay. More of that kind of professional packaging will help the pathology discipline. Well organized, simple to understand cost-benefit information also goes a long way, especially to convince administrators or legislators of a program you see a need for. Just tell 'em how much money they'll lose if they don't do it - that will get their attention! (Note: Read the editorial by Bill Merrill in the November 1980 Plant Disease, p. 981; Research, Peddling the Product.)

"For example, I received good administrative support for a recreation hazard tree program in Washington (even beyond my expectations) by digging up examples of hazard tree court cases and their costs. Above all else, an agency does not want to go to court. It's expensive, and think of the adverse publicity! The cost of the hazard tree program alone is a good insurance premium to minimize accidents and potential court cases, and demonstrate to the public that we are concerned about tree safety." Ken Russell
The researcher deep in the lab has sales problems similar to the extension forester who is trying to convince an uninterested or uneducated public of a disease problem. It's possible for both pathologists to use professional expertise in selling their respective packages. Such help is often available right in the agency where the pathologist works.

A terrific outlet of recent times which we all have opportunity to contribute to are Environmental Impact Statements. Those documents are thorough! If you want to educate someone about a disease just bring it up in the EIS. Your boss or administrators will find it is a good way to communicate too. The EIS can serve as a three-way communication to the administrator, the public, and back to the researcher (contributor) in the form of comments or questions. If you are good at selling concepts or programs and did a proper job in the EIS, the bosses may realize how important that fan-tailed rust really is. You may have beau coup support for it next budget time. The word is: sell!, and don't forget to sell it with very attractive packaging.

Here are some pitfalls in EIS's. Don't assume the pigheadedness or "Father knows best" attitude. For example, avoid mentioning that herbicides (major heading) are your only tool for controlling unwanted vegetation. Instead, say that vegetation management (major heading) is a serious problem and herbicides are one of our methods of controlling them. "Other alternatives are: hand brushing, burning, etc."

Don't say that "this is the way it should be done and there isn't any other way!" The public will jump right down your throat!

"Timing is a great thing for alerting to potential problems. In B.C. we taught private landowners about dwarf mistletoe through the bark beetle problem. When the landowner asked about the beetle we told him about both pests. A matter of high interest at the right time!" Mike Finnis

The matter of forester education and pathology ...

"There seemed to be a foregone conclusion in the group that we forest pathologists attempt to carry our message to the public ... Who should we really try to reach and how? We brought out the point that the public may not be our primary emphasis. We should be addressing within our discipline to problems relating to pathology." This wasn't discussed as much as could be and I think merits more. We are specialists in pathology, not communication and perhaps our time would be better spent in pathology. Tricia White
"Generally the public is not exposed to forest pathology even in places like forestry schools. Unless we can get to special interest groups like foresters, how can we expect to get to the general public? Pathologists need to spend more time selling, especially through the methods in the list noted previously. Foresters have little room for pathology as there are so many demands on their time that take higher priority. Begin by educating foresters." Mike Finnis

"One of the most effective ways to further the cause of forest pathology, has to be development of better education programs for forest managers, etc., through continued support of university programs, increased input in silvicultural training programs and more workshops." Borys Tkacz

"The initial primary concern should be the users and/or potential users of the information that we as pathologists (specialized resource managers) have to share. The problems with these users (other resource managers and policy makers) begin with their college and university training. The Society of American Foresters (SAF) no longer requires that forest managers have exposure to knowledge of forest diseases, and it is my understanding that many institutions of "higher learning" have no pathology requirements, do not even recommend exposure, or have a totally inadequate course.

This lack of exposure to pathology as an undergraduate not only represents a serious weakness in training, but introduces a sometimes strong bias in the philosophy of the young forester. If forestry professors seriously belittle diseases, why should the trainee listen to the pathologists? And if we pathologists cannot get the forester to listen and to use our knowledge, how can we hope to accomplish anything of practical value to humankind?

Hence, it seems that the first line of action should include a very serious attempt to influence the SAF and forestry faculties. Further, I am usually apologizing for a lack of concrete contributions to the area of forest resource management. We pathologists have made some very obvious contributions, mostly unrecognized, to the ways that stands are managed. First, we need to recognize these contributions, then point them out to the forest manager. Without us, foresters would be making a lot more errors than are currently being made now." Fields Cobb

"Well, we could begin a vigorous "sales" program by accepting the fact that we are a support discipline and throw in with foresters in their education process. Couldn't pathology or facets of pathology principles be integrated with silviculture courses? This is an area of pathology in great need of serious discussion. Maybe we are trying too hard to stand on our own." Ken Russell
"It is virtually impossible for our small group of professional pathologists to reach all of our publics. I feel we should concentrate our efforts on the professional forest manager (unit or area foresters). These are the people who have contact with and a feel for our publics' needs. Secondly, we should keep our area politicians apprised of current disease situations so when the general public contacts them, they can answer intelligently. A simple straight forward status report is greatly appreciated by most legislators."

Larry Helburg

"A need exists to have forest pathologists consider their recommendations in terms of forest management rather than just timber management."

Jim Hoffman

"It's important to educate the public about forest pathology and the need for it. However, I think that should be the responsibility of extension personnel. Researchers' prime responsibility should be toward research, teachers to teaching and FIDM people to working with land managers. If there is time for more extension people to educate the public, fine, but priorities should be in doing the jobs the pathologists are paid for. I didn't see any federal forest extension people at the meeting."

Bruce Schaffer

"The general public, which includes natural resource managers, must be reached. To do this, the forest pathology specialists (or at least some of them) should take the time to generalize knowledge in a format usable to the natural resource manager and general public."

Ron Morrow

A consensus appeared to be that we as pathologists should work within our own group and then go out and attend meetings of other groups such as Pest Action Councils or Western Forestry Conference or chapters of SAF to alert the general public and special interest groups to our problems.

The matter of dealing with the public . . .

"The message telling about forest pathology is a very important activity for all forest pathology - whatever their position, research, FIDM, etc. No one is exempt!"

Anon

"People in research should have better outlets for transferring their ideas to a public. They should know something about packaging and marketing a product, idea or program. They should know who to see and know when to push. The work isn't really finished until the idea is properly transferred.

Example: A researcher I know found a better spacing for soybeans. He had to get this to the farmers. Logically, the extension agent should do this. Unfortunately, the agent sort of shuffled the proposal off in a pile where it gathered dust because he was too busy. That's where it sits! This is pathology education?"

Valerie Scarpa
"The "problem" of public involvement (education) should first be identified (i.e., what is the problem, if any?). We discussed ways of overcoming the problem without first identifying an achievement goal. A problem solving flow should be followed to better construct problem solving means." Jack Marshall

"Why not develop a questionnaire on public perceptions of forest pathology?"

"The chain of command creates problems."

"Lets market our product better!"

"We must have adequate impact information to know better how hard we need to sell the product. NEPA and SEPA (Federal and State environmental protection acts) mandate more public involvement in resource management decisions - including attention to pest management problems."

"The public is biased."

"An extremely "bright light" is that graduate student involvement in our discussion was high - a good sign for the future of WIFDWC." Anon

"We need to involve communications specialists as well as other forest related sciences in our extension efforts when necessary. We are a service group to urban and forest management, so we need to work together not just as forest pathologists."

"The public's perception affects us directly because action, research and extension are in desperate shape for money, support, etc." Anon

"If the conference finds difficulty in implementing the results of its research, or in having its research supported, it should seek a broader base of support. This support will be derived from "the public" which is composed of many sectors, each important in its own way. These sectors should be suitably approached, by many means, and brought around to an understanding and participation." Norm Alexander

"Would it be a good idea to consult with a communication specialist when we need to go outside our discipline to the public?"

"We need a "Smokey Bear" for pathology. Sell our product!" Anon

"The public can be defined, but it must be defined, and addressed by a particular issue. The means of communication must then be tailored to the issue." Anon
"What are the publics our groups must identify and voice concerns to? We represent many groups within and should probably interact with those groups we most closely identify with." Anon

"We need to get a message to the general public as well as to user groups. Use more TV and popular media." Anon

"There is a great way to use timing as the attention getter. Use the free advertising available through TV and radio networks. By law they must devote portions of programming to free use." Dave Dubreuil

"Promotion of forest pathology will require an individual aggressive effort directed towards those whom we deal with on a common basis. Additional efforts can be directed towards situations and ideas as they arise." Paul Hinnan

"Who cares, who cares?! - A fundamental question underlying the entire session. Conclusions: Seize any opportunity to "witness the faith" without worrying about importance of the audience, i.e., whether J. Q. Public is more important than the foresters, etc." Anon

Have we finished this subject or did we just begin?...

Maybe we aren't finished with this discussion! We vacillated between assuming that special interest groups (forester, etc.) should be our main target to the idea that we need to address the general public more.

In a future session dealing with the "public" we should consider bringing in five "outsiders" to participate with us in discussions of public interest. An extension agent, politician, teacher, land manager or others would be good for starters.

Communication came up time and again. In fact we had to keep diverting to the true subject. People pointed out that we often don't do a proper job of communicating. Another session?

It might be interesting to see what we could do about integrating our discipline more with silviculture especially, and forestry education, in general. Maybe the entomologists are interested too....

It was obvious that everyone enjoyed the format of the session and appeared to have positive "vibes". Participation was uninhibited and continuous with numerous folk, including the normally quiet graduate students jockeying for the floor. The discussion brought people out of their "closets", putting them at ease, illustrating a need for more formats like this one.

One other reason the experience was rewarding was that several small arguments could be carried on within the group simultaneously.
with the main theme. This allowed some reinforcement dialogue before bringing up a subject to the group.

"As a newcomer graduate student to the group, I noticed that the inexperienced or otherwise bashful person who is normally quiet in another setting was as vocal as the oldtimers. Nobody seemed uptight." Valerie Scarpa
INTEGRATED PEST MANAGEMENT - A FAD, A THREAT, OR A TOOL FOR FOREST PATHOLOGISTS

The following comments were abstracted from the partially legible notes of Fields Cobb and the partially accurate recollections of the discussion leader ("leader" is perhaps a charitable appellation). No attempt has been made to credit individuals for the keen insights and wisdom that they shared with the group. This lack of acknowledgement stems mainly from an earnest desire to avoid a rash of angry letters alluding to misquotation, misrepresentation, gross incompetence, libel, exposure to ridicule, and other possible sources of odium. Let it suffice to record that the discussion was spirited, that a host of speakers contributed at various lengths and with various degrees of clarity, and that the reputation of WIFDWIC as a free-wheeling forum for untrammeled expression was not sullied nor diminished by the throng of participants.

Integrated Pest Management (IPM) has become a catch phrase in recent years. Government officials from the president (Carter) on down have proclaimed IPM to be Forest Service policy, and specifications to insure its application have been incorporated into the Forest Service Manual. Because of this current emphasis on IPM, it is important that pathologists understand what IPM is and what it means to us and to our profession. This no doubt inspired the inclusion of this topic on the WIFDWIC schedule.

Our discussion, begun nearly on time on a bright Thursday morning following an evening of California wine tasting made possible by the foresight of Bob Scharpf, first centered on (well, perhaps not so much on as more-or-less milled around in the general vicinity of) a definition of IPM and a brief examination of its historical setting.

Some speakers felt that the concept was as old as Hartig (Hartig was in May) and that forest pathologists have always practiced IPM, which was taken to be synonymous with incorporation of sound disease control into good forest management. Such a definition appears just slightly too narrow to work in motherhood and apple pie, but it certainly allows for the doctrine of pathological goodness. Others felt that IPM was a "gimmick" response to excessive reliance on insecticides and that it meant the use of several suppression tactics to control a disease or insect problem (or even the use of the same tactic by individuals of different ethnic, racial, or sexual persuasion). It was quickly pointed out, however, that integrated application of controls was at best only one small facet of IPM.

It is not practical to record here the definitions espoused by each of the contributors, since each definition was unique and probably representative of the fact that no concise, universal definition of IPM has been embraced by all workers. As pointed out by Stark (USDA, FOREST SERVICE General Tech. Rep. WO-14, 1979), "Defining IPM is somewhat like the six blind men of Indusitan describing an elephant - it all depends on where you are coming from".
Amalgamation of the comments of participants, especially those from Washington, D.C., suggests that IPM is a conceptual framework, a system to organize the collection of information and the incorporation of this information into the processes of management decision and of selection of research and development priorities in an orderly and obligatory fashion. This system ideally involves the thorough understanding of pest dynamics as they relate to forest stand dynamics and to impacts on management goals and forest values. Thus each stand prescription takes into account the probable impacts of disease, the probable needs for future suppression, and the probable costs in terms of values and of environmental goals for any of the available treatment strategies. Built into the system is a requirement for continuous monitoring to check the accuracy and efficacy of current information and methodology and to correct and refine our predictive capacity.

Assuming that the essence of IPM is somehow captured in the above welter of verbiage, is it a fad? My interpretation of what appeared to be nods of assent (or possible momentary lapses of consciousness) indicates that the group felt that the phrase IPM was rather faddish but that the concepts (under whatever acronym) and their applications are here to stay. The future of much forest disease research and "control" is intimately and irrevocably tied to IPM processes.

Is this apparently inextricable involvement of forest pathology with IPM a threat? If so, a threat to what or whom? The general drift (or thrust, if you like more assertive constructions) of the discussion was in the direction of "no". Some speakers recognized that some "pet" research projects have been torpedoed because they were not relevant to IPM needs. Others suggested that workers who are not "with it" may find it increasingly difficult to obtain support. We were cautioned to avoid promising too much under the IPM aegis and to be wary lest IPM preclude tactics such as might be needed in highly intensive management (as with Monterey pine in New Zealand). We were also cautioned not to kid the public by suggesting that IPM procedures are less expensive than traditional approaches. In general, however, the group appeared to consider the IPM approach as an asset, although the thought of working with entomologists seemed to elicit shuddering in a few participants.

Is IPM a forest pathology tool? If so, how do we make use of it? Again, the discussion leader could detect little opposition to the thesis that IPM is a valuable tool to guide both research and management. Properly applied, IPM forces us to consider systematically what information is most needed in order to make rational control decisions and evaluations. It provides a means of setting priorities.

In addition, integration implies the need to fit pathology into a broader context where work with other scientists (entomologists, ecologists, mathematicians, etc.) becomes not only desirable but essential. This provides the vehicle for broader, cooperative studies, and incidentally it also provides a framework for expanding the awareness of managers and other scientists of the prevalence and impact of diseases. It appears to your humble moderator that forest pathology and its practitioners can benefit greatly from an active role in IPM. Someone even suggested (rather
presumptuously) that educators could improve on the education of future foresters and scientists by recognizing and using IPM concepts and needs as educational frameworks.

Are there activities WIFDWIC should foster in response to or anticipation of IPM influences? Some members felt that we could help ourselves and others by inviting a few speakers from other disciplines (economics, entomology, etc.) to join us at our annual meetings. Panels of graduate students teams involved in interdisciplinary studies were suggested for the program. Everyone agreed (I think) that pathologists should move to their proper position at the forefront of developing IPM concepts and systems; that we should begin publishing in the IPM context; and that we should let it be known that some of the best working IPM systems (such as dwarf mistletoe management systems) have emerged from pathology.

Respectfully rendered and tendered,

Dick Parmeter
Technology Transfer Techniques, Examples and Ideas

David B. Drummond

Technology Transfer is a fad phrase; one used extensively lately. Does it stand for a concept or idea of substance. I believe it does. It represents a process that none of us are completely satisfied with and the present overuse of this term is an indication that deep-seated concern exists that important information is not reaching those who can use it best.

Today's discussion will not solve the problems in Technology Transfer, but it is hoped that some examples will be presented illustrating successes in this area and that we all will leave this room with new ideas to consider. The questions are easy. What is Technology Transfer? Who is responsible for Technology Transfer; the researcher or the extension specialist? Why are good examples of Technology Transfer hard to find? Is our reward system partially at fault for failures in this area? Why does so little Forest Pest Management (FPM) information go beyond in-house reports? Unfortunately the answers to such questions are more difficult to find.

Today we have four people who have consented to stimulate discussion and present what they think are successful examples of Technology Transfer. They are Bill Merrill, Penn State University; Art Partridge, University of Idaho; Dave Johnson, Region 2, FPM; and Ed Wood, Region 3, FPM. Bill Merrill could not be us; however, he has consented to allow me to read excerpts of an editorial on this subject that has been accepted for publication in Plant Disease, our new applied journal.

1 Survey Pathologist, USDA Forest Service, Forest Pest Management, Methods Application Group, Davis, CA 95616.
Research: Peddling the Product

W. Merrill

Information without application is sterile. Therefore, at least 50 percent of the phytopathological papers published in the past two decades are sterile--there are no immediate, or immediately foreseeable, applications. I am not saying that the contributing research was useless, because it probably had very important uses: (a) a basis for a thesis for an advanced degree, or (b) a basis to justify granting promotion tenure, or salary increment. But from the standpoint of production agriculture, the information is useless, at least at present. Applications may be found by future generations.

But consider the phytopathological research having immediate or foreseeable applications. Plant diseases have a direct and detrimental impact on the production and utilization of food, feed, and fiber, as well as on the aesthetic appearance of the landscape. Many producers are aware of this, and of the need to reduce losses to an economic threshold level. We have vital information. People should be pounding on our doors, clamoring for THE WORD. Yet often even the innovators and early adopters never hear of research results that could profoundly affect production practices. Why? Of course it can be argued (legitimately) that papers published for the "scientific world" are not intended to be read by anyone other than a few of the author's colleagues privy to the esoteric jargon and stilted, stereotyped style of such papers. Papers for the layman must be written with different style and emphasis. But far too few researchers ever take the time to write papers for a more general audience. Again, why? Simply because such papers earn few, if any, brownie points towards tenure, promotion, or salary increment. Indeed, it is precisely the fact that nonrefereed papers are almost totally disregarded by administrators that led to the demise of the late, lamented PLANT DISEASE REPORTER.

Another major contributing factor is the "publish or perish" syndrome, good in concept but detrimental in application. The problem lies in assessing the quality of research. The number of papers per year, rather than the significance of these papers, becomes the criterion of excellence. Petri plate, laboratory research is more paper-prolific than field research, and most young PhDs are astute enough to recognize which side of the bread is buttered. Hence, the high proportion of nonapplicable research.

Usually the writing of popular articles explaining research or relating it to practice is left to extension specialists, or worse, to textbook writers. But how can one or two people per state handle the bulk of material emanating from research laboratories dealing with all manner of diseases on a vast array of cultivated and noncultivated plants? The answer is simple: they can't, and they don't!

1 Excerpted from an unedited manuscript submitted and accepted for publication in Plant Disease.

2 Professor, Plant Pathology, Pennsylvania State University, University Park, PA 16802.
Researchers shy away from the public, and those who don't often are ostracized by their peers. Most scientists feel that communicating with the general public "taints" them that somehow it is "unprofessional." Many suffer from the von Braun Syndrome: "That's not my department!" Most think the public doesn't care, or won't understand. Perhaps the latter is due in great part to the method of peddling the product.

In the thinking of many extension specialists (and virtually all agricultural administrators) a one-page mimeo "fact sheet" naming a disease and briefly describing the current control recommendations is the epitome of SPREADING THE WORD. We have progressed little beyond this in the past 50 years. Only relatively recently have a few stations used color photos, and few offer for-sale publications. The consumer is well aware that you get what you pay for. Single-page mimeos are not highly regarded, have little lasting impact, and generally are of no use or interest to the general public. With an audience thoroughly conditioned by the advertising media to expect slickly done, professional communications, agricultural communications are unappealingly kindergardenish, at best. But if the packaging is professionally well done, if the message is interesting and intelligible, the general public will beat a path to the door, and not quibble about paying for the product. A case in point:

Heartrot of trees is of limited interest, even among forest pathologists. The concepts of succession of microorganisms in the decay process and of compartmentalization of infected tissues, developed by Dr. Alex Shigo, are, admittedly, somewhat esoteric. But the results of his research were attractively and professionally packaged into a series of five booklets beginning with "A Tree Hurts Too" and culminating in "An Expanded Concept of Tree Decay", three sets of slides with explanatory text illustrating the concepts and processes and how to treat trees, and posters describing how or how not to treat a tree. The booklets have truly excellent artwork and a limited yet clear and concise text readily understandable by a ten-year old. And these have been the fastest moving items ever published by the US Forest Service! "A Tree Hurts Too" has gone through six printings (over 100,000 copies) and has been translated into Spanish and German; total distribution of the five booklets has exceeded 500,000 copies; over 1000 slide sets have been sold; over a million posters have been distributed. In years to come Shigo will be recognized as much for his contributions to packaging pathological research information as for the research itself.

3 From the song "Wernher von Braun" by Tom Lehrer.

The views expressed are solely the author's and in no way reflect the views of the Department of Plant Pathology or the College of Agriculture, The Pennsylvania State University, nor the views of the author's wife, children, relatives, in-laws, colleagues, friends, or casual acquaintances.
SUMMARY COMMENTS FOR WIFDWC

TECHNOLOGY TRANSFER TECHNIQUES, EXAMPLES AND IDEAS

A.D. Partridge

Why not use the "tree doctor" image. Ordinary people can understand the term so let's capitalize our advantage. I think we present our subject blandly. A simple brochure like "Why's My Evergreen Brown" sells. We reprinted this three times by demand. Somewhere around 30 thousand copies went out. I maintain that insufficient information transfer occurs mainly because you and I don't really try either to get it out or find how to improve the ways we get it out. What about demonstration plots with signs, campground signs showing what diseases are there, "What's wrong with my tree" displays at fairs and shopping centers. And what about direct contacts developed by showing a willingness, no, an eagerness to help people with tree problems: the individual, woodland, foresters, extension agents, city foresters or arborists, forest technicians, rangers; anyone who's interested. We need salesmanship.

Let's not leave out some excellent media: picture books, comics, brochures, training manuals, simplified keys, a comprehensive article on all tree problems in the Journal of Forestry or American Forests. With help, anyone can simplify his subject so people will want to listen. You have all sorts of help in advertising specialists, editors, publicity, bureaus, professional illustrators, photographers, etc., but you'll never move forward until you are willing to accept their help and break the shell of indifferent excuse-making. Yes, it's work and it may make some of us "retread."

1 Professor, Forest Pathology, University of Idaho, Moscow, ID 83843.
"TECHNOLOGY TRANSFER - OPPORTUNITY OR FAD"

David W. Johnson

We hear a lot nowadays about "technology transfer". Is it just a fad or does this present an opportunity for us? Evidently this subject has been and still remains a popular topic for discussion since several past work conferences have dealt with this topic and it has also been discussed at the Dwarf Mistletoe Symposium held in 1978.

Today, I view my role on this panel discussion of technology transfer as agitator, stimulator or facilitator. I am hopeful that the few comments I make will generate some discussion of this topic, which I view as a key to our survival in this day of inflation, program cutbacks and restrictions. We need to emphasize the importance of our work and gain the support of forest resource manager and legislator alike.

During this period of intensive activity in forest land management planning, we need to make our concerns known to the planners. What is being developed and written down in these plans will influence forest management and forest disease management activities in the future. Throughout the country, our staffs are confronted daily by this planning effort and many offices have assigned entomologists and pathologists to these planning teams as subject specialists.

You may ask what does all this have to do with the subject "technology transfer". A good question! I view this period as an opportunity to really sell our product—whether it be forest disease research or forest disease management action programs. The ultimate recipient of our knowledge and programs is the forest resource manager or private landowner. They need to be convinced of the worthiness of our work and appreciate the impact of disease pests on management of the forest resource.

The overall Forest Service responsibility for the prevention and suppression of damage by forest pests is shared by Forest Pest Management (FPM) and Forest Insect and Disease Research (FIDR). Together, we have joint responsibility for the development of control methods for forest pests. Each organization has its separate and joint responsibilities and programs. Cooperation is essential to obtain the answers to new problems and put them into action. FPM is both a user and disseminator of research results. We are the principal contact and information source for all federal agencies dealing with insect and disease problems. We also serve the needs of the state and private forest landowners through the state foresters. FIDR has the major responsibility at the research end of the research and development process, and FPM has the dominant role at the action end. Research must be responsive to FPM's needs for information. We are heavy users of research results since we represent not only the needs of federal agencies, but also the states and private industry.

1 Supervisory Plant Pathologist, FPM, S&PF, R-2.
To meet our responsibility, FPM must fully utilize all available technology on pest control techniques and strategies. Knowledge and methodology must be brought together and adapted to specific field problems if FPM is to carry out an effective program of reducing forest pest losses. In turn, research benefits from a wide variety of field observations and expertise that FPM can provide.

What has been done so far to breach the technology gap?

We have a number of excellent examples of methods to use to reach the users produced by FIDR, FPM and state forest agencies:

- "How To" series produced by the North Central Forest Experiment Station and Northeast Area State and Private Forestry.

- Guides to identify and manage insect and disease pests - several available - emphasizing regional pest problems.

- Popularized publications on pests - dwarf mistletoe, mountain pine beetle, discoloration and decay.

- Annual summaries of major forest pests - regional and U.S.

- Film - 16mm film entitled "Dutch elm disease - catalyst to Community Forestry".

- Demonstration projects - the federally-funded DED demonstration projects conducted in California, Georgia, Minnesota, Wisconsin and Colorado.

- Workshops, training sessions, and symposia - FPM workshops or training sessions given yearly to federal and state land management agencies.

The Dwarf Mistletoe symposium invited research, pest management and resource managers.

Despite these examples, I feel we need to improve information dissemination and ask you how or what techniques you are using to reach your publics?
TECHNOLOGY TRANSFER: WHY IT DOESN'T WORK

Ed Wood

Fortunately almost all of the points I wanted to have discussed have been brought out by one of the previous speakers or by some of you in the audience. I am left with just two items that are pitfalls in the movement of knowledge gained through research toward its eventual user, in our case, forest land managers. They are:

1. Research results are published in a form which is aimed at the wrong audience. Part of this problem, publishing in a journal or other outlet which is not read by the proper audience, we talked about earlier. I am referring more to the content of an article, or other forms of publication, which is written in language or format that is difficult for an audience to use. As a result, many research results are ignored or misinterpreted by the appropriate audience. For instance, RMYLD is a powerful tool used to select dwarf mistletoe control options. The publication describing the program is written by computer specialists for other computer specialists; there is a great need for a publication aimed at the practicing forester who has little knowledge of computer language, etc., but who needs a lucid, step-by-step "cookbook." As a result of the lack of such a publication, we conduct endless training sessions on a one-to-one basis.

2. The lack of followup. Far too many of our projects, designed to transfer knowledge to land managers, lack a built-in followup process. How often do we followup on our work, advertising, or modifying, or extending, or heaven forbid, withdrawing unsuitable materials. For instance, an excellent training package, consisting of slides, specimens in Riker Mounts and vials, and other self-teaching aids, was prepared by a pest management staff. It was distributed to all National Forests in the Region, and was used for several years. Then, because of personnel transfers, deterioration of specimens over time, "borrowing" of parts of the package, and probably a number of other reasons, the remaining packages are seldom if ever used. If newly-transferred personnel in the pest management staff and on the forests had been informed about the packages, if the packages themselves had been reviewed every few years, I think the training package would be widely used today. But because of the lack of followup, this project, which was so well done, has now disappeared into history.

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1 Supervisory Plant Pathologist, FPM, S&PF, R-3.
The first national symposium on forest pathology and entomology was held in Uruapan, Michoacán on February 18–20, 1980. Mal Furniss, Forest Entomologist with the Intermountain Station and I were the only "gringos" among the 300 or so in attendance. About 35 papers were presented, including almost half on forest disease problems. Forest diseases discussed were: blue stain fungi associated with Dendroctonus, Fomes annosus, Cryptoporus volvatus, damping off control in pine seedlings, nematodes of forest trees, mycorrhizal fungi, cone rust, and dwarf mistletoes. A field trip was made to the vicinity of Paracutin volcano which erupted in 1943. Many square miles of forest were killed by ash but these have essentially all been reforested naturally with pines. Some "new" forests of Pinus montezumae are already being harvested (these 30-year-old stands have trees up to 50 feet high and over 1 foot in diameter). There is hope for Mt. St. Helens!

This was an excellent symposium and Mexican foresters and pathologists are anxious to cooperate with their counterparts "north of the border". The proceedings from this symposium will be published. Plans are underway for a second symposium but the time and locale have not yet been decided.

FRANK G. HAWKSWORTH, Rocky Mountain Station, Fort Collins
WFDWC 1980

DMLOSS:
A SIMULATOR OF LOSSES IN DWARF MISTLETOE INFESTED BLACK SPRUCE STANDS

F.A. Baker, D.W. French, and D.W. Rose
Department of Plant Pathology
And
College of Forestry
University of Minnesota

The eastern dwarf mistletoe, Arceuthobium pusillum Peck, is the most serious disease of black spruce, Picea mariana (Hill.) BSP., causing deformity and rapid mortality. This parasite is well distributed throughout the 1.6 million acres of commercial black spruce in Minnesota (14). Anderson (1) estimated that 3-11% of the black spruce area in the Big Falls Management Unit was infested with dwarf mistletoe. This disease problem is not unique to Minnesota, as dwarf mistletoe occurs throughout the range of black spruce (7).

Black spruce is the most valuable tree in Minnesota on the basis of total dollar income. The annual allowable cut for this species based on area control, is being exceeded. Stands with little or no dwarf mistletoe are harvested in preference to those with dwarf mistletoe. Thus, the future demand for black spruce must be met from diseased stands, i.e., stands with less merchantable volume per acre. Forest managers are aware that dwarf mistletoe is a serious pest, but they have no means to predict losses to justify the cost of control operations.

Models simulating effects of dwarf mistletoe in forest stands are relatively recent developments. LPMIST (10) and SWYLD2 (11) are simulators that compute yield tables for lodgepole pine (Pinus contorta Dougl.) and ponderosa pine (P. ponderosa Laws.), respectively. Effects of dwarf mistletoe on height growth, diameter, and tree mortality are quantified based on average stand dwarf mistletoe rating (DMR) (6). These three variables are subsequently used in equations predicting volume per acre. Each of these simulation programs has routines which project increases in DMR and other stand variables for 10-year intervals to rotation age. Recently, these two simulators were combined into a larger program called RMYLD (6). In RMYLD, dwarf mistletoe intensity can be entered as the percentage of infected live trees, or as the average age at the time of initial infestation, as well as the average stand DMR previously mentioned.

The use of these simulators is limited to uniform stands, that is, the stand simulated must have a uniform DMR, because the program does not allow for lateral spread of the parasite. One means of circumventing this is to divide the stand into uniform portions, and simulate each portion.

A model proposed by Strand and Roth predicts lateral spread of dwarf mistletoe in young, evenly-spaced stands of ponderosa pine (13). Recently, Bloomberg, et al. (3) published a model of spread and intensification of dwarf mistletoe in young stands of western hemlock.
Both groups used the approach of components analysis, which provides insight into the infection cycle of the parasite. These models, however, have little potential for use by forest managers, because their use is limited to stands of uniform spacing, height, and dwarf mistletoe intensity. The basic unit in these models is the individual tree, which provides a wealth of information for plant pathologists, but provides excessive and costly detail for the forest manager.

A simulator which predicts spread and intensification of dwarf mistletoe (Arceuthobium vaginatum subsp. cryptopodum [Engelm.] Hawks. and Wiens) in stands of ponderosa pine has recently been developed by Dixon and Hawksworth (4). This model uses overstory diameter breast height (DBH), average DMR of all overstory trees, overstory density, and infection age to predict spread of dwarf mistletoe through an even-aged stand. Spread through two-storied stands is predicted using the parameters listed above for each story and the site index. Intensification is expressed as a function of time (years) since initial infection. Proportion of trees infected increases linearly from 0 to 1 within 35 feet of the infection source.

The program SPREAD uses these three equations to project dwarf mistletoe spread and intensification. Present stand and site variables are used to project stand density, average DBH, area of infestation, and average DMR for the infested portion of the stand, as well as stand density, average DBH, and area of the uninfested portion of the stand.

In spite of the inability to directly accommodate multiple infection centers, SPREAD will be a very powerful tool when incorporated into RMYLD. SPREAD separates the stand into infested and uninfested portions, allowing for improved accuracy over that obtained with RMYLD, which averages DMR over the entire stand.

A simulator useful to forest managers must meet the following criteria:

1) Utilize input which can be obtained with minimal effort;
2) Predict spread and intensification of dwarf mistletoe in the stand;
3) Accommodate stands of different age, density, site quality, and number of infection centers;
4) Project future losses to dwarf mistletoe.

Of the models discussed so far, only SPREAD, once it is incorporated into RMYLD, can meet these criteria. Using these criteria as guidelines, a model simulating dwarf mistletoe effects in black spruce stands has been recently developed.

LMLOSS - A Simulator of Dwarf Mistletoe in Black Spruce Stands

As discussed earlier, three relationships must be described mathematically prior to model development: spread, intensification, and growth loss. Spread of Arceuthobium pusillum in black spruce stands averaged 2.42 ft/year over a 13-year-period (2, 8). Site index on plots
where these observations were made, ranged from 21 to 42 feet, while basal area ranged from 40 to 250 ft$^2$/ac and stand age ranged from 60 to 130 years. Spread of $A.\ vaginatum$ in ponderosa pine stands has been shown to be a function of stand age, density, and site index. Data are not available for examination of the effects of these stand parameters on rate of spread of $A.\ pusillum$ in stands of black spruce. The 2.42 ft/year figure represents the rate of spread averaged over these conditions. It is possible that the actual rate of spread may deviate from 2.42 ft/year. The effect of these deviations on the model output is discussed in a later section.

Intensification is the second relationship which must be quantified. The six-class dwarf mistletoe rating has been used to rate disease intensity in other tree species - dwarf mistletoe systems (6). The percentage of infection in each third of a tree crown is evaluated, and rated as a 0, 1, or 2, and the final score computed by adding the rating for each third of the crown. This system has not been used for black spruce, because dwarf mistletoe causes a rapid decline of the trees, far more rapid than in dwarf mistletoe hosts in the western United States. $Arceuthobium\ pusillum$ kills uninfected portions of a crown before it kills the witches' brooms (infected portions). Because infection is computed as a percent of live crown, the DMR can change from 1 to 6 as a tree declines, with little or no increase of the parasite. In contrast to many tree host-dwarf mistletoe combinations in the western United States, where DMR increases by 1 class in approximately 10 years, $A.\ pusillum$ may kill the tree within a short time, before new infections can manifest themselves. An average of 8.02% of the infected black spruce trees died each year on permanent plots (2). Using a simple interest disease model, the entire population of infected trees would die within 12.5 years. Trees which have been infected for periods longer than 12.5 years are commonly observed, suggesting that a compound interest disease model may be more appropriate. Although 75% of the initial population would die within 16.58 years, 0.01% could remain alive after 55 years. These results are more consistent with those observed in nature. In the model, the time used for 100% mortality is 16.58 years. This was obtained by doubling the time for 50% mortality. Although the compound interest disease model predicts that 25% of the trees will be alive after this time, many of these trees will be heavily broomed and unmerchantable. Also, mortality will have reduced the density of surviving merchantable trees to a level where a logger will not bother to harvest them. Thus, although there is volume present, it will not be harvested. It is this concept of merchantable volume which is used in the model.

The third relationship which must be quantified is growth loss. It has not been possible to demonstrate growth loss caused by $A.\ pusillum$, presumably because trees are killed before their diameter growth differs significantly from uninfected trees. Thus, volume loss is a function of mortality and deformity caused by witches' brooms. In DMLOSS, only volume lost to mortality is computed, since the volume loss to deformity has not been quantified. This introduces a bias in DMLOSS, resulting in underestimates of volume lost to dwarf mistletoe.
MODEL OF SPREAD AND MORTALITY

The areas of mortality, infestation and control are assumed to be circular (Figure 1). The initial radius of mortality, measured on an aerial photograph or ground transect, is used to compute radii of infestation and control. The radius of infestation is derived by reasoning that at some time in the past, the edge of the infestation coincided with the current area of mortality. It can be argued that this occurred 16.58 years ago, because 100% of the trees in the area of mortality are now dead. During those 16.58 years, the parasite was moving outward at 2.42 ft per year, thus the area of mortality and the edge of the infestation should be separated by 40.12 feet. This distance, measured in four infection centers, ranged from 30 to 42 feet. The agreement between the calculated and observed distances suggests that 16.58 years is a reasonable approximation of the time for 100% mortality. The 40 ft. distance from the area of mortality to the edge of the infestation is close to the 35 ft. distance from 0 to 100% infection reported by Dixon and Hawksworth for Ponderosa Pine infested with A. vaginatum subsp. cryptopodum(4). The radius of infestation is computed by adding 40.12 feet to the radius of mortality.

Current control practice is to utilize control procedures 1-2 chains beyond obvious infection, to remove latent infections. The radius of control is obtained by adding the width of this control buffer to the radius of infestation. The computer program uses these radii to compute the respective areas. Spread of dwarf mistletoe is projected by increasing the radius of mortality by the product of the rate of spread and the length of the simulation interval (years).

MODEL ASSUMPTIONS

Several assumptions are necessary for this model. First, the rate of spread is assumed independent of stand age, stand density and site index. The relationship of these factors to the rate of spread has not been described. The rate of spread used in the model, however, was estimated from stands representing a wide range of these conditions. The effects of deviations from the spread rate used in the model on model predictions were examined using sensitivity analysis, and are discussed later.

A second necessary assumption is that the time for 100% mortality is approximated to be 16.58 years. As discussed previously, even though infected trees may survive longer than this, it is unlikely their volume will be harvested. The close agreement between the calculated and observed distances from the area of mortality to the edge of the infestation supports this assumption.

The model assumes that mortality spreads through the stand at the same rate as the parasite. This seems valid, since the distance from the edge of the infestation to the area of mortality is similar in most stands. It is also assumed that the parasite (and mortality) spreads outward from infection centers at an equal rate in all directions. One need only examine the more or less circular pattern of dwarf mistletoe...
infection centers on aerial photographs to accept this concept.

During simulation, no allowance is made for the appearance of new (satellite) infection centers. These centers do occur, but the information needed to quantify their occurrence is lacking. This results in model output biased toward underestimating the effects of dwarf mistletoe.

Program Inputs, Computations and Output

The computer program, named DMLOSS, is written in standard FORTRAN, and requires 56,000 words of central memory storage for execution. DMLOSS is designed to be run from an interactive terminal, but with slight modifications, could be run in a batch (card) environment.

The stand is defined within a coordinate system of 10,000 points, 100 rows and 100 columns. The distance between grid points is set by the variable SCALE. Coordinates of stand boundaries are obtained with a dot grid from aerial photographs and supplied to the program as a list. In future versions, a digitizer will be used to trace stand boundaries from aerial photographs and to communicate the coordinates directly to the computer. The coordinate of the center of each infection center is input, along with the current radius of mortality. This information is also obtained from the aerial photograph. Stand age, rotation age, stand basal area, stocking, site index and cost parameters (stumpage value, discount rate, and control cost) are input. DMLOSS then compares the distance from each grid point in the stand to the center of infection with the radii computed earlier. If the distance is less than the radius of control, that point is compared with the radius of infestation. If the point distance is greater, the point is in the area of control; if less, the point is compared with the radius of mortality. Similarly, if the point distance is greater, the point is in the area of infestation; if less, the point is in the area of mortality. If multiple infection centers are specified (DMLOSS will accommodate up to 10 infection centers), points are also compared with other radii, each time they fall within the radius of control, to eliminate double counting. When all points have been checked, the point count for each area is multiplied by an area factor (= SCALE^2/43560) to give the area of mortality, infestation, or control. These areas are subsequently used in volume and cost computations.

Output from DMLOSS consists of three tables. The first is a summary of input variables: stand name, size (acres), site index, stocking (%), basal area (ft^2/ ac), stumpage value ($/ cord), control cost ($/ ac), discount rate (%), and acres per stand point (Table 1a). This table allows the user to store on a single computer page the output generated by the model as well as the inputs used to obtain it.

The second table contains information about the impact of dwarf mistletoe in the stand. Stand age, volume present, volume lost to mortality caused by dwarf mistletoe, areas of mortality, infestation and control, discounted control cost, discounted value loss, and present stand value are printed at 10 year intervals (Table 1b). Volume per acre is computed using the basal area in living trees and in trees which have
died in the past 5 years from causes other than dwarf mistletoe (Equations 4, 7, and 8 in Peralta [12]). Volume present is the product of volume per acre and the difference between the area of mortality and the stand area. Volume lost is the product of volume per acre and the area of mortality. Discounted control cost is calculated by multiplying the area of control by the control cost and discounting to the initial age. Discounted value loss is the product of the volume loss and the stumpage value, again discounted to the initial age. Present stand value is the discounted value of the volume present minus discounted control costs.

The third table of output from DMLOSS (Table 1c) contains information used in decision making, again printed for 10 year intervals. Volume with control is the sum of the volume present in the stand and the volume salvaged during control operations. Salvage volume is computed by subtracting the area of mortality from the area of control, and multiplying by the yield table volume for that age. In stands less than 90 years old, there is no salvage volume, because the products are not of merchantable size. Volume saved is the difference between volume with control and volume without control.

The value of net savings is calculated by multiplying the difference between volume saved and volume salvaged by the stumpage value, and discounting, adding the discounted value of salvaged volume, and then subtracting discounted control costs. The index of control priority is the ratio of net savings to control costs; that is, it is the return on the investment for control after control costs have been paid for, divided by the control costs. Selecting stands for control with the highest index of control priority will maximize income from a fixed control budget. In Table 1c, at period 4 (age 90), the index of control priority becomes positive. This is due to salvage volume become in merchantable.

DMLOSS assumes that the area of infestation is fully stocked. This is not the case in nature, but no relationship quantifying stocking is available. If we assume the area of infestation is 50% stocked (this is an underestimate due to the exponential nature of the compound interest mortality function), errors will be manifested in volume salvaged (and consequently volume with control) and in volume without control. However, carrying these errors through results in overestimates in net savings of $43.70 and $33.37 at 90 and 100 years, respectively. By inspection, these errors do not affect the economic feasibility of control, but only affect the return on the investment.

The cost of using this program can be separated into three areas: program storage, compilation, and execution. It costs $.17 per week to store DMLOSS in the computer, and $.20 to compile this program. Once operational, it is recommended that a binary version of DMLOSS be stored. This costs $.09 per week in storage fees, and eliminates compilation costs. Binary versions of the program, however, are unintelligible, which makes changes in the program virtually impossible.

Execution costs vary with the stand being simulated, but commonly range from $.15 to $.35.
Sensitivity Analysis

In a previous section, several assumptions were stated concerning rates of mortality and spread. It is important to know what effect violations of these assumptions have on model output. For example, what effect do changes in the rate of spread as a stand ages have on model output? In other words, what if the average rate of spread is different from that used in the model? A sensitivity analysis was performed to attempt to answer these questions.

As previously mentioned, the model predicts areas of mortality, infestation, and spread. It is these areas which will first be affected by violated assumptions. In addition to rates of spread and mortality, the variable SCALE could conceivably affect area determinations. If SCALE were too large, mistletoe spread could occur during the simulation period, but not be measured because the stand grid points are too far apart.

Output from DMLOSS was obtained using a factorial design with rate of spread set at 1.0, 1.5, 2.0, 2.5, 3.0, 3.5, or 4.0 feet per year, rate of mortality set at 6, 7, 8, 9, or 10%, and SCALE set at 10, 20, 30, 40, or 50 feet. The stand simulated was 17.22 acres with one infection center 0.10 acres in size at age 60. Rotation age was set at 120 years (See Table 1). Regression equations predicting area of mortality or infestation on age, rate of spread, rate of mortality, and scale were obtained using the multiple regression package, Multreg (School of Statistics, University of Minnesota). Predictions at age 70 and 100 were eliminated from the data set because Multreg can only process data sets with less than 1,000 observations. Resulting equations were:

\[(1.) \quad A_m = 0.0454885 \times A + 0.6963903 \times S - 4.634568 \]
\[r^2 = 0.7769 \quad \text{Standard error of prediction} = 0.64\]

\[(2.) \quad A_I = 0.0657656 \times A + 1.421615 \times S + 0.1405334 \times M - 8.516455 \]
\[r^2 = 0.7986 \quad \text{Standard error of prediction} = 1.04\]

where

\[A_m = \text{Area of Mortality}\]
\[A_I = \text{Area of Infestation}\]
\[A = \text{Stand Age}\]
\[S = \text{Rate of Spread}\]
\[M = \text{Time in years for 50% Mortality}\]

All terms were significant at \(P = 0.01\).

These equations provide information about the response of the model to changes in the variables. The coefficient of each variable is the amount the dependent variable (area) will change with a change of one unit in that variable. For example, in equation 1, area of mortality will increase by 0.696 acres for each increase of 1 foot in the rate of...
spread. Equation 2 is more sensitive to changes in rate of spread because this variable not only affects the radius of mortality (from which radius of infestation is calculated), but also the distance through the stand that trees have become infected.

Each one year increase in the time for 50% mortality causes an increase of 0.14 acres in the area of infestation, but this term has no significant effect on the area of mortality. Mortality rates of 6, 7, 8, 9, and 10% have times for 50% mortality (computed using a compound interest disease model) of 16.58, 11.42, 8.31, 6.58, and 5.42 years, respectively. Thus, rates of mortality greater than 8% have declining importance, while those less than 8% have increasing importance.

The coefficient of stand age is small because of the 10-year simulation period. This coefficient actually predicts the increase in area when all other variables are held constant.

Although data on factors causing variation in the rate of spread and the rate of mortality are limited, the effects of these deviations are known. The acceptibility of potential errors is related to the costs of undertaking control operations when they are not justified and to the costs of not using control measures when their use was justified. Simulations using a rate of spread of 1 foot and a rate of mortality of 10% show that dwarf mistletoe control is profitable, but does not offer as great a return on the initial investment (Table 1c). (Under these conditions, dwarf mistletoe spread is low, and the area of infestation is minimal, which results in decreased control costs.)

DISCUSSION

Using the relative annual mortality rate of dwarf mistletoe infected black spruce and the rate of spread, a model was constructed which projects future spread of dwarf mistletoe. With the incorporation of this model into DMLOSS, it is now possible for the forest manager to predict future losses to dwarf mistletoe. The response of the model to variation in rate of spread and relative annual mortality rate were examined using sensitivity analysis. Even under conditions where dwarf mistletoe caused minimal impact, investments in dwarf mistletoe control yielded a substantial return.

DMLOSS has not been validated. Model validation is a time-consuming, ongoing process. Where bias in the model is known, the tendency is to underestimate losses to dwarf mistletoe. Work is in progress to validate the model by comparing the increase in the area of mortality projected by DMLOSS with that measured on sequential aerial photographs of the same stand. This technique was previously used by Meyer and French to measure spread of this disease (9). The initial radius of mortality was obtained from 1952 aerial photographs, and DMLOSS was used to project the area of mortality in 1972. DMLOSS underestimated the area of mortality measured on 1972 aerial photographs by 25 to 50% in the three stands tested. If dwarf mistletoe control yields a positive return on the investment when losses are underestimated, then it follows that dwarf mistletoe control should be more profitable than projected by the model. Thus, although DMLOSS has not been validated, it suggests that
Dwarf mistletoe will cause substantial losses if left unchecked, and that control of this parasite is economically feasible.

Besides using sequential aerial photography, further attempts to validate DMLOSS should examine the effects of stand age, stand density and site quality on the relative annual mortality rate and the rate of spread. This should be done, directly, on permanent plots, and, indirectly, by measuring the distance from the area of mortality to the edge of the infestation.

Currently, the impact of dwarf mistletoe in black spruce stands throughout Canada and the United States is unknown. We believe DMLOSS, when coupled with a digitizer, could be used to inventory all black spruce stands in selected management units to provide estimates of current and future losses. This would permit the determination of allowable cut on a volume control basis, which is more accurate than the area control method currently in use. More important, however, forest managers would have information to aid in planning and justifying a dwarf mistletoe control program. Then, with a given level of resources available for mistletoe control, DMLOSS could be used to select the stands to receive treatment.
Figure 1. Schematic diagram of an infection center showing radii and areas of mortality, infestation, and control.
LITERATURE CITED


### TABLE 1. OUTPUT FROM IMSLOSS

#### A.

**STAND ACREAGE = 25.78**  
**SITE INDEX = 35.00**  
**CONTROL COST = 100.00**  
**DISCOUNT RATE = .08**

#### B.

<table>
<thead>
<tr>
<th>STAND AGE</th>
<th>STAND VOLUME</th>
<th>VOLUME LOST</th>
<th>VOLUME MORTALITY</th>
<th>AREA OF INFESTATION</th>
<th>AREA OF CONTROL</th>
<th>DISC'TD VALUE</th>
<th>DISC'TD PRESENT STAND</th>
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<td>0</td>
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<td>.36</td>
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<td>298.91</td>
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<td>.78</td>
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<td>3.47</td>
<td>6.23</td>
<td>6.14</td>
<td>11.24</td>
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</tbody>
</table>

#### C.

<table>
<thead>
<tr>
<th>STAND AGE</th>
<th>ACRES OF CONTROL MORTALITY</th>
<th>VOLUME AT ROT. WITH CONTROL</th>
<th>VOLUME SAVED CONTROL</th>
<th>CONTROL COST</th>
<th>NET SAVINGS</th>
<th>INDEX OF CONTROL PRIORITY</th>
</tr>
</thead>
<tbody>
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<tr>
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<td>37.92</td>
<td>169.68</td>
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</tr>
<tr>
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<td>779.54</td>
<td>33.50</td>
<td>37.92</td>
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<tr>
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<td>779.54</td>
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<td>12.18</td>
</tr>
<tr>
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<td>6.23</td>
<td>999.54</td>
<td>779.54</td>
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<td>6.16</td>
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</table>
Intensification is the rate at which the number of mistletoe plants increases over time. Normally it is expressed on a per tree basis because that is the way the data is collected. A good argument can be made for expressing the number on an area basis, particularly in even-aged dense stands.

Estimates of intensification of hemlock dwarf mistletoe have been made using at least two different techniques. Firstly it has been done by destructive examination of infected trees, counting and establishing the age of all infections. Such examination yields a curve of number of infections over infection age. A logarithmic transformation allows for easy extrapolation and prediction of doubling time. A second and more reliable technique involves remeasurement of infected trees to determine the increase in the number of infections over time. The difficulty with the latter technique is that it can only be applied realistically to young small trees. The result is that intensification is measured over a time period during which the total crown volume per ha. is still expanding rapidly, and a relatively large portion of the crown is exposed to light. Thus one would expect the percentage of seed reaching a suitable host to increase over the period of observation, and seed production to be rather high because most infections would be located in exposed parts of the crown. It follows that the rate of intensification so
established may not be a good predictor of events later in the life of the stand.

In the case of western hemlock the doubling times predicted by either technique are of the order of 2-5 years. Such a rate of intensification yields astronomical numbers of infection per tree at rotation age, i.e. number in excess of 1,000,000 infections per tree. While it is true that occasional open grown trees may reach very high levels of infection, the predicted levels are at least two orders of magnitude in excess of those observed in most older infected hemlock stands. In a recent attempt Bloomberg has constructed a model to predict hemlock mistletoe spread and intensification based on a number of experimentally derived relationships. While this model shows a marked reduction in the rate of intensification after crown closure, it still predicts very high numbers of infections.

Today I want to report on an investigation by Ted Wilford. What we tried to do is to locate two stands with very similar infection histories but of different ages. Figure 1 shows the location of the two stands on either side of an old fire boundary. Table 1 gives some of the stand characteristics. The older stand was chosen with great care in order to make sure that it had been infected early on. The evidence for early infection consisted of old but undatable large bole infections within 2 m of the ground, and old mistletoe brooms on long-dead branches within 4 m from the ground. Some of the lower leafless branches with dwarf mistletoe less than a metre from the bole turned out to be alive, and the oldest infection we were able to date was 92 years old. We assumed therefore that the two stands represented a continuum.
The younger of the two stands was chosen to be old enough to have achieved full canopy closure so that the total crown volume would remain relatively constant up to the age of the older stand.

| TABLE 1 |
|-------------------|------------------|
| **BASIC STAND DATA** | **STAND 1** | **STAND 2** |
| **STAND AGE**     | 44              | 130           |
| **PLOT AREA (ha)** | .01             | .025          |
| **TOTAL NUMBER OF INFECTIONS** | 278             | 413           |
| **AVERAGE NUMBER OF INFECTIONS PER TREE** | 46.3            | 83.5          |
| **AVERAGE TREE HEIGHT (m)** | 20.8            | 38.1          |
| **SITE REX (CH at 100)** | 41              | 30            |
| **AGE OF OLDEST DATED INFECTION** | 20              | 92            |

Having picked the sampling plots, we then proceeded to examine these destructively collecting and dating all live mistletoe infections. Figure 2 shows the number of infections over infection age for the younger stand. The trend here indicates a doubling time of about 5 years. Extrapolating this rate to the age of the older stand would yield in excess of 10,000,000 infections per tree. Obviously something is wrong with this interpretation. Figure 3 shows the number of infections over infection age for the mature stand. The curve is remarkably similar to that for the immature stand except for
the long asymptotic tail.

The curve may be divided into two sections. If this is done, the doubling time indicated by the younger set of infections is again about 5 years, while for the older infections it is 41 years.

We believe that the correct interpretation of these data is that the relatively high frequency of young infections is due largely to mortality of infections. The bulk of this mortality is due to branch senescence at the base of the crown and reduction in the number of trees per acre over time. Some mortality also occurs within the green crown of host trees.

Let us look again at the younger stand data now redrawn as number of infections over year (date) of establishment. (Figure 4). The dotted line indicates a straight extrapolation of the curve and represents an estimate what we would expect to find in 1985 if there were not mortality. In fact we suggest that in '85 the pattern would resemble that indicated by the dashed line. If we were to plot total number of infections over stand age, we would expect a pattern such as illustrated in Figure 5 with a marked levelling off in number of infections per ha. following crown closure. If this interpretation is correct, and particularly if the assumption that the two stands examined form a continuum in valid, then it may be concluded that hemlock dwarf mistletoe will not easily reach damaging levels in immature hemlock stands on medium to good sites with closed canopies and no overhead source of infection.
Fig. 2  Total number of Dwarf mistletoe infections over infection age in the 44 yr old Hemlock stand.

Fig. 3  Total number of Dwarf mistletoe infections over infection age in the 130 yr old hemlock stand.

Fig. 4  Two intensification models for Hemlock Dwarf Mistletoe.

Fig. 5  Intensification of Hemlock Dwarf Mistletoe
VERTICICLADIPELLA SPECIES AND ASSOCIATED ORGANISMS CAUSING BLACK STAIN ROOT DISEASE OF INLAND NORTHWEST PINES

By

C. L. Bertagnole and Dr. A. D. Partridge

We, at the University of Idaho, have studied a black stain of roots caused by two or more organisms for about ten years. During this time, we located study plots by random odometer readings and by the presence of symptomatic trees. These plots were scattered throughout Idaho, western Montana, Washington, and northern Oregon. Random plots gave an indication of the frequency of this disease and some non-random plots were used as permanent plots. The latter were examined repeatedly over a number of years and during any one growing season of a single year. During this process, we selected symptomatic and non-symptomatic trees for complete excavation and dissection. The excavation, using explosives, exposed lateral roots including fine feeder roots.

Our analyses involved a three-year study of information derived from insect infestations, systematic sampling, including isolations from incubated tissues, and concurrent histological work. We incubated samples from symptomatic portions of wood, non-symptomatic portions of healthy and diseased wood, bark beetles and their galleries. We kept all the cultures not less than six months to assure that all fungi in each specimen could be examined. As soon as a new fungus was found in the culture, we immediately subcultured and purified it using standard techniques.

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1Graduate Assistant and Professor of Forest Pathology, respectively, University of Idaho, Moscow, Idaho 83843.
We intensively examined more than 118 trees on 28 permanent plots using these methods. Approximately 4,600 isolates and sub-isolates of fungi were made and examined during this process. We also set up pathogenicity tests on 10 to 30-year old natural lodgepole pine and 52 two to four-year old seedlings. Two hundred fifty trees were inoculated each with one of five Verticicladiella species using one of three inoculation techniques. The species used were purified isolates obtained from symptomatic trees in northern Idaho. After one year, we excavated the roots of twelve trees previously inoculated with inserts of infested wood and found fusiform lesions extending above and below each wound. The lesions were measured and sections of the stained wood incubated on malt agar at room temperature. Histological sections also were made from each fusiform lesion.

Information from our plots showed some tendencies. We rarely encountered the disease as an important destroyer of natural stands. Instead, we found it destructive in planted stands, particularly planted ponderosa pine 20 to 60-years old and in areas where lodgepole pine had grown previously. The disease was particularly severe in ecological transitions between pine and fir zones. This destructive tendency occurred even though the disease was more common in natural lodgepole pine than in any of the other host species. In lodgepole pine it existed in a quiescent state, killing a few trees per year, and rarely an entire stand. We found the black stain complex in the Inland Northwest rare in Douglas-fir, common in planted ponderosa pine, occasional in natural lodgepole pine and uncommon in either natural or planted white pine. We have recorded symptoms of black stain disease and isolated at least one Verticicladiella species from ponderosa pine, lodgepole pine, western white pine, austrian pine, mugo pine, jeffrey pine, jack pine, scots pine, eastern white pine, and whitebark pine in Idaho. In the exotic pines, the disease is capable of destroying a stand in five to nine years. We have seen a four-acre stand of mugo pine wiped out in that time.
In the pines, we repeatedly found a number of fungi and sometimes concurrent insects. Again, these systems differed between natural stands and planted pines. In natural stands we rarely found root bark beetles associated with heavily-stained roots and stems. But large populations occasionally occurred in highly symptomatic trees in which black stain was a minor component, along with blue stain and root-rots. In the Inland Northwest, stem and root-inhabiting insects apparently do not begin to attack natural stands until the stand itself has become seriously deteriorated from other causes; such as root stain disease.

The isolations from symptomatic wood tissues, bark beetles and their galleries, coupled with pathogenicity tests and histological examinations, reveal a great many fungi associated with black stain (Table 1), particularly stain fungi in the genus *Verticicladiella*. These came from stained wood, new insect galleries in healthy bolts of pine used as traps, free-flying beetles, and insects found in the stained wood. Seven species of *Verticicladiella* have been isolated repeatedly from these sources in the past ten years. We have also isolated other stain-fungi such as *Scytalidium* spp., *Torula* spp., and *Alysidium* spp. We frequently isolate these from black-stained wood and therefore, by association, these genera of imperfect fungi may represent other stages of the stain fungi. They may represent oidiol or chlamydosporic forms of *Verticicladiella* spp.

In almost all of our isolates we found combinations of fungi. Even chips less than .5 mm yielded several fungi. We've repeatedly isolated as many as 12 different fungi from a single piece of ponderosa pine wood. The implication is that we are not working with one organism, but a progression of organisms in a complex fungal system. The most common fungi isolated from the black-stained root and stem wood were *V. penicillata* and *V. procera*. These same fungi
Table 1. Fungi isolated from black-stained pines, beetles, or beetle galleries associated with pines in the Inland Northwest.

"W" indicates isolation from stained wood, "B" from beetle, and "G" from beetle gallery.

A. Stain Fungi and Others

Alysidium sp. (W)
Ambrosia fungus (B, G)
Bispora sp. (B)
Candida sp. (B, G)
Cephalosporium sp. (W)
Ceratocystis spp. (B, G, W)
C. ips (B, W)
C. minor (W)
C. montium (W)
Cladosporium sp. (B)
Europhium clavigerum (W)
Eurotium sp. (W)
Fusarium sp. (W)
Gliomastix sp. (W)
Graphium spp. (B, G, W)
Harpographium sp. (W)

Leptographium spp. (B, G)
Mammalia sp. (W)
Mortierella sp. (B, G)
Periconia sp. (G, W)
Phialocephala spp. (B, G, W)
Scytalidium spp. (B, G, W)
Torula spp. (B, G)
Trichoderma sp. (B)
Verticiladiella abietina (B, G, W)

B. Associated Root-decaying Fungi

Armillariella mellea
Fomitopsis annosa
Inonotus tomentosus
Odontia bicolor

Perenniporia subacida
Phaeolus schweinitzii
Phellinus weirii
Stereum sanguinolentum

Table 2. Insects associated with black-stain root disease of major Inland Northwest pines.

Roots
Dendroctonus valens
Hylastes macer
H. longicollis
Hylurgops porosus
flat-headed borers
round-headed borers

Stems
Ambrosia beetles
Dendroctonus brevicomis
D. ponderosae
D. valens
Hylurgops subcostulatus
Ips emarginatus
I. latidens
I. mexicanus
I. montanus
I. pini
Orthotomicus caelatus
Pityogenes fossifrons
Pityokteines elegans
weevils
were commonly isolated from bark beetles or their galleries. V. wagenerii was frequently isolated from stained wood and when found usually was associated with V. penicillata. V. abietina was occasionally isolated from stained wood but more often from bark beetle galleries. This fungus occurred with V. procera and/or V. penicillata. V. serpens and V. antibiotica were relatively uncommon. V. dryocoetidis was isolated rarely and only from stained wood around galleries.

Our results indicate that pine mortality by root stain is caused by a complex system of fungi and insects. In one system Verticicladiella spp. entered and killed fine roots up to 3 mm diameter. Stress caused by drought, soil movement, wind damage or planting procedures may predispose roots to this invasion. The process continued via pathogenesis by Verticicladiella spp. or root-rot fungi to tree damage or mortality. In a second pathway, we found roots killed by one or more agents like Phaeolus schweinitzii or Verticicladiella. Such roots resisted attack by producing callus tissues and forming "nubbin roots." Interiors of these roots contained a black stain from which we isolated Scytalidium spp., a possible chlamydosporic stage of many stain fungi including Verticicladiella spp. In a third pathway, root-rot fungi like Perenniporia subacida, Fomitopsis annosus, Udontia bicolor, or Phaeolus schweinitzii earlier in the life history of the tree destroyed one or more major roots. The stressed tree was then invaded by Verticicladiella spp. directly through root contacts, by insect transmittal or from infested plant parts in the duff. In a fourth pathway, insects particularly Hylurgops porosus (Figure 1) or Hylastes spp. carried Verticicladiella spp. to trees weakened by other factors. We found such insects preferentially building galleries over black-stained wood and carrying the fungi to weakened wood. These beetles carried many fungi, especially yeasts, and several species of both Graphium and Verticicladiella. We frequently isolated the same fungi from stained wood.
Figure 1. Fungi isolated from stained wood of ponderosa pine and from a commonly associated root beetle. (A typical insect-fungus system found in diseased pines.)
None of the systems just outlined occurred uniformly in a stand. During this ten year period, we found many possible alternate and crossover pathways. Nevertheless, definable patterns were recognizable.

We also found a sequence of fungi and insects occurring in a single tree over time. In pines, a resinous zone preceded black-staining by 10 to 15 centimeters. Between this zone and the black stain was a red discoloration from which we obtained Scytalidium spp., Torula spp., Mammaria sp., or Alysidium spp. Two or three of the Verticicladiella species occurred in the black stain. In older black-stained wood, we isolated various Verticicladiella species and other stain fungi including Phialocephala spp., Leptographium sp., several Graphium spp., Periconia sp., Ceratocystis spp., and Euraphium sp. The latter fungi tend to be brought in by bark beetles, especially species of Dendroctonus, Hylurgops, weevils, ambrosia beetles, and buprestids; indicating advanced deterioration of the tree. Note, however, that all are potentially pathogenic and contribute in part to tree mortality as do associated bark beetles. The beetles represent part of the killing system and contributed both their cambial mining and a series of fungi to the pathogenic sequence.

Insects, however, are not always a part of the root stain complex. We found the disease in seedlings with no indication of associated insects and in trees 4 to 6 cm basal diameter with no stem or root insects.

The systems we defined, the interacting insects or their absence, and the alternate pathways indicate there is no one fungus or system causing root stain disease. Instead, we have a series of systems operating in different ways depending on predisposition, the presence or absence of specific fungi, and the presence or absence of specific insects on certain sites to cause stand deterioration. Several species of Verticicladiella are implicated along with
other root-disease fungi and insects. The *Verticicladiella* species most commonly associated with black-stained wood from roots and stems were *V. procera* and *V. penicillata*.

In addition to isolations and observations, we also tested five *Verticicladiella* species for their pathogenicity to naturally grown lodgepole pine. Purified isolates of *V. procera*, *V. penicillata*, *V. abietina*, *V. serpens*, and *V. antibiotica* were aseptically inserted into roots of live trees, one species per tree. After one year, twelve of the 450 inoculated roots were examined. All *Verticicladiella* species produced various sizes of resin-soaked lesions, some produced little or no black-staining. *V. procera* and *V. penicillata* produced the longest lesions, each averaging over 100 mm compared to the control lesions 43 mm in length. *V. penicillata* produced both black stain and foliar symptoms. In almost all cases, we recovered the same pure culture from the stain and lesion areas as was inserted the previous year. Contaminants noted in two root lesions were *Graphium* sp. and *Europhium* sp. These roots were discarded. There was no modification in the morphology of any recovered isolate. We did note, however, that the inoculations accomplished in August produced more black-stain and more rapid lesion development than those inserted in June. This was because little resin was produced after the tree finished its seasonal growth. In all cases, we wounded control trees for comparison with the inoculated ones. Inoculations produced longer lesions than the control wounds.

As we reisolated from these lesions, we also removed a section or several sections of the lesion for histological work. Sections of roots inoculated in August 1979, with *V. abietina*, showed hyphae inhabiting the ray cells and tracheids. Sections of roots with *V. procera* and *V. penicillata* showed heavy resinosus, encased mycelial fragments, and possible fungal deposits in sections of rays and tracheids. Results on *V. serpens* and *V. antibiotica* are not yet
available. Even though we didn't find hyphae in wood containing *V. procera*, we reisolated pure cultures with no morphological changes.

The evidence indicates several species of *Verticicladiella* causing what we call black-stain root disease in the Inland Northwest. Taxonomy is in question, but we definitely can isolate from diseased trees, inoculate healthy ones, produce symptoms, and reisolate the same species without morphological modifications. This demonstrates at least some consistency among published taxons. We have shown two, possibly five, species of *Verticicladiella* capable of producing stain in tracheids and rays of lodgepole pine. *V. abietina*, and *V. penicillata* produced symptoms while *V. serpens*, *V. procera*, and *V. antibiotica* produced lesions. Our records indicate that *V. penicillata* and *V. procera* are the fastest acting and most common fungi associated with root stain disease in this area.

Our information implies that in the Inland Northwest, root stain in natural pines is caused by a slow moving complex of fungi and insects. Regrettably, managers see only the insect component. Therefore, systems are ignored and misdiagnosis commonly is followed by mismanagement. We doubt that pine crops can be raised on sites infested by these complexes.
MEASURING GROWTH LOSS INDUCED BY PHELLINUS WEIRII

by

WALTER G. THIES

Phellinus (Poria) weirii (Murr.) Gilb. (laminated root rot) infects all commercially important coniferous species in the Northwestern United States and British Columbia. Annual losses have been estimated at over 0.9 million cubic meters (32 million cubic feet) in western Washington and Oregon (Childs and Shea 1967) and one million cubic meters (37 million cubic feet) in British Columbia (Wallis 1967).

Although losses to P. weirii are usually measured as direct mortality or windthrow, tree growth may be significantly reduced for a period prior to death. Reductions in annual height or diameter increments have been reported for at least three common Basidiomycete root pathogens of northwest forest trees: P. weirii (Gillette 1975, Ford 1977, Bloomberg and Wallis, 1979), Armillaria mellea (Vahl. ex Fr.) Kummer (Singh and Bhure 1974, Skelly 1974, Shaw and Toes 1977), and Fomes annosus (Fr.) Bref. (Froelich et al. 1977, Bradford et al. 1978). Thus, the concept that root diseases may cause a growth impact on forest trees seems well established. Quantification of volume losses are lacking, however.

In this paper, I report on two methods to determine volume growth loss attributable to infection of young-growth Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) by P. weirii.

MATERIALS AND METHODS

Description of Study Area. -- The study area, in the Oregon Coast Range near Apiary, Oregon, supported a naturally regenerated 40-year-old stand. Site II (McArindle et al. 1949), with 280 trees/ha of which 96% were Douglas-fir and 4% were western hemlock (Tsuga heterophylla (Raf.) Sarg.) with a basal area of 29.5 m²/ha.

Data Collection. -- The study area consisted of a 60-x 90-m plot surrounded by a 12-m-wide border. Each tree in the study area 10.2-cm d.b.h. (stem diameter at 1.4 m) or larger was numbered, its d.b.h. recorded, and its location plotted on a grid map (Thies and Hoopes 1979). All trees were felled in December 1977. Each of the 171 plot trees was measured for total height and height

1/ Research plant pathologist with Forestry Sciences Laboratory, USDA, Forest Service, Pacific Northwest Forest and Range Experiment Station, Corvallis, Oregon 97331.
of primary nodes between the tip and the 25th to 30th node down the stem where nodal location became indistinct. Stem age at nodes was verified by sectioning the stem at every 3rd to 5th node and counting annual rings. A 5-cm-thick cross-sectional disk was removed from each tree at the stump, breast height (1.4 m), 2.4 m, and each merchantable log (2.7-m to 7.6-m height) thereafter for the length of the stem to a 10-cm-diameter inside bark top. One more disk was taken between the 10-cm disk and the tip. A minimum of 10 disks was collected from each tree. The height of each disk was recorded. From each of the 140 border trees a 5-cm cross-sectional disk was removed at breast height. All disks were labelled with the tree number and north point, placed in a plastic bag, and stored at -18°C until radial increments could be measured.

The top of each stump was examined for typical stain or advanced decay of P. weirii infection (Wallis 1976); and when found, P. weirii infection was verified by collecting a second disk and culturing from stained or decayed areas. Presence of P. weirii was determined from morphological features of the colony (Nelson 1975).

Sequential radial growth (SRG) measurements were recorded to the nearest 0.025 cm along three radii of each disk at azimuths of 0°, 120°, and 240° from north. The SRG measurements for each radius were then proportioned so that the radius length equaled the mean disk radius. Data were combined for each disk to provide average reproportioned radial data (Herman et al. 1975). Reproportioned radial data were used for all calculations of basal area, volume, and production of stem profiles (Herman et al. 1975). Stem profiles provided both a visual representation of trees and a visual edit of SRG data.

The stump and roots from each plot tree were bulldozed from the soil in May 1978, washed, and examined for P. weirii based on the presence of decay or ectotrophic mycelium (Buckland et al. 1954).

Computation of Volume and Competition Index. -- Tree volume was calculated for each year. The 9 to 15 sections (logs) of each tree were classified as stump, lower bole, upper bole, or tip. Volume for any given year was found by totaling the volume calculated for each section at that year.

Arney's (1973) Competitive Stress Index (CSI) was computed for each tree at specified years in the stand's development. This index represents a quantification of the relative stress placed on a tree by its neighbors. The index source of stress quantifies relative levels of competition for growing space (crown overlap).
The computational requirements of the index are diameter breast high outside bark and x-y stem coordinates of all trees in the stand. Border tree data was collected specifically to complete data required to calculate CSI for all trees in the plot.

**Determination of Growth Loss.** Using data from 125 healthy Douglas-fir and P. weirii-infected Douglas-fir, growth loss due to P. weirii infection was determined using two approaches, regression and paired-tree.

**Regression Approach** The growth of individual trees was predicted from their past growth and CSI. Growth data from healthy trees was used to establish a multiple regression equation defining the final 10-year volume growth based on volume growth during the preceding 10 years and the CSI value at harvest. When this regression equation was used, the final 10 years of growth of each diseased tree was predicted from its growth the preceding 10 years and its CSI value at harvest. The difference between the predicted growth and the actual growth of diseased trees is assumed to be a reasonable estimate of the growth lost due to the disease. Selection of 10 years as the predictive period was a compromise between retaining a high correlation coefficient, predicting for the longest possible final period of growth, and assuring that during the base period (1957 to 1967), crowns were well above brush competition.

**Paired-Tree Approach** A paired-tree approach was used to provide a second independent estimate of the growth loss due to P. weirii. Diseased and healthy trees were paired that would have been expected to have had similar volumes at harvest without the disease. Trees were paired as they existed 15 years before harvest (1962), a point in time presumed to predate any significant slow-down in growth. Harvest volumes of diseased and healthy trees were then compared to estimate growth loss. Dead diseased trees were paired using their estimated year of death as their harvest year and 15 years before death as the pairing year. For simplicity of presentation, data for all trees will be presented as though harvest was 1977 and the pairing year was 1962. Pairing criteria included d.b.h., inside bark, height, volume, age at the stump, CSI 1962, and CSI 1977.

Pairing criteria for the diseased tree and the candidate trees were examined to determine the best match. This was done to reduce to a minimum differences between paired trees in growth potential and stand position. After pairing, growth during the last decade and harvest volumes of healthy and diseased trees were compared so as to quantify volume lost to P. weirii.

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

Growth Loss—Regression Approach. -- There were highly significant differences between regression-predicted growth and actual growth of diseased trees in the plot. The mean growth loss of all diseased trees was 6.7% of their predicted harvest volume and 13% of their predicted final 10-year volume growth. During their final 10 years of growth, trees classified as dead-infected lost more volume growth (31.8%) than live-infected (11.8%). This trend would be expected since live-infected includes trees in various stages of disease, while dead-infected includes only trees at the terminal end of the disease cycle.

Final 10-year growth volumes for diseased trees were predicted using a linear regression equation determined from healthy-tree growth and CSI data: Vol 67/77 (m³) = 0.341533 + 1.38273 Vol 57/67 (m³) = 0.000894 CSI 77; n = 125; S_{YX} = 0.10558; R^2 = 0.94. When the equation was determined using CSI 1967 values, a highly significant but slightly lower correlation was obtained than when CSI 1977 values were used. Thus, CSI 1977 was chosen for the predictive equation. A multiple regression analysis of Vol 1967/1977 on Vol 1957/1967 and CSI 1977 yielded a highly significant R^2, with both variables being highly significant independent variables.

Growth Loss—Paired-Tree Approach. -- There were significant differences in volume growth between paired healthy and diseased trees. The differences in growth detected by the paired-tree approach closely agree with the differences detected by the regression approach. The mean growth loss of diseased plot trees, as a percent mean growth of healthy paired trees, was 8.1% at harvest and 15.9% for the final decade of growth. During their final decade of growth, dead- and live-infected plot trees grew 38.8% and 12.9% less than their paired healthy trees, respectively.

There were no significant differences in values of pairing criteria between healthy and diseased trees at pairing time (1962); however at harvest (1977), mean volume and height of diseased trees were significantly less than healthy trees.

Variation in Response to Disease, -- Considerable variation was found among plot trees in response to attack by P. weirii. Some infected trees exhibited greater final decade growth than predicted by either the regression approach (9 of 35 infected trees) or by the paired-tree approach (10 of 35 infected trees). Five infected trees demonstrated greater volume growth than was predicted by either the regression or the paired-tree approach. One of the seven down trees had higher 1967/1977 volume growth than the paired healthy tree, while two of the seven down trees grew better than predicted by regression.
Additional Information. -- The mean detectable growth loss occurred within the final 10 years of growth. Incremental volume growth and cumulative volume were examined at 5-year intervals and compared for healthy and infected trees used in the paired-tree analysis. Significant differences were found only at harvest and for the final 5-year increment of growth. Although their losses were predictably larger, results for the dead-infected trees paralleled results for all plot trees.

**DISCUSSION**

This study demonstrated that *P. weirii* can induce losses in stem volume of Douglas-fir and that those losses can be estimated by regression analysis, or by paired tree analysis.

Infected plot trees lost an average 7% of their expected harvest volume. Since most volume loss appears to have occurred during the final decade of growth, an estimate of growth loss during this period may provide the most useful information to a forest manager. During the final decade, growth loss of live- and dead-infected trees averaged 12% and 32%, respectively. If these losses prove to be representative of second-growth Douglas-fir stands, they would influence timing of management activities.

This estimate of loss is supported by similar results from two different analytical approaches. Results of the regression and the paired-tree approaches, within the limits of predictive error, are essentially the same. This similarity provides a measure of confidence in an estimate that cannot otherwise be verified.

Although both systems yield comparable estimates of loss, the regression approach is probably easier to use and more useful than the paired-tree approach which is more difficult and time consuming. The regression approach uses data from healthy trees. It can be used to project performance of infected trees of any size within the range of healthy trees and requires less subjective judgment than the paired-tree approach. The regression approach allows analysis of uneven-aged stands containing relatively few healthy trees; the paired-tree approach does not. The paired-tree approach has the advantage of considering more variables in establishing comparison trees, but only when there is an adequate population of healthy trees from which to choose.

Because laminated root rot develops unseen on root systems and may take 15 years to kill an infected tree and because tree-to-tree variation is high, determining average growth losses associated with it poses especially difficult sampling problems. This study and others (Gillette 1975, Ford 1977, Bloomberg and Wallis 1979) have established the existence of both disease-caused growth reduction and tree-to-tree variation in that
reduction. The emerging picture of wide variation between trees in their response to infection and their ability to cope with disease confuses quantification of the actual growth loss.

This study was conducted in one stand in northwestern Oregon. Without additional data, growth loss information from this stand should not be extrapolated to other stands.

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REFERENCES


This study has two major objectives: 1) to determine the types of factors that influence the potential for spread of P. weirii in second-growth Douglas-fir and the manner in which they do so; and 2) to develop a model for inter-tree spread of laminated root rot in young-growth Douglas-fir.

The data which provided a basis for the analyses relevant to the first objective were obtained by hydraulic root excavations. The first objective concerns the influence of tree, stand and site factors on the probability of inter-tree root contact. The results of the analyses related to this work constitute a contribution to the second objective of the study.

The epidemiological model being developed is a model for inter-tree spread of laminated root rot. This model represents a very simplified and idealized system involving tree-tree and tree-fungus interactions. The goal associated with this aspect of the study is to characterize the behavior of the system for the purpose of evaluating how silvicultural options and environmental constraints influence the propensity for spread of laminated root rot. Though greatly simplified, the modeling effort required a simulation approach. The model and simulator are not covered in this presentation.

The inter-tree spread model should be readily adaptable for use as a submodel in a stand model for laminated root rot impact. Dr. W. J. Bloomberg (personal communication) has been developing such a stand model.

MATERIALS AND METHODS

Description of Study Areas

The root excavation work was carried out on Vancouver Island British Columbia Ministry of Forests Lake Cowichan Experiment Station, located approximately 58 km northwest of Victoria, B. C. (48° 49' N 124° 18' W). The experiment station is situated on the south arm of Cowichan Lake, approximately two miles west of the village of Lake Cowichan.

Thirteen plots were excavated in the immediate vicinity of the lake. Plots 1 through 9 were located on the south side of the lake on the Mesachie Unit of the B. C. Forest Service Lake Cowichan Experiment Station. The elevation for all plots is approximately 175 m. The timber type is relatively uniform throughout the area and has been characterized as a Pseudotsuga-Polystichum association (McMinn 1960). Stand characteristics for these plots, hereafter referred to collectively as the "south side plots," are summarized in Table I.

Plots 10 through 13 were located across the lake on the north side of the south arm on B. C. Forest Products holdings (fig. 1) and will hereafter be referred to collectively as the "north side plots." Here, also, the timber type is uniform and corresponds to McMinn's (1960) description of the
Pseudotsuga-Gaultheria association. Stand and site characteristics are summarized for the north side plots in Table II.

Description of Data Collection

Plot selection criteria

The hydraulic excavation of trees used in the study was performed in the summer of 1979. Plots to be excavated were selected on the basis of three criteria. Close proximity to a reliable water supply was the primary concern. The vicinity of Lake Cowichan was selected (streams on the island rapidly dry up in late spring due to the limited snow pack). Another consideration of practical significance was slope. The steepness of the slope and extent of unimpeded drainage for soil movement become particularly important in those instances where three or more trees were excavated as a group. Under such circumstances, it was necessary to move rather large volumes of soil. Overall, the plots selected had an average slope of approximately 40 percent. Finally, characteristics of individual trees were taken into account (Table I and II) in an attempt to include a range of these characteristics.

Excavation procedure

The general method of excavation has been described by McMinn (1963). The method employed in the current study differs from McMinn's in some respects, however. Except in one instance, no support was provided for stumps since enough surrounding or underlying soil was left in place to provide the necessary support. Additionally, the precise position of roots was not maintained by wire guys since the principal goal was to determine root contact and these could be noted and flagged accordingly as the excavation work was in progress.

The pump utilized for excavation was a Wajax Mark III, which is a high pressure centrifugal pump with a capacity to deliver 225 gallons per minute at a nozzle pressure of 10 p.s.i. at 10 feet. Hose pressure could be varied by an adjustable nozzle. Generally, lower pressures were used to clear soil away from the roots and higher pressures used to clear accumulations of soil.

Root mapping procedure

Once all the plots had been excavated, the mapping was begun. The general mapping procedure was developed by Dr. W. J. Bloomberg (Pacific Forest Research Lab, Victoria, B. C.). The method utilizes a system of root identification whereby roots are assigned to an order class determined by their point of origin. A root originating at the root collar, for instance would be classified as a primary root; a root originating at any point along the length of a primary would be assigned to the secondary root class, etc. Four root order classes were used in all, the highest order being a quaternary. Mapping proceeded in a hierarchial manner: all measurements were made on a primary root; next, the first secondary would be measured; if present, the first tertiary on the latter secondary root would be measured, etc. At all measurement points, diameter, azimuth, horizontal distance and depth were recorded. Measurements were made at the origin, terminus and all intermediate points at which a significant change in any of the measurements occurred. Measurements were also made at all points of root contact whether these were inter- or intra-tree in nature.
For each contact, its type was also recorded. Contact types are defined as follows:

Type 1 - a "simple" contact in which the roots were either touching or at least in very close proximity but which showed no physical indication of contact.

Type 2 - a contact evidenced by some deformation on at least one of the roots, indicating a confirmable and more substantial contact.

Type 3 - a contact in which either one root had grown around the other or along it for some distance, providing a still more substantial contact.

Type 4 - a graft, as determined by external evidence.

Roots were mapped out to minimum diameter of 0.6 cm. Roots with a diameter less than 0.6 cm at their point of origin were not mapped, but their frequency was recorded along each section of mapped root between measurement points. Hereafter, the word "roots" should be taken to mean root larger than 0.6 cm.

Plot data collection.

The most centrally located excavated tree was chosen as a reference tree and designated tree No. 1 on each excavation plot. The remaining excavated trees and all neighbors within 10 meters were numbered sequentially by starting at an azimuth aligned with the slope's aspect and proceeding clockwise around the plot. For each tree, horizontal and vertical distance and azimuth relative to tree No. 1 were recorded. Species, DBH, tree height and crown width were recorded for excavated trees. For neighboring, unexcavated trees, the species was also recorded.

Analytical Methods and Programs

Preliminary data summarization was performed using a program developed by D. W. J. Bloomberg (Pacific Forest Research Center, Victoria, B. C.). The program reports the number of roots, their length, cross-sectional area, volume and number of contacts of each type within specified volumes of soil for each excavated tree.

The data derived from the individual-tree root attribute summarization was used to study the influence of the tree, stand and site factors on the probability of inter-tree root contact. Program BMD07 (Biomedical Computer Programs 1977) was used for this analysis. The latter program performs a stepwise discriminant analysis. Two additional programs were prepared by the author to augment the discriminant analysis. Program PLOTDIS produces a graphic device plot of the canonical variables generated by BMD07. Program RESPOINS produces three-dimensional response surface plots (the response being the probability of no contact) for two to five predictor variables.

The results obtained from the discriminant analysis have been incorporated into a model for inter-tree spread of laminated root rot. At the present time, the simulation model has been validated, but analyses of the simulator's output are still in progress.
RESULTS

The analyses of this section deal with tree, soil and site factors that determine the extent of contact between a pair of trees. Initially, the total number of contacts occurring between a pair of trees was regressed on a wide variety of independent variables (Table III). Comments on units, etc... are included in the table. Two variables, however, require some explanation. The variable "angle" is the departure (in degrees) of a tree-pair from perfect alignment with the slope's aspect. Thus, "angle" represents a measure of relative slope position. The crowding index was obtained by establishing four quadrants about a tree, selecting the nearest tree in each quadrant, dividing the square of the latter's DBH by its distance form the tree in question and summing the resulting indices over the four quadrants.

No significant relationship between any of the variables listed in Table III and inter-tree root contact frequency could be established. Various transformations of these variables were equally ineffective. Discriminant analysis was tried as an alternative. Several group breakdowns were experimented with. Only the final version is presented. In the latter, three contact categories were established as a basis for grouping the data into contact groups:

1) no inter-tree contact (NOCON)
2) inter-tree contacts of Type I only (LOCON)
3) inter-tree contacts of Type I and higher orders (HICON)

A BMD (Biomedical Compute Programs 1977) stepwise discriminant analysis program (BMD07) was used in the analysis. Data on 50 pairs of trees were available as a basis for the analysis. The significant variables were inter-tree slope distance (which, for brevity will hereafter frequently referred to simply as "distance"), \( \ln(DBH_2 + DBH_1) \), effective rooting depth, gravel content of the soil and percent slope. The F-values for removal of discriminant variables from the full model are given in Table IV. The approximate F-statistic for overall model significance is \( F = 4.84 \) (df = 10,86) which is significant at the 0.001 level. In Table V, a matrix for inter-group significance is given. All contact types are significantly discriminated at the 0.05 level. Coefficients of the discriminant functions are given in Table VI. The functions are used to compute the discriminant score, \( S_{ij} \), for observation \( j \) with respect to group \( i \). For details concerning the calculation of the posterior probability of group membership, the reader should refer to the BMD documentation.

The incidence of misclassification for each group is shown in Table VII. The latter table provides some insight into the distinctness of each group. Note that the rate of misclassification is relatively low for the two groups, NOCON and HICON, their respective rates being 0.143 and 0.250. The intermediate group, LOCON, is still significantly different from both of the latter groups in spite of a rather high rate of misclassification (0.555).

The canonical variables from the discriminant analysis are plotted in Figure 1 using program PLOTDIS. One can see in this figure the basis for the misclassifications shown in Table VII. The groups NOCON and HICON are well segregated on the first canonical axis. The intermediate group LOCON is separated from the latter groups in the second dimension. Note, however, that
five of the nine observations from the LOCON group are effectively "buried" in the transitional zone between the NOCON and HICON groups.

As an aid in interpreting the influence of the significant variables and the nature of the interactions, Figures 2 through 7 present the posterior probability of no contact as a function of the five discriminant variables. The distance is held constant at 300 cm. in all of the figures. Two variables are fixed at their respective mean values while the remaining two (which appear on the X and Y axes) are allowed to vary over the range of values shown in Table VIII.

DISCUSSION

Factors Affecting Root Contact and their Relation to Site Hazard

The results of the discriminant analysis indicate that the probability of inter-tree root contact: 1) increases with increasing DBH, percent slope and soil gravel content and; 2) decreases with increasing inter-tree slope distance and effective rooting depth. An important consideration, in view of these results, is whether or not the probability of inter-tree contact can be interpreted as an index of laminated root rot site hazard.

The analysis, of course, did not deal explicitly with the transmissibility of *Phellinus weirii* and its spread behavior needs to be considered. Wallis (1976a) has demonstrated the *P. weirii* does not grow through the soil to any appreciable extent. Moreover, dispersal via basidiospores is apparently non-existent (Nelson 1971). These observations indicate that spread is necessarily through root contacts (or is limited to small distances through the soil) and the latter are thus of paramount importance to the spread of laminated root rot. Wallis (personal communication) has indicated a belief that, given contact, transmission of disease between structural roots is extremely likely if not, in fact, wholly assured. The above discussion clearly lends support to the proposition that root contact potential may serve as a direct measure of spread potential.

Further support for the latter proposition can be found in some of the site hazard studies that deal with *Fomes annosus*. Kuhlman (1963) observed:

The findings suggest that less frequent root contact and failure of the fungus to colonize the tree root results in reduced losses on low hazard sites.

Kuhlman (1974) later asserted that frequency of root contact was the greatest single factor contributing to differences in *annosus* root rot site hazard. Factors that specifically increase root contact are: 1) shorter inter-tree distances (Hodges 1974); 2) limited effective rooting depth (Alexander and Skelly, 1974; Alexander et al., 1975) and; 3) larger tree diameter (Greig, 1962). Effective rooting depth may be limited by very sandy soils (Alexander and Skelly, 1974) or simply a shallow profile development as often occurs on steep slopes. Not all factors limiting rooting depth, however, necessarily increase root contact among trees. Limited effective rooting depth caused by seasonally high water tables may actually decrease the tendency for contact (Froelick et al., 1966; Kuhlman, 1974). Under conditions of periodic soil saturation in the upper soil horizons, root systems tend to develop sparsely branched, stubby roots.
None of the above studies commented on the influence of either steep slopes or soil rock content. As noted, however, steep slopes are commonly associated with shallow soil profile development. In the present study, there is reason to believe that steep slopes exert a further influence by an effect on the spatial distribution of the roots. It was observed in the course of the root excavations (but never quantified) that root systems tend to become more asymmetrical on steeper slopes: roots tended to be more concentrated in the up- and downhill directions instead of evenly distributed about the bole (Also McMinn 1963). This being the case, one might anticipate that relative slope position (Materials and Methods) would also influence the number of inter-tree root contacts. In fact, in preliminary discriminant analyses, relative slope position did appear as a significant variable: as the angle two trees made with the slope's aspect increased the probability of contact diminished.

The influence of soil gravel content might be interpreted as an effect on "effective rooting volume," analogous to effective rooting depth. In both instances, root systems have less volume available for root occupation and the chances of root contact would increase accordingly. Studies dealing specifically with the influence of soil characteristics on root form suggest another effect: increased stoniness seems to promote limited lateral root elongation accompanied by more profuse branching (Harris, 1921; Faulkner and Malcolm, 1972).

One variable, which the present study did not deal with, was soil texture for the simple reason that no significant variation in texture occurred among the study plots. The annosus root rot site hazard studies provide some insight into ways in which soil texture may affect site hazard. It is useful to consider these in relation to laminated root rot site hazard. Very sandy soils, it has already been noted, limit the effective rooting depth, promoting shallow and extensive rooting (Alexander and Skelly 1974) and thus are associated with a higher incidence of root contact. At least in the Douglas-fir subregion of the Pacific Northwest, soils of sufficiently high sand content to produce an effect on rooting form are neither extensive nor common. It is the author's opinion that the soil textural variation that does occur in the subregion is, in general, not of sufficient magnitude to produce significant changes in root form and thus, also, root contact. The findings of Faulkner and Malcolm (1972) also support this contention.

Several of the annosus root rot studies have either suggested or demonstrated a further way in which soil texture may influence site hazard: sandy soils allow more effective spore percolation from the surface and increase the hazard of direct root infection (Kuhlman, 1969; Alexander and Skelly, 1974; Alexander et al., 1975; Froelich et al., 1965; and Froelich et al., 1966). Clearly, this influence on laminated root rot site hazard is irrelevant. In addition, it has yet to be demonstrated that the natural variation in soil texture, pH, base saturation or even microflora has any tangible effect on either the spread or survival of P. weirii.

Our knowledge of factors that determine laminated root rot site hazard is incomplete. One cannot, with complete assurance, take the results of the discriminant analyses generated in this study and use them to formulate an index of site hazard; factors affecting the biology of P. weirii were not taken into account and the present state of knowledge concerning that biology
is quite imperfect. I would like to propose, however, the following hypothesis in lieu of more complete information: those tree, stand and site factors that determine the probability of inter-tree root contact are the dominant variables determining laminated root rot site hazard but, to the extent that these same factors and/or others affect the ability of \textit{P. weirii} to spread and persist in a stand, the site hazard rating may be subject to qualification. It should be possible to provide evidence for acceptance or rejection of the first part of the hypothesis by surveying stands of comparable disease history. A prerequisite for such a study would be a collection of young-growth stands of similar age and residual disease distribution. The degree to which the central premise of the hypothesis fails to be fulfilled may be indicative of the extent to which environmental (physical and biological) influences modify that premise. Evidence for or against the hypothesis would be useful in either case.

The results of the discriminant analysis have also been employed in a laminated root rot simulation model which will be the subject of a separate paper.
### TABLE I. SITE AND STAND CHARACTERISTICS FOR SOUTH SIDE PLOTS

<table>
<thead>
<tr>
<th></th>
<th>No of Observations</th>
<th>Mean</th>
<th>S. E. of Mean</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tree height (m)</td>
<td>18</td>
<td>32.85</td>
<td>2.20</td>
<td>15.60</td>
<td>46.10</td>
</tr>
<tr>
<td>DBH (cm)</td>
<td>18</td>
<td>45.72</td>
<td>12.48</td>
<td>20.00</td>
<td>54.00</td>
</tr>
<tr>
<td>Age (years)</td>
<td>18</td>
<td>64.89</td>
<td>0.28</td>
<td>63.00</td>
<td>67.00</td>
</tr>
<tr>
<td>Gravel (cc/cu ft)</td>
<td>7</td>
<td>3094.14</td>
<td>136.05</td>
<td>2448.00</td>
<td>3617.00</td>
</tr>
<tr>
<td>Cobble (cc/cu ft)</td>
<td>7</td>
<td>741.86</td>
<td>142.32</td>
<td>370.00</td>
<td>1450.00</td>
</tr>
<tr>
<td>Soil depth (cm)</td>
<td>7</td>
<td>120.00</td>
<td>7.79</td>
<td>85.00</td>
<td>140.00</td>
</tr>
<tr>
<td>Slope (percent)</td>
<td>7</td>
<td>48.14</td>
<td>2.22</td>
<td>44.00</td>
<td>60.00</td>
</tr>
<tr>
<td>No of Observations</td>
<td>Mean</td>
<td>S. E. of Mean</td>
<td>Minimum</td>
<td>Maximum</td>
<td></td>
</tr>
<tr>
<td>--------------------</td>
<td>-------</td>
<td>---------------</td>
<td>---------</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td>Tree height (m)</td>
<td>18</td>
<td>24.56</td>
<td>1.56</td>
<td>10.50</td>
<td>33.20</td>
</tr>
<tr>
<td>DBH (cm)</td>
<td>18</td>
<td>26.19</td>
<td>1.85</td>
<td>9.70</td>
<td>38.60</td>
</tr>
<tr>
<td>Age (years)</td>
<td>18</td>
<td>45.39</td>
<td>0.26</td>
<td>43.60</td>
<td>48.00</td>
</tr>
<tr>
<td>Gravel (cc/cu ft)</td>
<td>4</td>
<td>2465.00</td>
<td>172.52</td>
<td>2180.00</td>
<td>2940.00</td>
</tr>
<tr>
<td>Cobble (cc/cu ft)</td>
<td>4</td>
<td>1445.50</td>
<td>236.95</td>
<td>893.00</td>
<td>1946.00</td>
</tr>
<tr>
<td>Soil depth (cm)</td>
<td>4</td>
<td>96.25</td>
<td>6.88</td>
<td>80.00</td>
<td>110.00</td>
</tr>
<tr>
<td>Slope (percent)</td>
<td>4</td>
<td>40.00</td>
<td>1.87</td>
<td>35.00</td>
<td>44.00</td>
</tr>
</tbody>
</table>
### TABLE III. VARIABLES USED IN INTER-TREE CONTACT DISCRIMINANT ANALYSIS.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>inter-tree slope distance</td>
<td>(cm)</td>
</tr>
<tr>
<td>$\ln (\text{DBH}_1 + \text{DBH}_2)$</td>
<td>the natural log of the sum of two trees' DBH</td>
</tr>
<tr>
<td>angle</td>
<td>see text</td>
</tr>
<tr>
<td>soil depth</td>
<td>(cm)</td>
</tr>
<tr>
<td>slope</td>
<td>(percent)</td>
</tr>
<tr>
<td>gravel content</td>
<td>(cc/cu ft)</td>
</tr>
<tr>
<td>cobble content</td>
<td>(cc/cu ft)</td>
</tr>
<tr>
<td>total rock content</td>
<td>(cc/cu ft)</td>
</tr>
<tr>
<td>crowding index</td>
<td>see text</td>
</tr>
<tr>
<td>number of roots per unit</td>
<td>for each root order: the sum of the number of roots of both trees divided by the sum of the volumes of both.</td>
</tr>
<tr>
<td>root volume</td>
<td></td>
</tr>
</tbody>
</table>
### TABLE IV. F-VALUES FOR REMOVAL OF SIGNIFICANT VARIABLES FROM THE FULL, 3-GROUP DISCRIMINANT MODEL

<table>
<thead>
<tr>
<th>variable</th>
<th>F to remove</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>distance</td>
<td>14.37</td>
<td>&lt;&lt;0.001</td>
</tr>
<tr>
<td>DBH</td>
<td>8.72</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>soil depth</td>
<td>3.49</td>
<td>0.05</td>
</tr>
<tr>
<td>gravel</td>
<td>4.99</td>
<td>0.025</td>
</tr>
<tr>
<td>slope</td>
<td>4.72</td>
<td>0.025</td>
</tr>
</tbody>
</table>

a  df = 2, 43

b  group definitions

1. NOCON - no inter-tree contact
2. LOCON - only type I contacts
3. HICON - type I and higher order contacts
<table>
<thead>
<tr>
<th>group</th>
<th>NOCON</th>
<th>LOCON</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOCON</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>HICON</td>
<td>0.001</td>
<td>0.05</td>
</tr>
</tbody>
</table>

a df = 5, 43

b refer to (b) in TABLE IV for group definitions
TABLE VI. COEFFICIENTS OF THE DISCRIMINANT FUNCTIONS FOR THE 3-GROUP MODEL.

<table>
<thead>
<tr>
<th>group</th>
<th>NOCON</th>
<th>LOCON</th>
<th>HICON</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>-175.66739</td>
<td>-208.94675</td>
<td>-196.08026</td>
</tr>
<tr>
<td>distance</td>
<td>-0.06028</td>
<td>-0.07130</td>
<td>-0.07817</td>
</tr>
<tr>
<td>DBH</td>
<td>79.74764</td>
<td>87.01414</td>
<td>87.05154</td>
</tr>
<tr>
<td>soil depth</td>
<td>0.55168</td>
<td>0.59947</td>
<td>0.51809</td>
</tr>
<tr>
<td>gravel</td>
<td>-0.03389</td>
<td>-0.03912</td>
<td>-0.03662</td>
</tr>
<tr>
<td>slope</td>
<td>2.31630</td>
<td>2.69519</td>
<td>2.50904</td>
</tr>
</tbody>
</table>
TABLE VII. INCIDENCE OF GROUP MISCLASSIFICATION FOR THE 3-VARIABLE MODEL

<table>
<thead>
<tr>
<th>group</th>
<th>NOCON</th>
<th>LOCON</th>
<th>HICON</th>
</tr>
</thead>
<tbody>
<tr>
<td>NOCON</td>
<td>18</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>LOCON</td>
<td>4</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>HICON</td>
<td>4</td>
<td>1</td>
<td>15</td>
</tr>
</tbody>
</table>
TABLE VII. VALUES OF INDEPENDENT VARIABLES USED IN FIGURES 2 THROUGH 7.a

<table>
<thead>
<tr>
<th>index</th>
<th>DBHb</th>
<th>soil depth (cm)</th>
<th>slope (percent)</th>
<th>gravel content (cc)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.500</td>
<td>80.0</td>
<td>30.0</td>
<td>2000.0</td>
</tr>
<tr>
<td>2</td>
<td>3.625</td>
<td>85.0</td>
<td>33.0</td>
<td>2200.0</td>
</tr>
<tr>
<td>3</td>
<td>3.750</td>
<td>90.0</td>
<td>36.0</td>
<td>2400.0</td>
</tr>
<tr>
<td>4</td>
<td>3.875</td>
<td>95.0</td>
<td>39.0</td>
<td>2600.0</td>
</tr>
<tr>
<td>5</td>
<td>4.000</td>
<td>100.0</td>
<td>42.0</td>
<td>2800.0</td>
</tr>
<tr>
<td>6</td>
<td>4.125</td>
<td>105.0</td>
<td>45.0</td>
<td>3000.0</td>
</tr>
<tr>
<td>7</td>
<td>4.250</td>
<td>110.0</td>
<td>48.0</td>
<td>3200.0</td>
</tr>
<tr>
<td>8</td>
<td>4.375</td>
<td>115.0</td>
<td>51.0</td>
<td>3400.0</td>
</tr>
<tr>
<td>9</td>
<td>4.500</td>
<td>120.0</td>
<td>54.0</td>
<td>3600.0</td>
</tr>
<tr>
<td>Mean</td>
<td>4.000</td>
<td>100.0</td>
<td>42.0</td>
<td>2800.0</td>
</tr>
</tbody>
</table>

a The index value in the table corresponds to the index used in labeling the axes.

b DBH is defined to be \( \ln(DBH_1 + DBH_2) \) where DBH₁ and DBH₂ are the actual DBH's (cm.) measured for the two trees.
Figure 1. Plot of canonical variables\textsuperscript{a} from 3-group discriminant analysis.

\textsuperscript{a} Plotting code:

1 NOCON
2 LOCON
3 HICON
Figure 2. Posterior probability of no contact plotted over DBH and soil depth.

See text for explanation of plotting procedure.
Figure 3. Posterior probability of no contact plotted over gravel content and soil depth.\(^a\)

\(^a\) See text for explanation of plotting procedure.
Figure 4. Posterior probability of no contact plotted over gravel content and DBH. * 

* See text for explanation of plotting procedure.
Figure 5. Posterior probability of no contact plotted over percent slope and soil depth.a

a See text for explanation of plotting procedure
Figure 6. Posterior probability of no contact plotted over gravel content and percent slope.

\[ a \] See text for explanation of plotting procedure.
Figure 7. Posterior probability of no contact plotted over percent slope and DBH.\textsuperscript{a}

\textsuperscript{a} See text for explanation of plotting procedure.
LITERATURE CITED


Fusarium has been suspected to cause losses in California forest nurseries for at least 20 years. (1) In the past five years a hypocotyl rot disease caused excessive losses in Sugar pine seedlings at two of these nurseries, USFS Placerville and CDF Magalia. At the Magalia nursery, more than 80% of the Sugar pine seedlings died of hypocotyl rot in 1979 despite soil fumigation and fungicide applications. Fusarium spp. had been isolated and suspected of being the causal agent; however pathogenicity was not tested. Recent experiments by the author proved that the causal agent is Fusarium oxysporum.

**Disease Development**

Within the first two weeks following emergence, small red fusiform sunken lesions begin to develop on hypocotyl tissue below the soil surface. These lesions expand both in width and depth until the hypocotyl is girdled causing seedling death. Until the girdling is virtually complete there are no above ground symptoms. The lesions always develop below the soil line at an average depth of 1.7 cm. Greenhouse experiments showed that susceptibility of hypocotyl tissue decreases rapidly from one week after emergence to near complete resistance at two weeks. However, lesions may develop past this infection period, and seedling deaths continue until approximately nine weeks after emergence. No additional losses occur after this period throughout the next two years of the 2-0 stock growth.

Large amounts of iron oxide present in the nursery soils collect in healthy cortical tissue turning it a dark reddish-brown. To visually inspect for root lesions it was necessary to remove these iron oxides from the roots. This was accomplished by soaking the roots for five minutes in a solution of 0.4% sodium EDTA and 0.4% sodium dithionite adjusted to a pH of 6.5 with NaOH. Although extremely rare, root lesions were found, but no significant root rot occurred. However, fusarium root rot is a major cause of losses in other species and areas (2,3).

**Inoculum Sources**

Since both nurseries fumigate before planting, the question arises as to how the fields become recontaminated. At the Magalia nursery the major problem has been insufficient fumigation to kill the soil-borne Fusaria, as was found from pre-and post-fumigation soil plating. The Placerville nursery, using commerical fumigation, has reduced losses during the past three years to below 15%.

A small amount of seed contamination could account for some of the inoculum in fields. The extent of this contamination is being investigated.

**Chemical Control**

Benomyl drenches are presently applied by both nurseries to reduce hypocotyl rot. Control plots at the Magalia nursery in 1979 revealed no significant control achieved by application of three benomyl drenches (27 lb/acre active) after the onset of seedling losses. Earlier applications were not made that year.
In 1980, benomyl 27lb/acre active drenches were applied two weeks prior to planting, and at one day, two and four weeks after planting. Although the results have not been statistically analyzed, even the combination of all 4 applications did not give adequate control. In addition, seed pelleting with benomyl at 25g active/Kg seed also failed to give adequate control despite giving 75% control in greenhouse tests.

Cultural Control

A planting date trial also was initiated in 1980 in cooperation with Bob Bega and Art McCain. Stratified Sugar pine seed was planted in soil artificially infested with barley straw inoculum in February, March, April and May. The February and March plantings had few losses to hypocotyl rot compared to substantial losses in the later plantings. This demonstrates the possibility of culturally avoiding the disease in addition to illustrating the importance of temperature to disease development.

Research is currently underway to further understand the relationships between disease development and temperature, tissue type, moisture, and stress.

Literature Cited


WIFDWIC ATTENDANCE

Analyses of attendance at the first 28 WIFDWIC meetings indicate an increasing interest, although the rate of increase has slowed somewhat in recent years:

<table>
<thead>
<tr>
<th>Meeting years</th>
<th>Average attendance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1953 - 1959</td>
<td>41</td>
</tr>
<tr>
<td>1960 - 1966</td>
<td>66</td>
</tr>
<tr>
<td>1967 - 1973</td>
<td>76</td>
</tr>
<tr>
<td>1974 - 1980</td>
<td>81</td>
</tr>
</tbody>
</table>

A comparison of meetings in the "Golden Triangle" (British Columbia, Washington, Oregon) vs. "Peripheral" sites over the last 14 years shows that those in the Pacific Northwest attract about 20 more than do meetings held elsewhere:

<table>
<thead>
<tr>
<th>Golden Triangle</th>
<th>Peripheral Sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of meetings</td>
<td>7</td>
</tr>
<tr>
<td>Average attendance</td>
<td>88 ± 10</td>
</tr>
</tbody>
</table>

The difference is significant at the 0.01% level.

FRANK G. HAWKSWORTH
I. Taxonomy, Hosts and Distribution

a. The western spruce dwarf mistletoe, Arceuthobium microcarpum, was discovered in the Hay Canyon area of the Sacramento Mountains in the Lincoln National Forest in southeast New Mexico. This is only the second known locality for the mistletoe in the state and represents an eastern range extension of nearly 300 km. in the known distribution of this species. The population only covers a few acres but detailed studies on the extent of its distribution have not been completed. (R. L. Mathiasen, University of Arizona).

b. In a small field test in Colorado, Scots pine was found to be very susceptible to the ponderosa pine dwarf mistletoe, Arceuthobium vaginatum subsp. cryptopodum. Twenty-five Scots pine seedlings were planted under a heavily infested ponderosa pine stand. Twelve of the 16 surviving seedlings were infected after exposure for eight seasons. Thus, Scots pine is not suitable for underplanting in infected ponderosa pine forests. (F. G. Hawksworth, U.S.F.S., Fort Collins, and J. G. Laut, Colorado State Forest Service).

c. The morphological study of pollen of the genus Arceuthobium is continuing. Scanning electron microscope and light microscope studies of each species have been completed and data are now being analyzed to determine whether specific identifications based on pollen are possible (Rolf Mathewes, Simon Fraser Univ., B.C., and F. G. Hawksworth, U.S.F.S., Fort Collins).

d. Additional results have been obtained from the inoculation of various hosts with the Arceuthobium campylopodum-occidentale complex. As reported last year, pinus attenuata and p. sabiniana were infected with both A. campylopodum and A. occidentale but p. ponderosa showed no infection. Since then p. ponderosa has developed infections by both Arceuthobium species. Further observations will be made on shoot and fruit development of the mistletoe plants and the appearance of new infections (R. F. Scharpf, PSW; W. Mark, Cal. Poly; F. G. Hawksworth, RM).
II. Physiology and Anatomy

a. Winter injury temperatures were determined for the shoots of six conifer dwarf mistletoes (Arceuthobium sp.) and (Phoradendron juniperinum) collected in Colorado and Arizona. Injury temperatures were determined by three viability tests: relative electrolyte loss, visual rating, and TTC dye reaction. A. cyanocarpum and A. americanum had the lowest injury temperatures, lower than -60°C. A. douglasii and P. juniperinum had the highest injury temperatures, -25 to -35°C. The shoots of these mistletoes do not avoid freezing by deep undercooling. Therefore, the tissues studied may have maximum winter hardiness levels even lower than those determined in this study (Mike Becwar, Colorado State University and F. G. Hawksworth, U.S.F.S., Fort Collins).

b. Winter collected seeds of two dwarf mistletoe species, Arceuthobium americanum and A. cyanocarpum, avoided freezing to near -33°C. by deep undercooling their tissue water. The principal hosts of these mistletoes are lodgepole pine and limber pine, respectively. Differential thermal analysis freezing profiles revealed that all freezable tissue water of each seed froze in one low temperature exotherm near -33°C. Freezing tests showed that seeds of A. americanum cooled to -40°C., below the low temperature exotherm point, had 0% germination. In contrast, seeds cooled to -30°C., above the low temperature exotherm point, had 52% germination, similar to 54% for control seeds held at -2°C. It is concluded that freezing avoidance by deep undercooling is the winter survival mechanism of the seeds of these dwarf mistletoe species. This winter hardiness limitation may be a factor in limiting the elevational or latitudinal distribution of the dwarf mistletoe species studied (Mike Becwar, Colorado State University and F. G. Hawksworth, U.S.F.S., Fort Collins).

III. Life Cycle Studies

No reports.

IV. Host-Parasite Relations

a. The Shigometer was tested as a means for evaluating vigor reduction due to dwarf mistletoe in lodgepole pine in Colorado. One hundred seventy-two trees in a 90-year-old stand were measured. Four electrical resistance readings at dbh were
taken on each tree. There was a direct relationship between increased electrical resistance and dwarf mistletoe intensity. However, only trees in Infection Class 6 had significantly higher ratings than those in Classes 0 through 5. The study area will be visited during the 1980 W.I.F.D.W.C. field trip at Pingree Park (Bruce Schaffer, Colorado State University and F. G. Hawksworth, U.S.F.S., Fort Collins).

V. Effects on Hosts

a. A study has been started to quantify the effects of dwarf mistletoe in cone and seed production in ponderosa pine (in Colorado) and in lodgepole pine (in Wyoming). This information is needed by forest managers to determine which mistletoe-infected trees are suitable for leave trees in seed tree and shelterwood cuttings (Bruce Schaffer, Colorado State University, F. G. Hawksworth, U.S.F.S., Fort Collins, J. G. Laut, Colorado State Forest Service, and D. W. Johnson, U.S.F.S., Denver).

b. The study on the evaluation damage due to Douglas-fir dwarf mistletoe in the Southwest has been expanded to development of a yield simulation program for all mixed conifers in the Southwest. This complex program will have to consider growth, in various combinations, of nine tree species and five dwarf mistletoes. The field work should be completed in 1981 (R. L. Mathiasen, University of Arizona; Ed Wood, U.S.F.S., Albuquerque; F. G. Hawksworth and C. B. Edminster, U.S.F.S., Fort Collins).

c. Studies are continuing on the relationship between dwarf mistletoe on true firs and cytospora canker. Investigations include changes in the incidence and intensity of branch mortality in dwarf mistletoe infected and noninfected thinned stands on the Eastside Sierra Nevada, changes in tree growth and live crown ratio over time from branch mortality, and differences in mortality and bark beetle attack among dwarf mistletoe-cytospora infected firs and noninfected firs (R. F. Scharpf, G. T. Ferrell; PSW).

d. A special project was initiated to assess the effects of dwarf mistletoe on lodgepole pine and ponderosa pine tree seed production and viability. The general statement that dwarf mistletoe affects tree seed production is often made in the literature; however, there is no information on the affect of dwarf mistletoe on cone or seed production in lodgepole pine and only one report on ponderosa pine.

The objective of this assessment is to provide needed information for forest resource managers on seed production and viability.

e. Eighteen of 51 surviving trees planted in 1963 around a western hemlock infected with dwarf mistletoe were felled in spring 1980. Infections were counted and measured for size, shoot and fruit production, and location in tree and were dissected for aging. Trees were 4.4 to 9.0 m in height. Numbers of infections per tree ranged from 19 to 901 (average 238) as compared with 3 to 274 (average 53) in 1975. Height of highest infection ranged from 25 to 72% (average 43%) of tree height. Because the residual tree was removed in 1969, all subsequent intensification has occurred in the absence of overstory inoculum source (R. B. Smith and W. J. Bloomberg, Victoria, B.C. PFRC, Victoria, B.C.).

VI. Ecology

a. Studies were continued on correlating the incidence and abundance of Arceuthobium americanum to understory vegetation in lodgepole pine. Because classification of habitat types for Colorado lodgepole pine has not been completed, we cannot yet correlate mistletoe with habitat types. However, there is a marked difference in mistletoe frequency and abundance in plots with different predominant understory plants: (for these analyses only plots with at least 12 plots are included)

<table>
<thead>
<tr>
<th>Predominant Understory Plant</th>
<th># Of Plots</th>
<th>% With Mistletoe</th>
<th>Average Dwarf Mistletoe Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shepherdia canadensis</td>
<td>12</td>
<td>50</td>
<td>1.3</td>
</tr>
<tr>
<td>Vaccinium scoparium</td>
<td>90</td>
<td>36</td>
<td>1.1</td>
</tr>
<tr>
<td>Juniperus communis</td>
<td>31</td>
<td>35</td>
<td>1.1</td>
</tr>
<tr>
<td>Arctostaphylos uva-ursi</td>
<td>41</td>
<td>29</td>
<td>1.0</td>
</tr>
<tr>
<td>Arnica cordifolia</td>
<td>12</td>
<td>33</td>
<td>0.7</td>
</tr>
<tr>
<td>Grass (no shrubs)</td>
<td>45</td>
<td>20</td>
<td>0.7</td>
</tr>
</tbody>
</table>

VII. Control -- Chemical

a. Field tests to control dwarf mistletoe on ponderosa pine were continued near Estes Park, Colorado. Dwarf mistletoe is killed back by a double spraying each summer for two to three years using such compounds as: ActiAide, D 40, Dacamine, 2139, Butyrac ester, 3724, Thistrol, and RP2, 4D, MCPA, PPC, Emuls-amine were applied for the first time in 1979 and rather late in a rainy season and they did poorly but should be tried again. Ethephon, 10637, and Modown may be effective if fresh and applied under favorable conditions. Velpar, 2915 did not do well again last summer under the prevailing concentrations and conditions. Some compounds were quite effective in killing the surface growth with one treatment (Arthur Moinat, Greeley, Colorado and F. G. Hawksworth, U.S.F.S., Fort Collins).

b. Product: Roundup R 41% glyphosate
   Sprayed to runoff 1:99 dilution

Location: Stanislaus National Forest

Hosts: Abies concolor, A. magnifica, Pinus ponderosa
       (Arceuthobium abietinum and A. campylopodum)

Date: Applications April 1977 and August 1977
      Evaluations August 77 and October 1977

Replications: 15-20/species, paired treated and non-treated infections

Results: Shoots killed August 77, re-growth October 1977.
         No damage to host. No or negligible control of infections.

         (A. H. McCain, J. R. Parmeter, R. F. Scharpf, U.C. Berkeley
         and PSW).

VIII. Control -- Biological

No reports.

IX. Control -- Silvicultural

a. The reexaminations of these plots for long-term silvicultural control studies of dwarf mistletoes scheduled for the summer of 1980 were postponed because of travel limitations: lodgepole pine in North Park, Colorado (15-year examination); ponderosa pine in the Mescalero Reservation, New Mexico (28-year
examination); and ponderosa pine at Grand Canyon National Park, Arizona (30-year examination). These examinations will be conducted in the fall of 1980 or in 1981 (F. G. Hawksworth, U.S.F.S., Fort Collins).

b. A study on the economics of dwarf mistletoe control in ponderosa pine in the Southwest was begun several years ago, but has been in limbo because the economist involved in the study was transferred. The work was reactivated in 1980 and a manuscript on this study will be completed soon (Steve Sherwood, F. G. Hawksworth and C. B. Edminster, U.S.F.S., Fort Collins and Ed Wood, U.S.F.S., Albuquerque).

c. Residual overstory ponderosa pines infected with southwestern dwarf mistletoe were killed using MSMA on 2,000 acres of the Tusayan Ranger District, Kaibab National Forest (Ed Wood, Region 3, Albuquerque).

d. Two hundred forty acres of pole-sized ponderosa pine infected with southwestern dwarf mistletoe were thinned on the Cloudcroft Ranger District, Lincoln National Forest. The optimum stocking level was chosen by using the RMYLD program (Ed Wood).

e. Three campgrounds, totaling 66 acres, on the Bradshaw Ranger District, Prescott National Forest, were subjected to a comprehensive program designed to control southwestern dwarf mistletoe. Methods used included thinning, pruning, buffer strips, and underplanting of resistant species (Ed Wood, Region 3).

f. Forest Insect and Disease Management Funds were used for dwarf mistletoe control on 2,480 National Forest acres in Oregon and Washington in Fiscal Year 1980. Approximately 6,800 acres were surveyed to develop control prescriptions. Total FIDM expenditures for dwarf mistletoe management in Fiscal Year 1980 were $170,000 (Hadfield, Region 6).

g. The Region 5, PSW cooperative project begun in 1978 to test the efficiency of controlling dwarf mistletoe in true firs by thinning pre-commercial stands was continued again in 1980. By the end of the summer field season, it is expected that all the plot areas will have been thinned and much of the post thinning data taken (R. F. Scharpf, PSW; R. S. Smith, Det Vogler, Region 5).

h. The Region 5, PSW cooperative project to test the effect of pruning dwarf mistletoe brooms from Jeffrey pines to increase growth and reduce mortality is continuing. Mortality is being recorded yearly and growth will be measured after five years (1982) (R. F. Scharpf, PSW; R. S. Smith, Det Vogler, Region 5).
i. Five-year work plans for survey and suppression of lodgepole pine dwarf mistletoe have been completed for the Redfeather Ranger District, Arapaho and Roosevelt National Forests and the Wyoming State Forest Service. These plans outline needed dwarf mistletoe control strategy in a systematic manner that can be budgeted more efficiently than in the past (D. W. Johnson, U.S.F.S., Region 2).


k. Overstory removal of *A. americanum* infected lodgepole pine from 23,000 acres on the Targhee National Forest in Idaho. Same activity on 100 acres on Salmon National Forest in Idaho (Hoffman, Region 4).

X. Surveys

a. Although eastern dwarf mistletoe (*Arceuthobium pusillum*) has been known to be widely distributed in New Hampshire for many years, it has been generally regarded as a botanical curiosity and not a forestry concern. However, our observations in many parts of the White Mountains show that it is a damaging parasite of red spruce. Not only is the parasite causing growth reduction and tree mortality, but marked trunk swellings are a serious degrade factor as they usually occur on the most valuable butt log. An evaluation is suggested to determine the impact of dwarf mistletoe and the implications of the parasite in the management of red spruce in the White Mountains and elsewhere in New England. A manuscript on this has been accepted for *Plant Disease* (F. G. Hawksworth, U.S.F.S., Fort Collins and Alex Shigo, U.S.F.S., Durham, New Hampshire).

b. Loss assessment on the Bitterroot and Lolo National Forests is now complete.

(Please see next page.)
Infestation percentages:

<table>
<thead>
<tr>
<th>Species</th>
<th>Bitterroot</th>
<th>Lolo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Douglas-fir</td>
<td>43%</td>
<td>17%</td>
</tr>
<tr>
<td>Lodgepole pine</td>
<td>44%</td>
<td>23%</td>
</tr>
<tr>
<td>Western larch</td>
<td>52%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Annual cubic foot volume losses:

<table>
<thead>
<tr>
<th>Species</th>
<th>Bitterroot</th>
<th>Lolo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Douglas-fir</td>
<td>3,258 m</td>
<td>2,122 m</td>
</tr>
<tr>
<td>Lodgepole pine</td>
<td>467 m</td>
<td>701 m</td>
</tr>
<tr>
<td>Western larch</td>
<td>32 m</td>
<td>240 m</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>3,757 m</strong></td>
<td><strong>3,063 m</strong></td>
</tr>
</tbody>
</table>

Details of the survey were published in FI&D&M Report No. 80-14. The impact survey is now complete for Montana; this year we completed the field work on the Flathead and Kootenai National Forests. Loss data will be reported later (O. Dooling, Region 1, Missoula).

c. Pre-suppression surveys were conducted on 17,933 acres of the ponderosa pine-type on four National Forests and two Indian Reservations in Arizona and New Mexico. All stands surveyed were analyzed using the RMYLD program to determine the combination of cutting interval and intensity which would maximize fiber production (Ed Wood, Region 3, Albuquerque).

d. Pre-suppression surveys for A. americanum were conducted on 7,600 acres on the Shoshone National Forest; 2,600 acres on the Arapaho and Roosevelt National Forests; and 600 acres on state-owned sections in Wyoming. Surveys initiated in 1979 on the Gunnison National Forest on 17,000 acres of lodgepole pine-type were continued in 1980 (D. W. Johnson, U.S.F.S., Region 2).

e. Dwarf mistletoe loss assessment survey -- Colorado National Forests. As part of a continuing effort to assess growth loss and mortality caused by A. americanum in the Rocky Mountain Region, a road-plot survey was conducted during the 1979 field season. The road portion of the survey revealed that 46.9% of the 763 miles traversed within six National Forests were adjacent to mistletoe-infested stands and 32.2% of the 264 plots established in conjunction with the road survey contained mistletoe-infected trees. The incidence of the disease ranged from 0 on the Rio Grande to 52.1% on the Routt National Forest.
All forests combined averaged 46.6%. Estimates of annual merchantable cubic-foot volume loss for infested forests ranged from a low of 31.2 M on the White River to 2,398.6 M on the Arapaho and Roosevelt. The combined annual loss for all forests is estimated to be greater than 4.6 million cubic feet and is equivalent to 1.3 times the annual harvest of lodgepole pine sawtimber for these forests (D. W. Johnson, U.S.F.S., Region 2; D. Drummond, U.S.F.S., MAG; F. G. Hawksworth, U.S.F.S., RMFRES).

f. Six plots on southern Vancouver Island, 180-450 m² were examined for spread and intensification of hemlock dwarf mistletoe infections from residual trees to regeneration. Time elapsed since logging ranged from 11 to 37 years. Age of regeneration ranged from 12 to 50 years, including some advanced regeneration less than 2 m in height at time of logging. Plots contained two to five infected residual trees aged 35 to 233 years. In each plot, one residual and four to six regeneration trees were felled and infections were counted. Results are as follows:

<table>
<thead>
<tr>
<th>Plot</th>
<th>Residual Tree No. Infections</th>
<th>Regeneration Trees No. Infections</th>
<th>Average Range</th>
<th>Average Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>373</td>
<td>179</td>
<td>16 - 599</td>
<td>33</td>
</tr>
<tr>
<td>B</td>
<td>3,888</td>
<td>455</td>
<td>1 - 984</td>
<td>30</td>
</tr>
<tr>
<td>C</td>
<td>NA</td>
<td>287</td>
<td>110 - 592</td>
<td>40</td>
</tr>
<tr>
<td>D</td>
<td>NA</td>
<td>77</td>
<td>2 - 287</td>
<td>41</td>
</tr>
<tr>
<td>E</td>
<td>1,086</td>
<td>192</td>
<td>29 - 396</td>
<td>29</td>
</tr>
<tr>
<td>F</td>
<td>4,085</td>
<td>3</td>
<td>0 - 49</td>
<td>17</td>
</tr>
</tbody>
</table>

(W. J. Bloomberg and R. B. Smith, PFRC, Victoria, B.C.).

g. Pre-suppression survey on 400 acres of lodgepole pine-type to determine levels of dwarf mistletoe infection in Targhee National Forest in Idaho (Hoffman, Region 4).

XI. Miscellaneous

a. A special project was initiated on the Pingree Park Campus of Colorado State University, northcentral Colorado, to display the various techniques available to land managers for reducing losses to *A. americanum*. A series of treated and non-treated plots will be signed to inform visitors of the biology, impact and control strategies for dwarf mistletoe. The main objective of this demonstration is to create a highly visual, easily understood and attractive information system. Each treatment and its corresponding non-treated area will
be identified and explained by a series of routed-wood signs. A brochure, for public handout, will be provided (J. G. Laut, Colorado State University; F. G. Hawksworth, U.S.F.S., RMFRES; and D. W. Johnson, U.S.F.S, Region 2).

b. On May 18, dwarf mistletoe infestations were eradicated on more than 85,000 acres in southern Washington. Cost of suppression was zero. Control was achieved by blasting, uprooting, burning, heating, toxic gases, abrasion, and generally one hell of a large explosion. The degree of control achieved was extremely complete in most areas. There should be no need for re-treatment to control infected trees missed in the initial treatment. Although the degree of dwarf mistletoe control achieved was highly satisfactory, the general consensus of people involved in post-suppression evaluations is the method of treatment is highly undesirable and should not be used again. Environmental damage from the dwarf mistletoe control treatment resulted in the loss of 2.5 billion board feet of prime timber, total destruction of 100 miles of streams, damage to 3,100 miles of stream, destruction of two lakes, damage to 167 lakes, 2,000 dead deer, 300 dead elk, 20 dead black bears, 12 dead mountain goats, destruction of 27 recreation sites, obliteration of more than 200 miles of road, more than 25 bridges destroyed, reduction of the height of a mountain by 1,270 feet, 29 people killed and 36 others missing and presumed dead. Damage to the resources within the treatment area is estimated to be at least $1 billion. Damage to resources outside the treatment area is estimated to exceed $1 billion.

No environmental impact statement was filed to cover the dwarf mistletoe control treatment. Public response has been not to repeat the dwarf mistletoe treatment.

The name of the project was Mt. St. Helens Dwarf Mistletoe Eradication (Hadfield, Region 6, GOD).

Late Reports

II. Physiology and Anatomy

Bill Livingston has joined our department and will attempt to extract and characterize plant growth substances from dwarf mistletoe infected black spruce. He will also develop recommendations for dwarf mistletoe seed storage, germination and inoculation of seedling hosts. (F.A. Baker and D.W. French, University of Minnesota)
III. Life Cycle Studies

Seeds of Arceuthobium tsugense have been placed on needles and branches of young western hemlock during the autumn of 1977, 1978, and 1979. By August, 1980, plants had been produced on several of the 1977 inoculations, but none from the later years. These inoculation studies should help to determine some basic aspects of the life cycle of hemlock dwarf mistletoe in southeast Alaska. These include: percent infection from seeds placed on various host tissues; length of time from infection of initial swellings; shoot production; and seed production. (T. Shaw, USFS, Juneau)

VII. Control - Chemical

Aerial applications of sodium TCA eradicated residual black spruce from only one of three trial sites in Minnesota. Overstories present on the three sites where spruce trees survived prevented coverage of target trees. If all trees taller than 5 feet are removed during harvesting, as is currently recommended, sodium TCA should eradicate residual black spruce. (F.A. Baker and D.W. French, University of Minnesota)

IX. Control - Silvicultural

Studies reported on last year (Hemlock dwarf mistletoe and young-growth management in southeast Alaska - A problem or concern? Proceedings of the 27 annual Western International Forest Disease Conference: 21-29) were expanded to a third area. Over 1000 young-growth western hemlock beneath infected residual overstory were felled and examined branch by branch for infection. Results were similar to those reported last year. Thus indicating that had the study plots been thinned at age 15 and the residuals removed during this entry, as per current guidelines, then the population of infections present within the trees remaining is likely too low to support an intensive disease build-up within the planned rotation age. This conclusion is further supported by the lower crown location of the vast majority of established infections; a position that biologically limits their ability to intensify the disease within newly developing crown. (T. Shaw, USFS, Juneau)

XI. Miscellaneous

A model predicting spread and mortality in black spruce stands infested with A. pusillum has been incorporated into an interactive computer program called DMLOSS. More information about this program is included elsewhere in these proceedings. (F.A. Baker and D.W. French, University of Minnesota)
I. Seedling Diseases

A. Fusarium Hypocotyl Rot
   Host: Sugar Pine, Red Fir, White Fir
   Causal Organisms: Fusarium oxysporum
   Control: Chemical
   Development Stage: Field Trial

   Soil drench timing and seed coating with Benomyl have not provided adequate control. (Keith Brownell and Dave Adams, U.S. Berkeley and California Dept. of Forestry)

B. Fusarium Hypocotyl Rot
   Host: Sugar Pine
   Causal Organisms: Fusarium oxysporum
   Control: Biological
   Development Stage: Field Trial

   Planting date trial has shown potential of avoiding disease by early planting. (Bob Bega, Art McCain, Keith Brownell, PSW and U.C. Berkeley)

C. Fusarium Root Rot
   Host: Red and White Fir, Sugar Pine
   Causal Organisms: Fusarium oxysporum
   Control: Chemical, Biological, Silvicultural
   Development Stage: Field Trial

   Benlate is being evaluated as a soil drench and seed coating. Planting is early in spring in soil fumigated the previous fall. The purpose of this test is to minimize reentry of fungus into seedbeds after fumigation. (D. Adams, K. Brownell, B. Krelle, 1 & 3 CDF, 2 U.C., Berkeley)

D. Fusarium Root Rot
   Host: Sugar Pine
   Causal Organisms: Fusarium spp.
   Control: Chemical
   Development Stage: Field Trial

   Evaluation of Captan, Benomyl and two rates of Banrot (61 lbs/acre, 25 lbs/acre) revealed the best control so far is the high rate of Banrot, followed by Captan, the low rate of Banrot, and Benomyl. (Sally Cooley, USDA Forest Service, Region 6)
E. *Phytophthora* Root Rot  
Host: Douglas-fir  
Causal Organisms: *Phytophthora megasperma*  
Control: Chemical  
Development Stage: In Vitro - Greenhouse

Natural tolerance to Ridomil has been found. Tests are planned to determine if Douglas-fir can be effectively protected from these isolates. (Robert M. Hunger and Philip B. Hamm, Oregon State University)

F. Water Molds  
Host: Douglas-fir, Sitka spruce, western hemlock, Pacific silver fir, ponderosa pine  
Causal Organisms: *Pythium* and *Phytophthora* spp.  
Control: Chemical  
Development Stage: Greenhouse

Effectiveness and phytotoxicity of Ridomil 2E as a soil drench and foliar spray at three application rates is being tested. (K. Russell, WDNR, Olympia, WA)

II. Foliage Diseases

A. Swiss Needlecast  
Host: Douglas-fir  
Causal Organisms: *Phaeocryptopus gaumanni*  
Control: Chemical  
Development Stage: Field Trial

Chlorothalonil (Bravo 500), Benomyl and Dithane M-45 are being evaluated. Effectiveness of control trials cannot be measured until October 1980. (Jim Hadfield, USDA Forest Service, Region 6)

B. Swiss Needlecast  
Host: Douglas-fir  
Causal Organisms: *Phaeocryptopus guamanni*  
Control: Chemical  
Development Stage: Operational Trial

Three fungicides have been registered for control of Swiss needlecast on Christmas trees. These fungicides are: Manzate 200, Benlate and a combination of Benlate and Manzate. The spray regime begins in May and ends in October for a maximum of five applications. (K. Russell, WDNR, Olympia, WA)
III. Root Rots

A. Armillaria Root Rot
Host: Red Fir
Causal Organisms: Armillaria mellea
Control: Silvicultural
Development Stage: Field Trial

Four paired plots were established to evaluate the effects of precommercial thinning in red fir plantations infected with Armillaria in southern Oregon. (Greg Filip, USDA Forest Service, Region 6)

B. Armillaria Root Rot
Host: Douglas-fir (minor and others)
Causal Organisms: Armillaria mellea
Control: Silvicultural
Development Stage: Pilot Operational

A 350 ha area near Grand Forks, B.C. which is infected with A. mellea will be stumped and root raked. Controls and treated blocks will be planted to pine, larch and Douglas-fir in fall 1980. (D. Morrison, CFS, Victoria)

C. Fomes Annoeus Root Rot
Host: Hemlock
Causal Organisms: Fomes annosus
Control: Chemical
Development Stage: Field Trial

A field trial is now in progress to determine the effective concentration of zinc sulfate (powder and liquid) on hemlock stumps. (D. Morrison, CFS, Victoria)

D. Phellinus weirii
Host: Douglas-fir
Causal Organisms: Phellinus weirii
Control: Silvicultural
Development Stage: Pilot Operational

A 24 ha tract of land near Vernon which was heavily infected with P. weirii was felled. The area was divided into 2 control blocks, 1 block stumped and root raked, and 1 block stumped. Blocks will be planted to fir and lodgepole pine. (D. Morrison, CFS, Victoria)

E. Phellinus weirii
Host: Many conifer species
Causal Organisms: Phellinus weirii
Control: Silvicultural
Development Stage: Pilot Operational

Three stump removal tests are just getting started in Oregon with planting to be completed this year. (Larry Weir, OSDF)
IV. Rusts

A. Comandra Blister Rust
   Host: Pine
   Causal Organisms: Cronartium comandrae
   Control: Chemical
   Development Stage: Field Trial

   Roundup was tested as a chemical control of Comandra pallida (alternate host) in provincial tree nursery. Aerial growth mortality of Comandra was: 73% - 1.3 lb/ac, 87% - 2.6 lb/ac, 99% - 39 lb/ac. (Eric Allen, Canadian Forestry Service)

B. White Pine Blister Rust
   Host: Pinus monticola
   Causal Organisms: Cronartium ribicola
   Control: Silvicultural
   Development Stage: Field Trial

   Field Trial of 100 paired trees was set up in summer 1980. One member of each pair was pruned to a fixed height depending on size. (Richard S. Hunt, Canadian Forest Service)
Root Disease Committee

Highlights of the 1980 Meeting

1. Greg Filip was elected Chairman for the 1981 meeting.

2. A workshop concerning Regionwide root disease surveys will be held in Davis, California in February 1981. Contact Dave Drummond.

3. Art Partridge and Sue Dubreuil presented a stimulating exhibition with a slide carousel concerning the pitfalls of hasty root pathogen identifications (from a prone position in a dimly lit room). With promptings a heated discussion often followed each slide.

4. A summary of the Root Disease Workshop held in Corvallis, Oregon in February 1980 was distributed. The controversial nature of this document provided a forum for discussion which continued beyond the wall of the meeting room.

The following summaries of work concerning root diseases were submitted:

**Biology**

1. Black-stain root disease
   a. Data collected from 5-year spread plots in Oregon (Hansen, OSU).
   b. Associated insects were documented.
   c. Perfected "technique" for seedling inoculation (Hessburg).
   d. Measured spread plots in Oregon and Washington (Goheen, FIDM).

2. Laminated root rot
   b. Disease transmission among root systems and effect of root destruction on tree growth (Reynolds, Hall, Bloomberg).

3. Mycorrhizae (Edmonds/Driver, UW)
   a. Decomposition of logging residues.
   d. Effect of mycorrhizae on litter decomposition.
   e. Effect of mycorrhizae on N-uptake of Douglas-fir seedlings.
Survey/Impact

1. Laminated root rot
   a. Prediction of future losses and infection beyond visible symptoms (Wallis-PFRC).
   b. Use of crown symptoms to predict extent of infection in East Side mixed-conifer stands (Filip, FIDM).
   c. Survey in both East Side and West Side stands in Oregon and Washington (Goheen, Filip, FIDM).
   d. Survey in northern Idaho (James, FIDM).

2. Annosus root rot
   a. Found with Armillaria in true fir in Colorado (James, FIDM).
   b. Found with Armillaria in grand fir in Oregon (Filip, Goheen, FIDM).
   c. Use of photo plots to detect disease in firs in California (Parmeter, UC, Berkeley).
   d. Colonized A. amabilis stumps most and Douglas-fir least in northern British Columbia (Wallis, PFRC).
   e. Effect of stain on wood quality (Jordon, UW).

3. Armillaria root rot
   a. Completed survey of disease areas in central and southern Oregon (Filip, FIDM).
   b. Extensive occurrence in western Montana (James, FIDM).
   c. Survey of 125 ha of Engleman spruce in central British Columbia (Muir, Selkirk College).

4. Black-stain root disease
   a. Extensive occurrence in ponderosa and lodgepole pine in central Oregon (Filip, Goheen, FIDM).
   b. Distribution and site/stand characteristics of infected Douglas-fir in California (DeNitto, FIDM).

5. Others
   a. Distribution of P. lateralis in California (DeNitto, FIDM).
   b. Use of multi-stage, multi-phase sampling design to relate incidence of root disease with insect attacks in spruce-fir forests in Colorado (Fuller, FIDM).
c. Distribution of root disease in southern Utah (Hoffman, FIDM).

Control

1. Laminated root rot
   b. Establishment of a control demonstration area in Coastal Douglas-fir (Hansen, OSU).

2. Armillaria root rot
   a. Effects of precommercial thinning in red fir plantations in southern Oregon (Filip, FIDM).

3. Annosus root rot
   a. Incidence after precommercial thinning of western hemlock (Chavez, UW).

Notes:

1. On February 4–8, 1980, a root disease workshop was held in Corvallis, Oregon. The latest information concerning Phellinus weirii, Armillaria mellea, Verticicladiella vagenerii, and Fomes annosus was discussed. The meeting was well attended and received. Similar workshops will be held periodically.

2. Fields Cobb and Company lead an expedition into the heart of "black-stain" country including the wilds of southern British Columbia, northern Idaho, and western Montana. The wily Verticicladiella was observed on several occasions.
NEW AND ACTIVE PROJECTS

A. Forest Disease Surveys - General


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


73-A-4 Forest Disease: diagnostic and taxonomic services and research (R. S. Hunt).

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

74-A-2 Verticiladiella in Douglas-fir in Oregon (E. N. Hanson).

76-A-1 Annual disease and insect detection surveys in Idaho forests (J. W. Schwandt and R. L. Livingston).


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


B. Non-infectious Diseases

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

68-B-2 Physiological impact on ponderosa pine growing under natural conditions of chronic exposure to oxidant air pollution (P. R. Miller).
Influence of the forest canopy on total oxidant concentrations (P. R. Miller).

The effect of atmospheric effluents on the forest (R. Blauel, D. Hocking, and S. S. Malhotra).

Effects of smoke on forest disease fungi (J. R. Parmeter).

Chronic effect of photochemical oxidant air pollution on the composition of the ponderosa pine-sugar pine-fir forest cover type (P. R. Miller).

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


Trend of ozone injury to conifers in the southern Sierra Nevadas (Pronos and Vogler).

C. Cone, Seed, and Seedling Diseases

Occurrence of endophytic fungi in conifer seedlings (W. J. Bloomberg).

Composting for organic matter additives at the nursery (K. W. Russell).

Pathology of forest seedlings in storage (J. C. Jopkins).

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

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

Potential of several species of Phytophthora for damage to coniferous forests and forest nurseries (E. Hansen, L. Roth).

Effects of pathogen control on performance of container-grown Douglas-fir seedlings (Thies, Owston).

Nursery disease problems at the Albuquerque Tree Nursery (E. Wood and J. W. Riffle).

Greenhouse and nursery pathogenicity and symptomatology of four soil-borne fungi on five commercial species of conifers at various ages of growth (R. V. Bega).
78-C-3 Chemical and biological control of sugar pine root diseases at USFS, Placerville Nursery using seven fungicides and one suppressive soil (R. V. Bega).

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

80-C-1 Fungicide efficacy tests to evaluate control of Botrytis blight at the Coeur d'Alene Nursery, Idaho (R. L. James, J. Y. 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 (Li, Thies and 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-6 Fungicide tolerances of Botrytis cinerea from forest nurseries (S. Cooley).

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

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

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

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

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

69-D-2 Stump infection by basiospores of Poria weirii (E. E. Nelson).

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

71-D-2 Poria weirii root rot: Biology and control (G. W. Wallis, D. J. Morrison).

71-D-3 Fomes annosus root and butt rot: Biology and control (G. W. Wallis, D. J. Morrison).
Armillaria mellea root rot: importance and biology (D. J. Morrison).

Identification, distribution and intensity of root rots in western Montana and northern Idaho (R. L. James).

Testing native conifer plantings for resistance to Poria weirii (K. W. Russell).

Testing red alder plantings to reduce Poria weirii development (K. W. Russell).

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

Taxonomy and distribution of the endomycorrhizal fungi of the family Endogonaceae (J. M. Trappe).

Distribution and epidemiology of Verticicladiella wagenerii on pinon pine in Colorado (L. B. Helberg).

The role of ectotrophic mycelium in the initiation of Phellinus (Poria) weirii infections (E. M. Hansen).

Survival infectivity of P. weirii in Douglas-fir stumps (E. M. Hansen).

Changes in severity of P. weirii resulting from forest management (E. M. Hansen).

Cytology and sexuality of P. weirii (E. M. Hansen).

Silvicultural prescriptions for management of stands affected by root diseases (N. E. Martin and R. L. James).

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

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

An evaluation of Verticicladiella in Oregon (E. Hansen).


Fertilization and root disruption to control laminated root rot of Douglas-fir (Thies, Nelson).
76-D-6 Effect of surface-applied and incorporated chipped slash, with and without supplemental nitrogen, on soil microflora and survival of Phellinus (Poria) weirii in buried wood cubes (Nelson).

76-D-7 Effect of N, P, and K on survival of Phellinus weirii in buried wood (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. Y. Li).

77-D-5 Characterization of a bacterium antagonistic to Phellinus weirii, Armillaria mellea and Fomes annosus (Anita S. Hutchins).

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. W. Riffle).

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

78-D-4 Inhibition of Fomes annosus on western hemlock stem disks by a Streptomyces sp. (C. Y. Li).

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

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

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


78-D-10 Mortality systems in Douglas-fir incited by Phaeolus schweinitzii (S. Dubriel and A. D. Partridge).

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 (Fuller and 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-10 Comparison of forest survey methods to estimate mortality loss and area infected by root disease in Oregon and Washington mixed conifer stands (G. Filip and A. Partridge).


79-D-12 Evaluation of root disease in 50-year old or older plantations in the Pacific Northwest (D. Goheen).

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 (Terry Shaw).

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

79-D-16 Relative abundance of conidia and basidiospores of *Fomes annosus* in airborne inoculum (Terry Shaw with Dr. E. R. Florance, Lewis and Clark College).

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

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

79-D-20 Protecting western hemlock stumps from colonization by *Fomes annosus* (C. Y. Li).

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

79-D-23 Susceptibility of PNW conifers to laminated root rot (W. G. Thies and E. 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 Pathogenicity of **Verticicladiella** spp. and interaction of **Verticicladiella** spp. and associated insects (A. D. Partridge).


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

79-D-30 Effect of red alder, cottonwood, and Douglas-fir on nitrogen and microbiological activity in soil (C. Y. Li).


80-D-3 Distribution and activity of conifer mycorrhize 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 (Jorgensen 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-6 Incidence of root disease in stands affected by western spruce budworm in northern Washington (G. Filip and R. Harvey).

80-D-7 Losses caused by black stain root disease in intensively managed Douglas-fir stands, Coos Bay District, BLM (D. Goheen).
80-D-8 Use of crown symptoms and root collar examinations to estimate *Phellinus weirii* infection in East Side mixed-conifer stands (G. Filip).

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-12 Occurrence of *Phytophthora lateralis* in the forests of California (Klenjunes and Adams).

80-D-13 Systems of organisms causing black stain in pine roots (Partridge).

80-D-14 Stump reservoirs for *Phellinus weirii* (Partridge).

E. Foliage Diseases

68-E-1 Needle disease of western conifers (J. M. Staley).


77-E-1 *Dothistroma pini* resistance in ponderosa pine (G. W. Peterson).


77-E-4 Resistance to *Phomopsis juniperovora* in geographic sources of *Juniperus virginiana* and *J. scopulorum* (G. W. Peterson).

F. Stem Diseases, Malformations, Witches-brooms, Dwarf mistletoe, etc.


62-F-4 Taxonomy, hosts, and distribution of *Arceuthobium* (F. G. Hawksworth and D. Wiens).
Silvicultural control of ponderosa pine dwarf mistletoe in the Southwest (F. G. Hawksworth).

Spread and intensification of dwarf mistletoe in ponderosa and Jeffrey pines in California (R. F. Scharpf, and J. R. Parmeter).

The effect of dwarf mistletoe on growth of western hemlock (K. W. Russell).

Silvicultural control of dwarf mistletoe in young lodgepole pine stands (G. A. Van Sickle).

Spread and intensification of dwarf mistletoe in young unistoried stands of western larch, Douglas-fir and lodgepole pine with controlled stocking (N. Martin).

Growth impact, associated mortality, and spread and intensification of dwarf mistletoe in stands of Douglas-fir, lodgepole pine, and western larch (O. J. Dooling and N. Martin).

Dwarf mistletoe control in rural and suburban residential developments (J. G. Laut and F. G. Hawksworth).


Inoculation studies to determine the host ranges of Arceuthobium campylopodum and A. occidentale in California (W. Mark, R. Scharpf, F. G. Hawksworth).

Biology and epidemiology of a Peridermium associated with lodgepole pine dwarf mistletoe (F. G. Hawksworth).

Evaluation of herbicides as chemical control agents of dwarf mistletoe (D. M. Knutson).

Expanded field plot study (into SW Oregon) of Douglas-fir dwarf mistletoe development in thinned precommercial stands (D. Knutson).

Control of dwarf mistletoe-caused losses in young true fir stands by thinning (R. S. Smith, R. F. Scharpf, and D. Vogler).


The effect of dwarf mistletoe on mortality and volume loss in released true fir stands (R. F. Scharpf).
Reduction of dwarf mistletoe-caused mortality of Jeffrey pines by broom pruning (R. S. Smith and R. F. Scharpf).

Lodgepole pine dwarf mistletoe surveys on the Gunnison National Forest (Johnson).


Dwarf mistletoe infection in young-growth western hemlock beneath infected old-growth residuals in southeast Alaska (Terry Shaw).

Genetics of resistance of western hemlock to dwarf mistletoe (B. van der Kamp).

Relationship between spread of dwarf mistletoe and stand development in western hemlock (B. van der Kamp).

Growth loss in managed, even-age, dwarf mistletoe infested stands of ponderosa pine in the PNW (E. E. Nelson).

Impact of dwarf mistletoe in the Intermountain Region (J. Hoffman).


Dwarf mistletoe loss assessment surveys (Johnson, Hawksworth, and Drummond).

Seed production and viability loss assessment of dwarf mistletoe of lodgepole and ponderosa pines (Johnson, Hawksworth, Laut, and Schaffer).

Dwarf mistletoe control demonstrations - Pingree Park, Colorado (Johnson, Hawksworth, and Laut).


Root disease fungi found on black spruce infected with eastern dwarf mistletoe (W. H. 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).


80-F-10 Evaluation of dwarf mistletoe control projects in southwestern Idaho (J. W. Schwandt).

G. Stem Diseases - Stains and Decays

63-G-1 A study of Ophiostomaceae wood staining fungi in North America (R. W. Davidson).

66-G-1 Hazard in red fir on federal recreational lands in California (L. A. Paine).

72-G-2 Characterization and development of heartwood stain in *Populus trichocarpa* (A. A. Gokhele).

73-G-1 Decay associated with logging damaged conifers in Oregon and Washington (P. E. Aho).

73-G-2 Tests of wound dressings on artificial injuries on western hemlock and Sitka spruce (P. E. Aho).


73-G-4 The role of microorganisms associated with bark beetles attacking conifers (H. S. Whitney).

73-G-5 The biology and pathology of *Polyporus volvatus* (C. G. Shaw).

77-G-1 Survey for *Fomes fraxinophilus* heart rot of green ash in natural stands in Nebraska (J. W. Riffle and E. Sharon).


79-G-3 *Phellinus robineae* stem decay of black locust: distribution, damage, and biology (J. W. Riffle).

79-G-4 Decay associated with logging wounds in the young-growth white and red firs in northern California (P. E. Aho, R. S. Smith and G. Fiddler).

79-G-6  Taxonomy and pathogenicity of Phellinus ferrugineofusca and Phellinus weirii (A. D. Partridge).

79-G-7  Improved methods for identifying cultures of common wood-inhabiting fungi (A. D. 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 (Aho).

80-G-2  The role of Actinomycetes in the discoloration and decay process of living trees (F. A. Baker).

80-G-3  Inonotus andersonii and decay of oaks in Arizona (Karin Yohem R. L. Gilbertson).

80-G-4  Rate of decay in mature Abies grandis and Tsuga heterophylla infected by Echinodontium tinctorium in northern Idaho (J. W. Schwandt).

80-G-5  Evaluation of decay in advanced grand fir and hemlock regeneration after logging in northern Idaho (J. W. Schwandt).

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. T. Bingham).

61-H-1  Streamlining pollination and progeny test methods in breeding for blister rust resistance in western white pine (R. T. 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. T. Bingham).

66-H-1  Comparative physiology of varieties of western white pine with respect to their reaction to the blister rust fungus (R. J. Hoff).

66-H-4  Numbers and kinds of resistance-genes and their relation to rust symptomatology (G. I. McDonald and R. J. Hoff).

66-H-5  Precise estimates of heritability and combining ability of rust resistance (G. I. McDonald).

66-H-6  Development and pathogenicity of Hypoxylon fuscum on northwestern species of alder (Alnus) (J.D. Rogers).

67-H-1  Etiology of aspen cankers (T. E. Hinds).
67-H-2 Field level of blister rust infection in early-generation, partially resistant western white pine stock (R. J. Hoff).

69-H-1 Thinning and pruning western white pine to control the blister rust disease (J. W. 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 (McDonald and Hoff).

74-H-1 Rust fungi of Cupressaceae and Taxaceae: taxonomy and life histories (R. S. Peterson).

74-H-3 Biology of Hypoxylon serpens (J. D. Rogers).

74-H-4 Biology, development, and systematics of Hypoxylon and its allies (J. D. Rogers).

74-H-5 Biology of Cytospora species causing brown stain in pine logs (J. D. Rogers).

74-H-6 Seed production areas for obtaining western white pine that is genetically improved for resistance to blister rust (Hoff and McDonald).

77-H-1 Characterization of Champion Mine race of Cronartium ribicola (G. I. McDonald and E. M. Hanson).

79-H-1 Diplodia tip blight in the Black Hills of South Dakota (Peterson, Johnson, and Telfer).

79-H-3 Intensity of Sirococcus shoot blight on young-growth western hemlock released through thinning at Thomas Bay, Alaska (Terry Shaw).

79-H-4 Ecological studies of spruce rust diseases in subarctic Taiga forests. USFS Co-op aid with Univ. of Alaska (J. Huang McBeath).


80-H-1 Evaluation of aspen harvesting practices in Colorado and New Mexico (Johnson, Hinds, and Walters).


80-H-3 Site characteristics related to the occurrence of stalactiform rust (Partridge).
80-H-4 Genetic variation of gall frequency in lodgepole and ponderosa pine seedlings inoculated with western gall rust (R. J. Hoff).

80-H-5 Inheritance of horizontal resistance mechanisms (R. J. Hoff).

80-H-6 Verification of white pine blister rust simulation (G. I. McDonald).

I. Wilt and Blight Disease

71-I-1 Dutch elm disease detection surveys in all municipalities in Colorado (J. G. Laut).


77-I-1 Distribution of Dutch elm disease and its principal vector, the smaller European elm bark beetle in Montana urban areas (O. J. Dooling and S. Kohler).

77-I-3 Diplodia pinea tip blight of pines: etiology of stem infections (G. W. Peterson).

77-I-4 Herpobasidium deformans blight of honeysuckle: infection and control (J. W. Riffle).


79-I-2 Resistance to Cerospora sequoiae var. juniperi in geographic sources of Juniperus virginiana and J. scopulorum (G. W. Peterson).

80-I-1 Microbial antagonists as a biological control for Dutch elm disease (F. A. Baker).

80-I-2 Methyl bromide fumigation of oak wilt infected oak logs (F. A. Baker).

J. Defects and Decays of Forest Products


68-J-2 Role of heartwood microflora in the breakdown of thujaplicin in western red cedar heartwood (B. J. van der Kamp).

71-J-1 The evaluation of potential wood preservatives—Thiram and Thiram-Oxathiin mixtures (R. S. Smith and Mrs. C. B. Johansen).

72-J-1 Decay and shock resistance of western red cedar transmission pole in service (J. W. Roff and W. McGowan).

72-J-2 Utilization of decayed wood in pulp manufacture (K. Hunt).

72-J-3 Degradation and preservative treatments of western red cedar shingles and shakes (A. J. Cserjesi, R. S. Smith and T. Littleford).


76-J-1 Microdistribution and efficacy of preservatives in treated wood and their effects on microorganisms (W. W. Wilcox).

79-J-1 Diagnosis of wood decay (W. W. Wilcox).

K. Miscellaneous Studies

67-K-1 Impact of hazardous tree failure on forested recreation sites (L. A. Paine).


67-K-3 Effectiveness of hazard reduction programs on recreation sites - losses and various costs of protection (L. A. Paine).

71-K-4 Species of Mycosphaerella 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.S. Hodges).

72-K-2 Species susceptibility to mechanical failures on recreation sites - replacement of hazardous species (L.A. Paine).

73-K-1 Trees - development and people: Develop a guide on how to do it right for architects, planners, contractors, and homeowners (K. W. Russell).

73-K-2 Forest disease simulation model (W. J. Bloomberg).

73-K-3 Fungi of Washington state and the Pacific Northwest (C. G. Shaw).
74-K-3 Biological delignification of wood (C. G. Shaw).


77-K-5 Development of operational use of biological control of forest pests in British Columbia PC-45 (H. S. Whitney).


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 (Terry Shaw).


79-K-4 Revision and update "Keys to major disease and insects --" in color (A. D. Partridge).

80-K-1 Evaluation of hazardous trees in forested recreation sites and ski areas (Johnson and Sharon).


80-K-3 Interactions among the pine wilt nematode, fungi and bark beetles in the Midwest (F. A. Baker).

80-K-4 Evaluation of the Mt. St. Helens eruptions on insect and disease activity in the blast area (J. Hadfield).

80-K-5 Comprehensive hazard tree survey in Priest Lake State Park (J. W. Schwandt).
80-K-6  Computer programs to analyze street tree inventory data in urban areas of Idaho (J. W. Schwandt).

80-K-7  Using explosives to cut and simultaneous inoculate ponderosa pine tops for cavity nesting birds (Partridge).
PUBLICATIONS


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BUSINESS MEETING MINUTES

The business meeting of the 28th Conference was called to order by Chairman Bob Gilbertson at 1:30 p.m. September 18, 1980.

The minutes and the treasurer's report as printed in the Proceedings of the 27th Conference were approved.

Old Business and Committee Reports

Mistletoe Committee. Met the evening of September 16, 1980. The committee report by Chairman John Laut is included in the Proceedings.

Disease Control Committee. Met the evening of September 17, 1980. The committee report by Chairman Ken Russell is included in the Proceedings.

Root Disease Committee. Met the evening of September 17, 1980. The committee report by Chairman Greg Filip is included in the Proceedings.

Interim Program Chairman. Jim Hoffman's report follows the Treasurer's report.

New Business

Meeting Place. Bart van der Kamp announced that the 1981 Conference would be the third week of September in Vernon, British Columbia.

John Schwandt invited the 30th Conference to northern Idaho. Bot Scharpf invited the Conference to the Lake Tahoe area. Greg Filip invited the Conference to Bend, Oregon. Walt Thies moved and the motion was carried to vote on the three locations and eliminate one from the list; and then vote on the other two. The vote was lopsided in favor of Lake Tahoe; Walt Thies further moved and the motion was carried that the 30th Conference be held in the Lake Tahoe area in 1982.

Election of Officers. After long and intensive work by the Election Committee, Sue Dubreuil and members recommended candidates for 1981: Chairman, Larry Weir (no qualifications stated); Secretary-Treasurer, Terry Shaw (good looking legs). Since the WIFDWC Limited was on time, the Committee's recommendations were accepted; Larry and Terry are on board and headed for Canada.

Honorary Life Members. Linnea Gillman, Don Graham, and Al Tegethoff were elevated to Honorary Life Member status.
Balance on hand following twenty-seventh meeting ....................... $362.38
Interest paid through June 30, 1980 ........................................ $ 15.62
Total ................................................................. $378.00

Receipts - Twenty-seventh WIFDWC Meeting
Registration (51 persons) ................................................. $612.00

Expenses - Twenty-eighth WIFDWC Meeting
Conference expenses ......................................................... $300.00
Total ................................................................. $300.00

Balance from Twenty-eighth WIFDWC Meeting ($612.00-$300.00) = $312.00
Balance September 18, 1980 ................................................ $690.00

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

Our thanks for Ken Russell for maintaining the continuous WIFDWC account at the Washington State Employees Credit Union.
The following topics were suggested for the 1981 WIFDWC meeting in Canada:

1. Roadside dwarf mistletoe surveys - what have we learned?
2. New regression equations to update dwarf mistletoe programs.
4. Bark beetles or root disease - which is the real problem?
5. Update on rust diseases of western conifers.
6. The pinewood nematode - problem or not?
7. Outsiders perceptions of forest pathology.
8. What does the forest manager expect from the forest pathologist?
9. Relationships between Forest management and forest pathology.
10. Forest Pest Management - what about weeds and rodents?
11. The role of co-op education positions to further IPM.
12. Communications improvement workshop.
13. Readability of forest pathology publications and papers.
14. Stand hazard ratings for forest diseases.
15. Economic justification for forest disease control efforts.
16. Workshop on statistical methods to develop sampling schemes for disease surveys.
17. Update on newly accepted common and scientific names for forest disease fungi.
18. Uses and misuses of the Shigometer.
19. Reforestation problems and solutions.
Suggestions for program format and proceedings:

1. Have another "hands on" workshop similar to the needlecast workshop.
2. Update the active and terminated projects' lists.
3. Encourage the open meeting discussions central themes.
4. Provide specific smoking areas in the meeting area.
5. Have night sessions on items of local geographic, geologic, historical or other interest.
<table>
<thead>
<tr>
<th>Meeting No.</th>
<th>Date</th>
<th>Place</th>
<th>Chairman</th>
<th>Secretary/Treasurer</th>
<th>Program Chairman</th>
<th>Local Arrangements</th>
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<tr>
<td>1.</td>
<td>1953</td>
<td>Victoria, B. C.</td>
<td>R. E. Foster</td>
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<td>1954</td>
<td>Berkeley, California</td>
<td>W. W. Wagener</td>
<td>P. C. Lightle</td>
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<td>1957</td>
<td>Salem, Oregon</td>
<td>G. P. Thomas</td>
<td>T. W. Childs</td>
<td>R. L. Gilbertson</td>
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<td>Pullman, Washington</td>
<td>H. R. Offord</td>
<td>R. E. Foster</td>
<td>C. G. Shaw</td>
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<td>1965</td>
<td>Kelowna, B. C.</td>
<td>J. E. Bier</td>
<td>H. S. Whitney</td>
<td>R. V. Bega</td>
<td>A. C. Molnar</td>
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<td>1967</td>
<td>Santa Fe, New Mexico</td>
<td>A. C. Molnar</td>
<td>E. F. Wicker</td>
<td>L. C. Weir</td>
<td>P. C. Lightle</td>
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<td>19.</td>
<td>1971</td>
<td>Medford, Oregon</td>
<td>J. A. Baranyay</td>
<td>D. A. Graham</td>
<td>R. B. Smith</td>
<td>H. H. Bynum</td>
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<td>22.</td>
<td>1974</td>
<td>Monterey, California</td>
<td>R. V. Bega</td>
<td>D. Hocking</td>
<td>J. R. Parmeter</td>
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<td>27.</td>
<td>1979</td>
<td>Salem, Oregon</td>
<td>T. H. Laurent</td>
<td>T. E. Hinds</td>
<td>B. Van Der Kamp</td>
<td>L. G. Weir</td>
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