PROCEEDINGS OF THE 27th ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE

Salem, Oregon
September 1979

Proceedings of the 27th Annual Western International Forest Disease Work Conference

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Compiled by:
Thomas E. Hinds

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FOREWORD

The twenty-seventh annual Western International Forest Disease Work Conference met at the Marion Executive Inn in Salem, Oregon, on September 25-28, 1979. A gala event was had by all who attended the retirement dinner held for Lew Roth on Monday evening preceeding the conference.

The Conference was opened promptly at 9:00am Tuesday morning, after the night before, by Chairman Tom Laurent. His brief welcoming remarks about keeping the conference running on time and on an informal basis were so short they were not included in the proceedings. An official welcome to Salem by Mayor Kent Aldrich was extended to the Conference guests but was likewise short and is missing from the proceedings. Jim Johnston, Environmental Forester with Crown-Zellerbach, presented the keynote address that morning and henceforth follows the proceedings.

Ninety-six persons registered for the Conference. Nineteen wives joined the group for the banquet Wednesday evening which was highlighted by the Salem Community Chorus under the direction of our own Larry Weir.

The officers for the 27th Conference were:

Tom Laurent - Chairman
Tom Hinds - Secretary-Treasurer
Bart van der Kamp - Program Chairman
Larry Weir - Local Arrangements

We would like to express our thanks to everyone who helped make the Conference a success.

The disclaimer appearing on the cover of this year's proceedings is not new. A statement of policy concerning conference organization and activities adopted in 1957 states, "To avoid any conflict of interests between the conference, its members, and the sponsoring agencies of its members, each issue of the Proceedings will be prefixed by the following statement: The contents of these Proceedings are not available for citation or publication in whole or in part without the consent of the authors concerned."

JAMES L. MIELKE

1899-1978

Dr. James L. Mielke was born in Ottertail County, Minnesota, March 26, 1899. He earned a B.S. degree in forestry at Oregon State College in 1925, and returned there for his Masters degree in 1933. Under a scholarship he attended Yale University in 1937 and 1938 and received his Ph.D. in Forestry Pathology there in 1941.

After his junior year at Oregon State in 1924, Jim started as a field assistant in the Division of Forest Pathology working under Dr. J. S. Boyce out of the Portland, Oregon office. Jim's early work was primarily research on white pine blister rust, much of which was carried out in British Columbia, where he worked not only with Dr. Boyce, but also with Ray Hansbrough and numerous other budding forest pathologists.

Mielke worked with the blister rust disease from its northern limits south of Prince Rupert in Canada and throughout the ranges of western white and whitebark pines in western Canada and the United States. When the disease spread into sugar pine in Oregon and southward into California, Jim transferred to the San Francisco Office of Forest Pathology. Here he continued his blister rust research throughout the range of sugar pine. Dr. Mielke became the top authority on this important tree disease, and his Ph.D. dissertation was published in 1943 as a Yale School of Forestry Bulletin, reviewing the history of the white pine blister rust in western North America.

During World War II, when Dr. Lake Gill took leave to join the armed forces from his position as head of the Albuquerque, New Mexico office of Forest Pathology, Dr. Mielke transferred there to take over the work at that office. At that time, one of the principal disease problems was in the giant saguaro cactus plants in Arizona. When Lake returned to Albuquerque, Jim transferred to Region IV where he established an office on the campus of Utah State University in cooperation with the Forest School in Logan, Utah. Dr. Mielke was the first federal forest pathologist to be stationed in Region IV. He thus completed the circuit of working in all the western Regions, except Alaska. In Region IV, Jim's principal forest disease problems were caused by various rust fungi in the pines and firs there.

Dr. Mielke continued his research from this Logan station until his retirement. Even after retirement he collaborated with other scientists in the completion and publication of work he had previously started, while retaining his Logan home where he and his family had cultivated many cherished associations and friendships.

Jim died in Logan November 15, 1978, at the age of 79. He is survived by his wife, Ernestine, and his son Jim, Jr.

Dr. Mielke was a member of the American Association for the Advancement of Science; Society of American Foresters; American Phytopathological Society; Mycological Society of America; National Shade Tree Conference; and a charter member of the Western International Forest Disease Work Conference.

Jim Mielke will be fondly remembered by his many fellow scientists and other friends for his great congeniality and his outstanding sense of humor, which he retained and shared, until his death. Dr. Mielke's name will long be remembered, even by younger researchers who knew him only slightly or had never met him, by his numerous scientific publications and his many specimen collections in all of the Forest Service disease herbaria in the West.

(James W. Kimmey)

A REASONABLE EFFORT

James P. Johnston

Good morning, gentlemen and fellow foresters. We are gathered today to discuss once again the diseases that plague the forests in which we work, to plan for a golden, better tomorrow and to strive for thriftier, more productive stands.

As a bright and optimistic graduate I left my alma mater in the early '40s to make my mark on the world. My first job was as a forester working in the eastern shadow of Mt. Adams in Klickitat County, Washington. In the first several weeks I was to learn that our old enemy, Arceuthobium, was everywhere present in those magnificent Ponderosa stands. Yes, I came early to know Arceuthobium. A graduate forester, at least this graduate forester, is well aware that although this pest won't necessarily kill the tree outright, it will have an impact on the growth and development. And there's so much Arceuthobium!!!

So, as you mark the stands for harvest, the logical course of action is to remove the blighted trees as well as prepare harvesting plans, and to pray for a better tomorrow.

It has been over thirty-seven years since I first went into the woods where Arceuthobium was present, and I know that it is still very much present with us.

In January of 1948 I arrived at my new job in the pure stand of Pinus occidentalis on the top of Morne La Selle in Haiti. This was now "my territory". I was now employed as forester for SHADA, financed by the U. S. Export-Import Bank. My predecessor had begun a find program of selective cutting. Moreover, he had overseen the installation of a small test plot of coffee. Mind you, coffee grows only at higher altitudes. Don't let Mrs. Olsen fool you. It's in Nature's plan that coffee grows only above the low places.

Well, there I was with my pine, my coffee and my new bride. The next six years were to be spent preparing myself for my eventual return in the fall of '53 to the United States and, more specifically, to our great Northwest.

Imagine, if you will, my naive shock and loathing when, in the spring of 1954 in Clackamas County, Oregon, I found our adversary, Arceuthobium, very rampant on the western hemlock. I had travelled several thousand miles by now, I had directed the marking and harvesting of many thousands of board feet of timber, BUT I felt, deep in my heart, that I hadn't gotten off square one.

And I know, as I speak with you this morning, that we all have, still, much work to do. In the intervening years, I've met and walked in the

woods with Lew Roth. Of course, Lew's specialty is the Cinnamon Root rot.

Here, in the Douglas fir region we still see and live with Fomes. Pick your species, pick your disease agent and cry, complain and despair if you wish, BUT, most of all, LEARN and ACT!!

You will hear, in the course of these proceedings, about hemlock mistletoe, about the spread and impact of Fomes annosus, and all manner of butt decay and rot.

One of the stations I passed through on my way today was that of Protection Forester. Now that job title has a nice ring to it. My current job title is Environmental Forester--I staunchly hold that each of us is one and both of these. Foresters, by the very nature of their jobs, must protect and keep in good state the acres under their charge. In 1963, I directed an aerial program that brought relief to some 15,000 acres blighted with the hemlock looper. But loopers are sexy stuff--you can see the blighters flying about. Meanwhile, back at the Double Bar-A Ranch, this expletive deleted Arceuthobium is robbing us out of growth, house and home. So, as you return to your own spread of acres, feel a new sense of dedication. Make friends with your state and federal pathologists. Realize once again that a good forester carries a sharp axe. Don't try to remake your part of the world overnight. But remember that time, tide and Arceuthobium are dynamic--they just ain't standing still.

We read each week of the mounting wood and fiber demands of our nation. We are all charged to return to our acres more mindful of what is expected of us. A fine agenda is layed out for the next three days.

So listen well, take notes, and with reasonable effort--ACT!!

FOREST PATHOLOGY IN THE WEST--PAST PRESENT AND FUTURE

by

Lewis Roth

I'm unsure why I've been invited to speak today under the program title. Perhaps it's because I've been around longer than most of you who are still actively employed. Perhaps it is because of my imminent retirement which will no doubt alter my relationship to the group. I might add that I'm even less sure why I accepted. Only a few years ago the program chairman asked if I'd talk about "Forest Pathology in Retrospect" and I was offended, I didn't feel that old. Today I lack that excuse.

This day and hour represent prime time on your program and I wish I had true pearls of wisdom to give you. Though I may appear quite old to the younger of you and quite rustic to the more suave, I am no sage with profound knowledge of the past, deep understanding of the present and penetrating insight into the future. For the first time in my life I am unsure of the present and a bit apprehensive of the future. Later in the program, however, Ed Wicker may be able to assure you of good times ahead.

The young, inspired and energetic of you may see a bright and expanding future. I regret that I do not. Is this but the pessimism of the old and weary or do I read the present signs correctly?

One might ask, is not the health--in fact the survival--of our forest, street and ornamental trees sufficiently important to society to insure a healthy profession to us? Media releases concerning forest use, protection and policy regularly and glibly refer to "... Protection from diseases and insects" as though these were readily accomplishable and as routine as fire suppresion. The notion is so popular (for publicity purposes) that it served as the ostensible reason around which some of the U.S. research labs were built. You know that the protection from disease that is represented by these releases is substantially false and that at the same time executives initiating this material, for the most part, are indifferent to, or ignorant of, what's required to achieve defacto protection. Why should this paradox between sensitivity to public concern on the one hand and, on the other, insensitivity towards our need for knowledge prevail?

There are more explanations for this than we can consider here and probably more than are known, but somehow we've been unable to get our message across and to demonstrate to those making policy decisions, our worth and capabilities. Our credibility is poor, our material is mystifyingly complex and, since it concerns loss,

distasteful, so that often we simply aren't believed. Perhaps there is historical insight into this matter that will cast some light into the future. Let's look briefly into the past.

Human nature itself fails us. A child runs after the fire engine in excitement (often followed by parents) wanting to be a fireman when he is grown. The youth, frequently with the aid of a teacher, transforms an ugly worm into a beautiful butterfly and is captured with interest, but the youngster who stoops to pick a mushroom by the path is snatched away by an apprehensive parent and is turned off at the start.

Our problems can't be attributed to historical professional antecedants. Robert Hartig, our forbear, was among the great 19th century biologists who established the germ concept of disease. His discoveries quickly found their way into management of the ailing European forests of his time and into the classroom. The work of de Bary and his early successors on rust fungi was quickly applied to white pine blister rust in Europe. The European knowledge of disease as an influence in the forest community was brought to America, often by way of emigration of our early silviculturists. But the knowledge of these men soon submerged in the rush of forest commercialization. Also, early disease challenges arose too near the beginnings of the modern concept of disease for us to hope that adequate working knowledge of the many complex biologic systems involved might have been available. Consequently, early control efforts were often failures.

Fortunately success with disease control, and with it scientific progress, was much greater in work on the simpler crop systems of agriculture from which some transfer to forestry was possible.

The complexity of forest systems remains as one of our greatest challenges, first in convincing both forest practitioners and administrators of the validity of our knowledge and second in getting the facts ourselves. But a lot was known about control of plant diseases, indeed forest diseases, before 1910 when, roughly, work began in western North America.

In the States, forest pathology began at the turn of the century. It was soon given momentum by Herman Von Schrenk[1] at Washington University (St. Louis) and the Missouri Botanical Garden. S.M. Zeller, who had a degree (1909) from Greenville College in dendropathology took his PhD. at Washington then came to Oregon State where he retained an interest in forest diseases (Armillaria,

[1]Cronin, James H. 1959. Herman Von Schrenk (a biography). Kuehn-publisher, McCormick Bldg., Chicago.

canker diseases and trunk rots) but directed the bulk of his effort toward the orchard and nut groves.

Schrenk (later, with the federal Missouri Valley Laboratory) was a prodigious worker. But, because his interests shifted from field pathology toward wood preservation, he came into political conflict with the Secretary of Agriculture and left the department to work with industry. At this timeHaven Metcalf was placed in charge in Washington, D.C. The St. Louis lab was closed and Schrenk's two assistants, Pearly Spaulding and G.G. Hedgecock, were moved to Washington. Metcalf certainly was an Equal Opportunity employer for he soon had at least 6 women on his professional staff. He maintained particularly high standards of professional performance.

In 1907 the Office of Investigations in Forest Pathology, then in the Bureau of Plant Industry of the Department of Agriculture, entered an agreement with the Forest Service to place a consulting pathologist in each National Forest District to assist and advise the district forester on disease matters. This arrangement continued until 1953 when pathology was transferred to the Forest Service's Forest and Range Experiment Stations. At the outset, E.P. Meinicke was assigned to San Francisco (1910), J.P. Weir to Region 1 (Montana) in 1913, W.H. Long to the southwest (Arizona) in 1914 and J.S. Boyce from San Francisco to Portland (PNW) in 1920.

Application of knowledge of disease to forest management by means of this consulting arrangement left something to be desired technically. Apparently asministration wasn't always too smooth either. Years ago, as I read Weir's early papers I wondered where that man really worked. The papers seemed to come from Missoula and also Spokane. Long since I learned that, while it seemed to Weir's interests to be in Spokane, the regional forester refused to pay his rent there (Forest Service responsibility for facilities was part of the agreement) and kept pulling him back to Missoula.

While retention of the research in the Bureau of Plant Industry contributed outstandingly to the science of forest pathology, I feel it established and entrenched in the Forest Service especially a precedent (followed also in too many forestry schools) that remains very damaging to us to this day; that is, the notion that responsibility for disease is alien to forest management and that responsibility for disease management is up to the pathologist rather than the forester and silviculturist. Nothing could be farther from the truth since so many of our disease problems are of man's making and have their solutions in management.

It should be mentioned here that federal pathology research, being authorized in 1915, considerably predated establishment of the forest experiment stations in 1925. By 1918 work was begun in

British Columbia on white pine blister rust which by then was recognized as a real threat. Early work was cooperative by Canadian and U.S. workers. The permanent laboratory at Victoria was established in 1940.

Isolation of forest research from management during the early years was unfortunate because the insight and scholarship available from our predecessors was exemplary. They could have contributed so much more had there not been the necessity of information transfer between agencies. As you know, Weir's publications were both perceptive and prolific. Long's writings on mistletoe and Boyce's on decay are outstanding and Meinicke's papers are classics, especially his "Pathology in Forest Regulation". Certainly this is the basic document in forest pathology concerning integrated pest management.

Instruction on forest pathology began fairly early in western universities. As early as 1912, while forestry was still a curriculum in the Department of Botany at Oregon Agricultural College (OSU), instruction in general pathology was available to forestry students, and in 1916 the college catalog carried a listing "Diseases of Forest Trees".

Starting in 1917 a special course in forest pathology was taught at Washington State University by F.D. Heald who was later to distinguish himself as one of the greatest of American plant pathologists. I regard him also as the father of chemotherapeutic treatment of forest trees.

In 1919 instruction began at Idaho by Henry Schmitz, a schoolmate of Zeller's at Washington University. Schmitz went on to become forestry dean at Minnesota and later president of the University of Washington. Work brgan somewhat later in the other western universities.

These early men in the west were giants in the field. They left us a rich heritage and lofty standards to emulate. However, those who followed need not be modest about their accomplishments. Let me mention just a few at random:

1. The whole series of Studies in Forest Pathology by various Canadian workers, especially those concerning decay.

2. Equally valuable to industry, the work of Kimmey, and related papers on deterioration of killed timber.

3. Dave Ethridge's more recent work on infection by heart rot fungi revitalizing research in this important field of pathology.

4. Frank Hawksworth's general accomplishments on the dwarf mistletoes.

5. Toby Child's investigations of pine needle blight.

6. Gardner Shaw's Host/Fungus Indexes; the excellant taxonomic and compilation work of Bob Gilbertson concerning fungi that attack ponderosa pine; Wolf Ziller's Rusts of British Columbia and the work of Ross Davidson and of John Hunt on Ceratocystis.

7. Toby Child's and Keith Shea's Annual Losses from Disease in Pacific Northwest Forests.

8. Development of the excellant forest disease survey system in British Columbia involving Joe Baranyay and others.

9. The continuing development of a strong extension program by Insect and Disease Management in the several regional forestry offices.

10. No less than any of these, the establishment in 1953 of WIFDWC, and its long useful survival.

I've not even mentioned the fine work on root rots and so many other accomplishments familiar to you and in fact done by you.

In spite of solid past accomplishments our track record hasn't always been outstanding. Our failures have damaged our credibility. White pine blister rust is perhaps best known. As I see it our deficiency here lay in inadequate study of epidemiology, unquestioning acceptance of extraregional biological data, disproportionate effort on survey, and some administrative mistakes in the Office of Blister Rust Control. But our encounter with blister rust isn't over and we still could come out of this one looking all right. Our confusion, contradiction and uncertainty over the widely publicized pole blight of wetern white pine, bacteriosis of giant cactus, and ponderosa pine twig blight did little to improve our image.

But where do we stand today? I think we are in very real trouble. We are suffering a serious attrition of scientists. Financial support has decreased both actually and comparitively, and we are nearly destitute of leadership. Some of our most able men (perhaps appropriately) are sent to Ottawa and Washington, but once they are there they seem to vanish into the abyss, often, no doubt because of their high abilities they are transferred to "more important" jobs.

In this depleted condition we are met by a very real consciousness among our forester constituents of need for our services. This

awareness has spun off in part, I'm sure, from problems encountered in preparation of environmental impact statements and partly from intensified I & DM activities.

As for myself, I'm glad I'm retiring from teaching and training of graduate students because I can no longer encourage students into research careers in forest pathology with confidence that they will find personal fulfillment, or even employment.

While I've recieved my share of criticism I believe I have been able during my career to identify and work on matters of importance (not necessarily the most important as usually these were in the hands of stronger agencies) and to properly orient my students. I am not sure as I view the matter today that I could continue through many more years with equal confidence. We can all recognize the major disease problems: decay, root rots and mistletoe. Problems in the nurseries are very real, but we are advised that it is the minor endemic diseases operating every day throughout our forests that in the aggregate cause our really big losses. Now, so often, when I look for these damaging problems I see only the tightly woven web of the forest ecosystem, and nature sloughing off a bit of "dead skin" as it were.

Administratively in Washington periodic reorganizations have changed things so that if the present structure continues, it is most unlikely that we will ever have there a man who can maintain the dedication, much less the influence, of Metcalf. Hutchins, who followed Metcalf was dedicated, but being a fruit pathologist and virologist, was necessarily at a disadvantage and isolated from forestry. Ray Hansborough served capably following Hutchins and Ray had had some experience in the west. But since his retirement the office has seemed to be in a caretaker state with such frequent turnover and vacancy that little could emanate in the way of policy, realistic funding or program development.

Look for a moment at an example of what our failure to tend the administrative store is costing us. In a recent conference to develop a "National Program of Research for Forests and Rangelands", sponsored by the Department of Agriculture, the Association of State University Forestry Research Organizations, and the Association of Land Grant Universities, scheduled increases in employment goals for 1985 were as follows:

	Percent	Scientists
Recreation	220	--
Resource inventory (Two completely nonproductive activities)	159	--

	Percent	Scientists
Timber management	50	597
Harvesting and marketing	37	449
Soils and watersheds	86	410
Range, wildlife and fisheries	150	273
Forest protection	13	365
(Within protection)		
Insect research	13	170
Fire research	25	103
Disease research	11	92

That, gentlepeople, is not just the splitting of a federal pie but it is also the establishment of administrative policy for application of state and private support as well. These relative losses are the price pathology is paying for lack of assertive leadership. Don't ever think these projections are hypothetical and not to be taken seriously. Nothing better illustrates the situation than our experience at Oregon State University. It seems to me not unreasonable in a state such as Oregon where forestry is the number one contributor to the economy that some investment be made by state forestry to research that would facilitate protection of the forest assets from disease. My position as a forest pathologist, largely generated by my own efforts, has been funded by the schools of Science and Agriculture, not by Forestry. A short time back, by working especially through the concerns of people off campus, I encouraged the hiring of a second staff member by forestry, and I dared hope that our effort in pathology could, with my retirement, move into the future at the existing manpower level or greater based on funds available. Certainly this modest effort is needed. For example: the majority of our forest nurseries are suffering seriously from disease in one form or another. Production is down, quality is often very poor, diseased trees die in storage or are planted an die on site. Investments in stock, site preparation and planting are lost and because of failures, sites grow up in weeds and brush.

Nurserymen stream through our offices with stock--its roots black and rotted away, but often robust--begging for help. And how does the administration respond? The pathology dollars are transferred from agriculture to forestry and in conformance with the above

schedule of priorities a pathology position is cut and a soils scientist hired at the professor level.

Events of this kind seem to me to speak something of the future. I can't predict the future for forest pathology in the west, but let me suggest the following considerations with hope that they will contribute to the brightest future possible. In an era when credibility seems lacking everywhere I dare to hope that reliability will be rewarded. As individuals and as a group, we should work to achieve that reliability.

1. Let us cooperate closely with each other both within and between agencies, striving to accommodate and help each other. The wonderful esprit generated by WIFDWC will continue to facilitate this. Not only will close association make our efforts more efficient but it will contribute to balance in the total program as research slows and extension continues to expand, often beyond the limits of a published research foundation. It will enhance a consistent communication with our public.

2. Let us be scrupulously analytical and honest with ourselves and others and let us be genuinely scholarly in order to avoid both the cry of "wolf wolf" and the rediscovery of the wheel when unfamiliar disease situations arise. We have already too much damaged our credibility by sometimes distorting perspective, Of course I realize that the temptation to promote is great when one is starving for support.

3. A great deal of applied research is currently in progress and there is an urgency to be "useful". Let us be careful of going too far on assumptions and preliminary research results without essential followup monitoring to assure that the assumptions are holding. We are vulnerable throughout our work of falling into traps of the same kind that repeatedly damaged the efforts of the old blister rust control program. My fears of this danger are increased when I encounter silviculturists--as is occurring --who assure me that they know all they need to know in order to control dwarf mistletoe.

4. We must remain tireless and undaunted in an effort to increase our knowledge of basic biology, for we operate on a very fractional knowledge of the interlocking web of life that makes up the forest ecosystem that we and the forester manipulate.

5. We must rapidly increase our quantitative capabilities. This will improve the efficiency of our research, open new channels of computer communication, and help assure that computer projections by foresters contain honest representations of the disease situation.

6. As for WIFDWC I wonder if in a time of steady rather than expanding programs we will continue to have adequate research productivity to maintain creative annual meetings. We started in 1953 with a membership and meeting attendance of roughly 90 percent researchers (generators of knowledge) and 11 percent extension people (distributors of that knowledge). Today we have equal numbers of each. It's the fresh new ideas and discoveries that keep us alive technically, not the rehash of old materials. I suggest that we watch carefully for the time when we should meet less often. Failure to discipline our basic workings may destroy the spark that in the past has made our group so successful.

Informality has been a tenet of WIFDWC but undisciplined informality in the absence of bylaws or other guides can destroy us. This meeting is the first where I've heard serious criticism of presentations. While I'm sure this is directed at the type of material presented (which may include this paper) rather than the individual preparation, it is real. Perhaps increasing travel restrictions will force us to less frequent meetings and accompanying greater depth in our presentations.

7. At the point of retirement, I am personally more excited than ever before about the world of interesting, useful discoveries to be made "out there", and more enthusiastic about their application. There is no decrease in the challenge. Nevertheless I think forest pathology, among the plant sciences, is now in a greater than average slump for the times. I urge that a committee of WIFDWC be appointed whose members will dedicate themselves to a study of the health of the profession so as to identify points of weakness and opportunities for advancement. I know such work takes time from our main responsibilities and that we have no funds to support the work but I feel the net gain can be substantial for all of us and can brighten our future.

Panel: Influence of diseases on the management of western hemlock

J.S. Hadfield, Moderator

Introduction - J. S. Hadfield

Western hemlock is a species of major importance to Alaska, British Columbia, Washington, and Oregon. Inventory statistics reveal that there is an estimated 110 billion cubic feet of hemlock distributed along the continental west coast. Hemlock occurs as the principal species on approximately 14 million acres.

For many years, western hemlock was considered a low value species. Recent prices paid for stumpage, logs, and lumber offer dramatic proof of a turnabout in demand for hemlock in the marketplace. Hemlock has been found to be equal to or better than Douglas-fir for many manufactured products.

Coastal zone western hemlock forests are probably the most productive forest lands in North America. The species is capable of truly impressive volume production in short time spans. It responds well to intensive management practices. It is for these reasons that west coast foresters no longer view western hemlock as a low value weed, but as a highly productive, valuable species.

The darkest cloud hovering over western hemlock management is the pathology of the species. Hemlock has developed a reputation of being a disease-prone species. However, much of this reputation stems from observation made in old-growth stands.

Hemlock stands of the future will be carried to rotations of 35 to 100 years with several intermediate entries. How will diseases affect these short rotation stands? Will dwarf mistletoe continue to be a serious problem? The biggest disease question facing hemlock management is *Fomes annosus*. Several foresters are delaying treatment of hemlock because of the uncertainty caused by this disease. Practically all stands are infected. How much loss will this fungus cause in managed stands? Should stand entries be kept to a minimum? Should stumps be treated or removed? These are questions being raised by foresters and pathologists. Todays' speakers will address these issues.

INTENSIVE MANAGEMENT OF WESTERN HEMLOCK

Donald B. Malmberg

Forest Consultant

INTRODUCTION

At one o'clock on the afternoon of August 14, 1933, Hoffman Mountain, a lookout in northwestern Oregon, reported a fire on Gales Creek. Within two weeks that fire spread over 250,000 acres of one of the finest forests of Douglas-fir, western hemlock, and western red cedar that ever grew out-of-doors, and killed 12 billion board feet of timber. That burned timber would have produced enough lumber to build more than a million five room homes had it gone through the mills. That tragic day in 1933 created the Tillamook Burn. It was the first forest fire I fought in my life. What an experience for a teenager. About 40 years later, following reburns in 1939 and 1945, years of snag removal, road building, salvage logging and tree planting, the Tillamook Burn was officially designated as the Tillamook Forest. In 1983, the Society of American Foresters will acknowledge the fifty year anniversary of the forest by holding its annual meeting in Portland, Oregon. I look forward to papers some of you in this audience will present at that landmark meeting in 1983. Oregonians especially want to know about the health and progress of their new Tillamook Forest. Forest pathologists and entomologists have a story to tell. We want to know if all is well with the new crop of timber covering that mountainous terrain.

BACKGROUND

When lumbering began in the Pacific Northwest 150 years ago, lumbermen said, "we must find better ways to get the job done," and they did. "The natural resources of America one day will need intensive management and conservation," they preached. Their outlook was correct, but sooner than most men realized. "Trees out in the far west will last forever," some said. Not so, said President Teddy Roosevelt in the late 1800's. Conservation of forest resources is right and proper, said Colonel William B. Greeley, Chief of the U.S. Forest Service in the early 1900's. In 1945, at the end of World War II, a forester in industry was still somewhat of a novelty in the northwest. Forestry schools across America began to expand in the 50's. Today forestry schools are full and the issue is employment for these men and women ready to go to work in their chosen profession.

GOAL

Ladies and gentlemen, my goal today is to discuss with you a new business. The business of growing and harvesting better trees faster in the Pacific Northwest. This we call intensive forest management. Known well to growers of grains, fruits, and vegetables, it applies now to our young growth forests. It is especially appropriate as we start and grow the "third forest." The objective is better utilization of the timber resources with a minimum disturbance to soil, water, and wildlife resources.

It is a different business than the one the western forest industry has engaged in for the past 150 years, namely harvesting old growth and advanced second growth timber. This new business also involves work and lots of it. The benefits are worth the efforts. We think we can double the yield per acre of merchantable wood by applying intensive management practices. Here are 10 of those practices.

- Prepare the land for planting or seeding.

- Collect seed from genetically superior parent trees.

- Grow seedlings to target specifications.

- Adopt quality regeneration practices which avoid off site planting of tree species.

- Space dense young forests early by precommercial thinning, then follow up later with applications of fertilizers.

- Build roads to impact less on land masses.

- Pretrail the forest in advance of commercial thinning operations for safer more efficient tractor logging operations.

- Close utilization of timber and prompt salvage of timber harmed by weather, insects, diseases, and fire.

- New markets and processing facilities for small wood.

- Adoption of short rotation management, 40 to 50 year cycles on fast growing coastal sites of the northwest.

From this list of options, I have selected thinning as the main topic of my presentation. Of the many species of trees in our northwest woods, western hemlock is the tree I will highlight for you.

Hemlock Habitat

Let me refresh your memory about where hemlock grows, how much volume is standing and identify a few hemlock characteristics. Then we can examine some results and findings of hemlock thinning experiments. Western hemlock is the state tree of the state of Washington. That is proper and fitting, since the largest western hemlock tree is located in the rain forest in the Olympic National Park, and is 27 feet in circumference. The importance of western hemlock (Tsuga heterophylla) is indicated by the distribution of the species plus the acres and volume involved. This tree grows from the San Francisco Bay area of California, north along the coast to Prince William Sound in Alaska, and inland to western Montana. The species exhibits its finest performance in growth on the cool and moist sites along the coast. A standing saw-timber volume of nearly 709 billion board feet throughout the habitat has been reported. Of particular interest with respect to potential

thinning opportunities are the additional 46 billion cubic feet of young growth stands in the western hemlock zone. Western hemlock is further described as the dominant species found on nearly 23 million acres. This tree once regarded as a secondary species has become a primary species of great importance. What happened to western hemlock in the 43 years from 1936 to 1979? May I illustrate by borrowing a case in point from my own company, Crown Zellerback. In 1936, when the last pre-World War II inventory was started at our Northwest Oregon Managed Forest near Seaside, Oregon, certain factors prevailed.

- Hemlock was not wanted in any form.

- No hemlock under 20" d.b.h. was counted in the inventory.

- There was no peeler grade even for Douglas fir logs in 1936.

My, how times have changed. In 1979, hemlock trees are being cruised for inventory purposes and utilized to a 4" top diameter. Approximately 80% of all hemlock production can be marketed as some kind of specialty log in the marketplace. Hemlock pulp grade wood has a firm demand for paper products of many kinds. Can we say hemlock has arrived? Is hemlock management a viable management? All this has happened in part of a person's lifetime.

Silvicultural Factors

Western hemlock is a heavy seed producer and bears some seed almost every year. Germination takes place on organic matter or mineral soil if sufficient moisture is present. Seeds are tiny. About 300,000 seeds per pound. Hemlock commonly occurs from sea level to 3,000 feet elevation in the Coast range and at higher elevations in the Cascade Mountains. Young seedlings grow best in moderate shade. Sudsworth states that western hemlock is perhaps the most shade tolerant of all its associated species in the habitat. Mature trees reach heights of 200 feet on the best sites. Diameters of 3 to 4 feet at breast height are common in mature stands. Although western hemlock is a thin-barked shallow-rooted tree, susceptible to sunscald, logging damage, and weather damage, its silvical characteristics suggest the species is well suited to thinning operations. Hemlock grows well in pure as well as mixed stands.

Management Opportunities

In young stands of western hemlock, the mortality trees have meaning in two categories affecting production. One is recovered mortality, the other is unrecovered mortality. At young ages (under 30 years) most of the mortality trees are of small nonmerchantable sizes. The volume involved has little meaning. The amount of wood contained in mortality trees of merchantable sizes then becomes the item of economic interest. In any event, time is the critical factor where recovery of mortality trees is concerned. About 2 years after a young hemlock tree dies, the sapwood is rotten. Within 3 years the merchantable size young growth hemlock trees

which die are worthless. The salvage. The salvage of mortality wood or, stated another way, the prevention of mortality wood becomes a significant management opportunity.

Response to Release by Thinning

The response to release from competition by early thinning has been noted with enthusiasm but not surprise. Western hemlock, when thinned at early ages, may prove to be one of the most remarkable trees available for intensive management in the Pacific Northwest. Thinning experiments now 20 years old, reveal western hemlock out produces Douglas fir in net yield per acre on short rotations. Today the once poor reputation of western hemlock has truly been reversed. For hemlock growers, the future looks bright. On the negative side, however, we are still concerned about the impact of the root disease Fomes annosus in thinned stands. Specifically, we want to know the impact of Fomes annosus on cubic foot volume loss on a per acre basis on short rotations (40 to 50 years). We want to know the impact of this disease in two categories of forest management.

First - in precommercial thinning operations.

Second - in commercial thinning operations.

Field work by Hadfield, Goheen et al. in 1978 and 1979, plus the fine regional workshops sponsored by the forest pest action council, is shedding more light on the impact of Fomes annosus in thinned stands of western hemlock. Earlier work by Ken Russell, Charlie Driver, Bob Edmonds, Gordy Wallace, and others got our attention real good. Hemlock growers and operators want this scientific work continued. By 1985, we hope the findings by scientists can be pooled, analyzed, and the issue placed in perspective for managers of western hemlock operations.

Conditions for Growing Western Hemlock

Along the northwest Pacific coast, we are blessed by one of the finest growing areas in the world - exceeded only by a small area of coastal Redwoods in California, and a few locations in the southern hemisphere. Coastal soils are deep and high producing. Our trees grow 200 to 260 days a year along the coast, and from 140 to 200 days along the lower Cascade region. That's 24 hours per day by the way, so we are talking about numbers up to 6,000 hours of growth period per year. Compare that to eastern Oregon and Washington where trees grow 60 to 80 days annually but not all day at the higher elevations. So we are talking about 400 to 500 hours of growth period. Also, we like our 8 to 10 feet of rainfall on the coast. It helps grow trees, and helps fish to migrate upstream. For what it's worth, we have learned that our trees grow best on cloudy days, not sunny days. Furthermore, our trees grow better from midnight to dawn, than during the daytime. No wonder we get excited about results of intensive management practices.

The Evidence

What evidence do we have to explain our interest in short rotation management of western hemlock?

- 1084 field plots.

- 33 thinning experiments in Oregon and Washington.

- Stand ages of 10 to 45 years old when first thinned.

- 97 different treatments involving intensity of thinning, interval of thinning, and method of thinning.

- 1,000 acre hemlock thinning model to test various machines and logging systems both tractor and cable.

- Computer programs to process data and produce growth and yield reports for economic analysis.

This program involves much precommercial thinning as well as commercial thinning. The failures exceed the successes when measured in economic terms. To illustrate one of the successes, I have selected Project #7 from the list of 33. The location is near Seaside, Oregon, at low elevation. The old growth hemlock forest in the Vollmer Creek Basin was killed by hemlock looper attacks from 1945 to 1947. Salvage logging of the basin was completed in 1951. Slash was not burned. The new forest was created from seed dropped by the hemlock looper-infested trees. Ten years after clearcut logging of the dead forest was completed, Project #7 was established on a 40 acre parcel of land within the salvaged area. The year was 1961. The reproduction was very dense averaging about 6,000 trees per acre. The age of this sea of hemlock was 15 years, indicating the seeds has fallen and germinated about 5 years before clearcut operations had been completed. This was a valuable lesson to learn by accident and has stimulated work in shelterwood management plans for hemlock. Now I would like to show you a few slides to compare the thinned and unthinned compartments of the new forest. The purpose of Project #7 was to test 3 levels of growing stock-trees per acre against the unthinned control in 1961.

	Trees per Acre			
Levels	300	500	680	at age 15
Goals	170	160	200	at age 40
Rotation age	40 years, not to exceed 100' in height			
Specifications	Favor hemlock, remove all alder, save the biggest and best trees in the forest to last.			

Definitions

Defined: Thinning is the systematic regulation of growing stock in a young forest. (Malmberg-1965)

Objective: The objective of thinning is to develop stands for clearcut harvest containing trees of maximum size, with maximum volume per acre. (Walkup-1975).

Goal: The goal of thinning is to produce larger trees of higher quality in less time than without thinning. (Malmberg 1968).

Purpose: The purpose of thinning is to prepare young forests for clearcut at rotation time. (Walkup 1976).

Role: The role of thinning is the business of growing and harvesting better trees faster on short rotations at least cost. (Malberg 1968).

Project #7, Site Index 168

	Year Age	1961 15	Removed in Thinning	1976 30	Mortality	MAI Growth/Acre Net Cu. 4" Top
Trees						
Plot 22-Control		6190		1305	4885	
Plot 4		5940	5631	310		
Plot 8		6350	6190	160		
SQFT Basal Area						
Plot 22-Control		103		276	141.6	
Plot 4		109	83	249		
Plot 8		105	107	154		
AVE DBH Inches						
Plot 22-Control		1.7		6.3	2.3	
Plot 4		1.8		12.2		
Plot 8		1.6		12.5		
Net Cubic 4" Top						
Plot 22-Control				7142		238
Plot 4				7070		235
Plot 8			2116	4098		224

Highlight: Periodic annual increment on Plot 4 in net cubic feet per acre to a 4" top between 1972 and 1976 was 528, which is about 34,000 pounds of useable green wood each year. My reaction to that finding in one word, was "WOW".

Comment: Dominant trees were about 30 feet tall at age 15. Dominant trees were about 66 feet tall at age 30.

CONCLUSION

Obstacles in the form of brush, stumps, old windfalls, ravines, swamps, and rock outcroppings found in mountainous terrain render uniform spacing and "orchard type forestry" impractical. On the other hand, intensive management by early thinning with variable wide spacing is practical.

LESSONS LEARNED

- Western hemlock responds well to release by heavy thinning at early ages.

- Hemlock trees need at least 40% of their total height in full live crown to maintain rapid rates of growth.

- By age 30, trees in the thinned stand averaged 12.5 inches d.b.h. compared to 6.3 inches in the unthinned control area.

- By age 30, all trees in the thinned stand are of merchantable sizes on today's market.

- By age 40, the goal of 10,000 cubic feet of useable wood per acre will be exceeded. Then we can declare "Mission Accomplished." This happy news gives managers and planners room to consider alternatives of harvesting, depending on the rise and fall of wood products in the market place. Yes, intensive management of western hemlock looks like the way to go. The financial rewards are worthy of the early investments necessary to get a dense young hemlock forest in shape and then to keep it that way. In general, I have found the working managed forest out-produces the struggling wild forest.

A NEW LOOK AT R&D IN THE 1980'S

ASSUMPTIONS

- Technology is advancing rapidly on a broad front.

- A strong and well directed R&D program holds the key to survival in the forest products industry.

- A new breed of scientist is moving into the forest industry.

- The forest products industry can be self-sufficient in energy. Specifically, we should be able to run on our own wood waste products to produce energy for mill operations.

R&D GOALS REGARDING FOREST PESTS

With your permission, I would like to endorse 4 R&D goals in the 80's, regarding forest pests.

- An early warning system. Simply telling us what happened is not good enough.

- A forecasting tool or model to adopt into our long range planning program.

- Orient graduate students to the problems of industry as opposed to the problems of government. Result will be professionals oriented toward the problems of industry and significant research results applied promptly.

- Bring in some talent from outside your specific area of expertise. For example; space programs, military photographs, nuclear science and computer sciences, which bring new contacts and new knowledge to bear on your problems in forest pathology and entomology.

Thank you for your attention, and best wishes for a successful annual meeting here in Salem, Oregon.

HEMLOCK DWARF MISTLETOE AND YOUNG-GROWTH MANAGEMENT IN SOUTHEAST ALASKA--A PROBLEM OR CONCERN?

Charles G. "Terry" Shaw III[1]

INTRODUCTION

Since the mid 1950's some 270,000 acres of old-growth Sitka spruce/western hemlock forest have been clearcut on the mainland and islands of southeast Alaska. These cuttings were largely initiated and continue mainly to supply wood for two pulp mills. Ninety percent of the 400,000,000 to 500,000,000 bd. ft. annual cut is directly or indirectly controlled by the two mills with 60% committed to them through long term contracts with the U.S. Forest Service. Past and present long term contracts have not called for harvest or even felling of trees under 12" d.b.h., and occasionally even larger trees have been left standing.

Hemlock dwarf mistletoe is scattered throughout much of the old-growth forest in southeast Alaska with many stands containing numerous trees with high levels of infection. Stands from sea level to 500', where logging has been concentrated, are often heavily infected. As cutting requirements did not specify removal of unmerchantable stems many of the cut-overs are now studded with residual trees. Where the harvested old-growth forest was heavily infected with dwarf mistletoe the logging aftermath often appeared classic for development of dwarf mistletoe in the naturally regenerating young-growth forest.

Essentially all literature on hemlock dwarf mistletoe biology and effects on hosts originates from outside Alaska. Studies in the Pacific Northwest and on Vancouver Island have suggested that relatively rapid rates of spread and intensification occur in young-growth hemlock developing beneath infected residuals. Region 10, USDA Forest Service, designed a management policy for control of dwarf mistletoe based on Alaska's proximity to these more southerly areas and information gleaned from unpublished letters and reports filed by visiting forest pathologists. These included D.P. Graham in 1966, K. Shea and J. Stewart in 1970, D.R. Miller in 1971, and E.F. Wicker in 1975. The Region was prudent to use this information as no local data on disease development or potential impact were available.

The current management strategy in regenerating clearcuts that contain residual trees infected with dwarf mistletoe is divided into three categories:

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1) Future harvests and stands cut within the past five years: Where dwarf mistletoe is present, remove all residual hemlock from the harvested area. To the extent feasible, harvest dwarf mistletoe infected residuals from uncut borders.

2) Stands that are between 5 and 20 years old that are not scheduled for other timber management treatment for at least five years: Residual hemlock should be removed from these stands if infested residuals are present and if intensive management is planned for the area.

3) Stands that are between 5 and 20 years old that are scheduled for precommercial thinning or hand release within the next five years: Dwarf mistletoe control should be scheduled as part of the work to be done at the next entry.

Along with these direct control efforts silviculturalists in the Region have continually tried, with little success, to add a clause to felling contracts that will require all unmerchantable stems to be cut at harvest.

To date, approximately 20,000 acres have been treated for dwarf mistletoe by residual removal, and there is a backlog of some 36,000 acres scheduled for treatment. An additional 5,000 acres of the 14,000 acre annual cut will also need control work under present guidelines. Costs for residual removal fluctuate from 13 to 21 $/acre, with estimates on the number of residual trees remaining per acre varying widely with area and estimator (i.e. from 8 to over 200).

While this control strategy may appear wise and appropriate, it is not based on local information. To supply these necessary data a cooperative effort by Research and Forest Insect and Disease Management was initiated to determine the extent and nature of dwarf mistletoe in young-growth hemlock developing beneath infected residuals. The preliminary results of this study are reported here.

METHODS

A 30' (radius) circular plot, 0.065 acre, was established around each of 16 living residual hemlock (12" to 28+", d.b.h.) that were heavily infected with dwarf mistletoe. The residual trees were located in a 35 and 43 year old stand at Edna Bay on Kosciusko Island and a 19 year old stand along the Harris River road on Prince of Wales Island.

Within each plot the positions of all spruce and hemlock trees greater than 4.5' in height were mapped. Crop trees were designated using the current guidelines issued to contract thinners for precommercial cutting with a nominal 14' x 14' spacing. All mapped hemlock were numbered; measured for diameter at breast height; height to live crown and total height; felled and aged at the stump; and examined for presence of dwarf mistletoe infections. Inspection for mistletoe was meticulous with each branch, whether living or dead, clipped from the stem and individually scanned for infection.

For each infection the location within the crown and, if possible, sex was determined. All infections were collected for subsequent determination in the laboratory of infeciton age and age of host tissue at the time of infection. These ages were derived from calculations based on locating the first abnormally swollen annual ring as viewed in a cross section cut through the center of the infected branch.

Data were tabulated by percentage of trees infected, stand age, tree age, infection location within the crown, distribution of infections between crop and non-crop trees, infection age, and age of host tissue when it became infected.

RESULTS

Percentage of trees infected varied from 2 - 12% in the 19 year old stand, 1 - 13% in the 35 year old stand and 4 - 41% in the 43 year old stand (Table I). When only trees with infections on live branches were considered there was little change in the proportion of infected trees in the 19 and 35 year old stands, but the level was markedly lowered in the 43 year old stand (4 - 15%) (Table I). Few of the trees bearing infections on live branches had more than 2 infections per tree and few had any infections in the mid or upper crown (Table I).

In all three stands hemlock crop trees had a higher percentage of infection than for the stand as a whole (Table II). Few infected crop trees had more than two live infections per tree and the total number of infections on all crop trees (all infections that would remain if the stand were thinned) was relatively low, generally less than 10 per plot.

In all three stands the proportion of infected hemlock that were either advanced regeneration or residual was consistantly greater than for all trees on each plot (Table III). Thus, a higher proportion of trees that were present within the stand at the time of cutting bear infections than do trees that have developed since logging.

To date only a portion of the infections have been aged. In the 35 and 43 year old stands approximately 10% of these aged infections became established within the first 18 years after harvest (Fig. 1); the approximate age at which the stand would be scheduled for precommercial thinning. Not enough infections have yet been aged from the 19 year old stand to determine a temporal distribution of infection establishment. Infections were established on tissue from 1 to 21 years of age with over 50% of the infections established on tissue 5 years of age or less (Fig. II).

DISCUSSION

These data suggest that in southeast Alaska young-growth hemlock developing beneath residual trees heavily infected with dwarf mistletoe are currently infected by dwarf mistletoe at a relatively low level. The amount of infection present on such trees appears to be lower than that encountered in stands with a similar cutting history along the coast of Oregon, Washington and British Columbia.

These data should represent the area of highest disease risk within the study stands as the young-growth trees were in close proximity to an overhead source of mistletoe seed. Thus, infection levels for the stand as a whole would likely be even lower than found here.

If the study stands were thinned at their present age several crop trees would bear some infections. However, the residual trees would be cut or otherwise killed during this thinning, thus forever removing the overhead source of infection. The population of infections present within the trees remaining appears to be to 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 the biologically limits their ability to intensify the disease on newly developing crown.

The important question for management is: If these relatively low levels of infection are consistent throughout the geographic area (an assumption supported by cusory observation) is a separate entry for removal of overstory trees to control dwarf mistletoe justifiable? Certainly every effort should be made to insist on felling residuals at harvest and infected residuals should continue to be removed during precommercial thinning. But what of the areas currently scheduled for treatment - is the disease serious enough to warrant a separate control operation?

TABLE I.--Distribution of dwarf mistletoe infections in the 19, 35, 43 year old stands.

	Plot number	Number of live hemlock examined	Number of infections located	% of trees infected	% of trees with live infections	% of trees with > two live infections	% of trees infected in mid or upper crown
19 year old Stand	1	124	3	2	2	0	0
	2	183	12	5	5	1	1
	3	208	60[a]	11	11	3	2
	4	396	73	10	9	1	1
	5	399	89	12	11	2	1
35 year old Stand	6	89	1	1	1	0	0
	7	66	3	5	5	0	2
	8	154	7	4	4	0	1
	9	265	2	1	1	0	0
	10	154	24	13	13	0	0
43 year old Stand	11	54	62	41	14	2	7
	12	44	6	7	5	2	5
	13	144	101	26	15	4	3
	14	48	2	4	4	0	2
	15	68	11	7	4	0	3
	16	115	62	19	12	2	5

[a]108 infections on one 55 year old tree not included.

TABLE II.--Distribution of dwarf mistletoe infections on crop trees in the 19, 35, 43 year old stands.

	Plot number	number of hemlock crop trees examined	total number of live infections	% of crop trees infected	% crop trees infected on live branches	% crop trees with > two live infections	% all trees infected[a]
19 year old Stand	1	8	3	38	38	0	2
	2	9	5	33	33	11	5
	3	5	12	20	20	20	11
	4	12	15	58	50	8	9
	5	14	16	50	43	14	11
35 year old Stand	6	6	0	0	0	0	1
	7	8	3	25	25	0	5
	8	6	2	17	17	0	4
	9	10	1	10	10	0	1
	10	1	0	0	0	0	13
43 year old Stand	11	8	11	75	50	13	41
	12	5	4	20	20	20	7
	13	7	10	57	43	29	26
	14	6	0	0	0	0	4
	15	8	2	38	25	0	7
	16	11	28	64	45	18	19

[a]See table I.

TABLE III.--Distribution of infected trees by age classes in the 19, 35 and 43 year old stands.[1]

	Plot Number	Number of Trees		Percent Young-growth		Percent Advanced-regeneration		Percent Residual	
		Total[2]	Infected	Total	Infected	Total	Infected	Total	Infected
19 year old Stand	1	124	3	52	33	40	67	8	0
	2	183	9	35	33	51	56	14	11
	3	208	22	33	0	61	73	6	27
	4	396	40	68	52	32	48	0	0
	5	399	48	38	17	60	73	2	10
35 year old Stand	6	89	1	84	0	14	100	2	0
	7	66	3	60	0	27	33	13	66
	8	154	6	92	83	8	17	0	0
	9	265	2	75	50	23	50	2	0
	10	154	20	84	80	16	20	0	0
43 year old Stand	11	55	22	50	43	33	29	17	29
	12	44	3	79	0	21	100	0	0
	13	144	37	64	38	31	46	5	16
	14	48	2	73	50	21	50	6	0
	15	68	5	83	60	16	40	1	0
	16	115	22	64	36	29	41	7	23

[1] Young-growth are trees that have developed since harvest; advanced regeneration are trees that were present at harvest but were 10 or less years old; Residual trees were greater than 10 years of age at harvest.

[2] Includes infected trees.

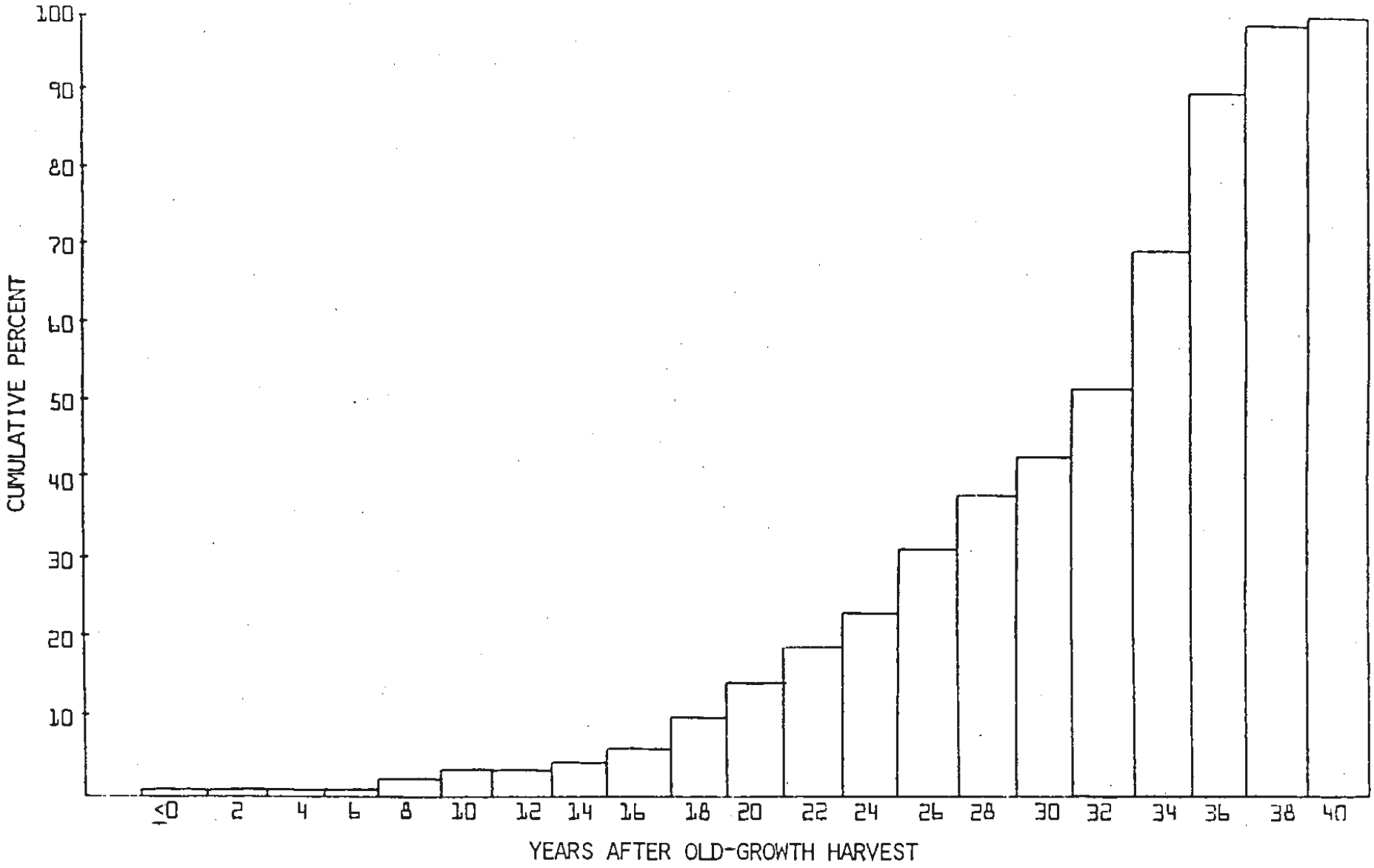

FIGURE 1. NUMBERS OF YEARS AFTER OLD-GROWTH HARVEST UNTIL DWARF MISTLETOE INFECTIONS BECAME ESTABLISHED ON REGENERATION BENEATH INFECTED RESIDUALS; BASED ON 452 INFECTIONS FROM 185 INFECTED TREES OF 1384 HEMLOCK EXAMINED AT CAPE POLE, SOUTHEAST ALASKA.

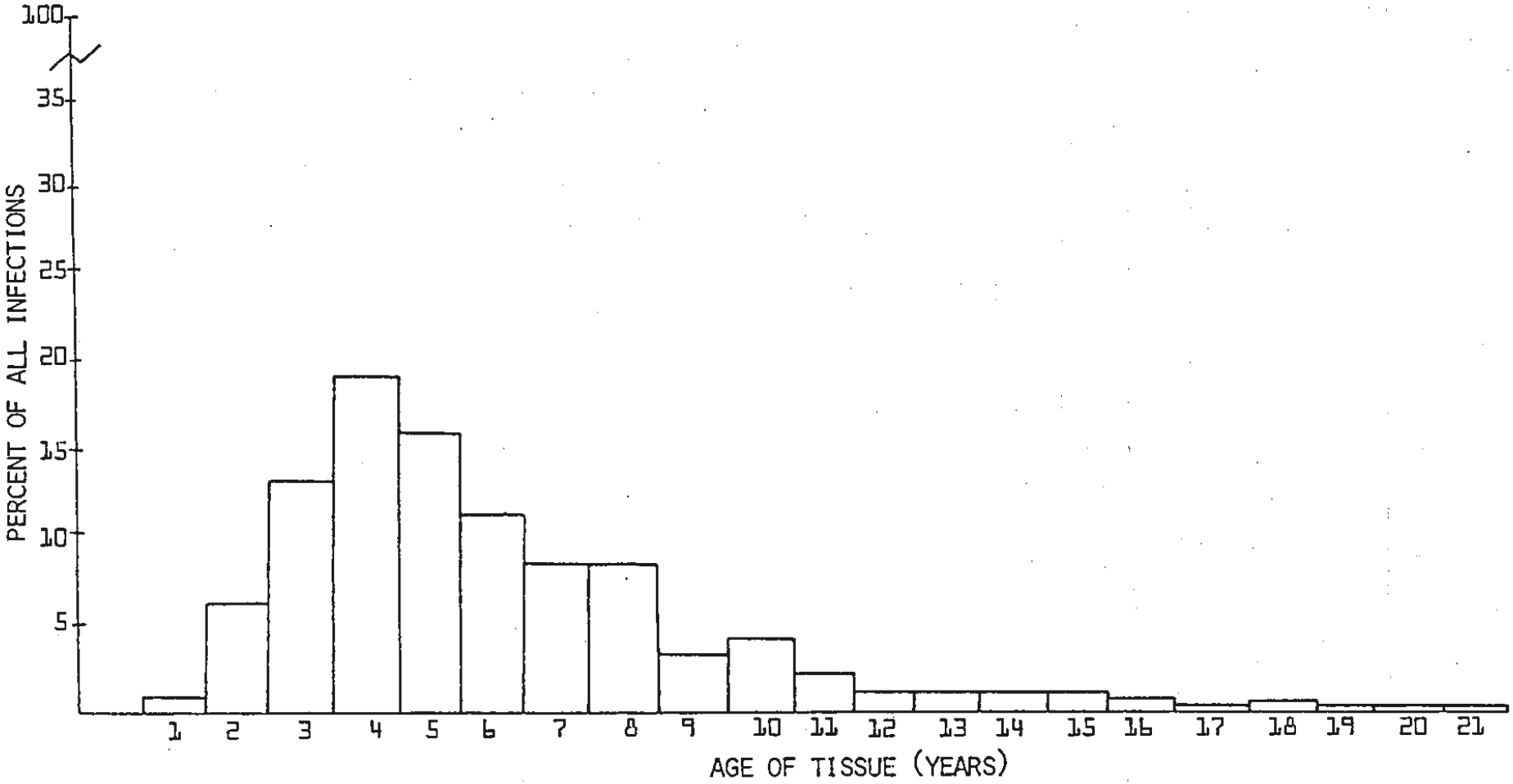

FIGURE 2. AGE OF TISSUE (YEARS) WHEN DWARF MISTLETOE INFECTIONS WERE ESTABLISHED; BASED ON 523 INFECTIONS COLLECTED FROM WESTERN HEMLOCK REGENERATED BENEATH INFECTED RESIDUALS AT CAPE POLE AND HOLLIS, SOUTHEAST ALASKA.

INFLUENCE OF DWARF MISTLETOE ON 40- TO 80-YEAR OLD WESTERN HEMLOCK STANDS ON THE WASHINGTON COAST

James S. Hadfield

Hemlock dwarf mistletoe is estimated to occur on 21 percent of the western hemlock-mountain hemlock type in Oregon and Washington (1). Well over one million acres are thought to be infected. According to loss estimates made by Childs and Shea, western hemlock suffers an annual loss of 42 million cubic feet from dwarf mistletoe (2).

Several hundred thousand dollars have been spent on hemlock dwarf mistletoe control in Oregon and Washington in the past 15 years. Control decisions were based upon published reports documenting the seriousness of mistletoes in other tree species and to a lesser extent in hemlock because few studies have been made on hemlock. A review of literature dealing with the impact of hemlock dwarf mistletoe indicates that most studies were conducted in stands today's foresters would consider past optimum economic rotation. Discussion with private and agency foresters managing hemlock for commercial timber production reveals that rotations now commonly range from 40 to 60 years with some carried for a maximum of 100 years.

Observations made in 40- to 100-year-old hemlock stands along the coast of Oregon and Washington seemed to indicate that dwarf mistletoe was not causing as much damage as previously thought. A moratorium was placed upon spending additional funds for hemlock mistletoe control until disease impact could be better documented.

Evaluations of the impact of hemlock dwarf mistletoe were made in two stands on the Washington coast.

A western hemlock stand scheduled to be clearcut was located on the Quinault Indian Reservation. Trees in the stand averaged 43 years of age. It had never been thinned. This level of management is typical of stands on the Reservation. The stand had developed on a site previously occupied by hemlock that had been clearcut. Scattered dwarf mistletoe-infected residuals from the previous stand remained. Ten plots were established in the stand. Five of these plots were centered around single, obviously infected, residual trees. The other five plots were established in spots devoid of obvious residuals. The plots were 1/30 acre in size and were in the shape of a cross. This shape was chosen to attempt to separate effects of mistletoe infection from residual tree competition on tree growth. All plot trees were cut. Their stump

positions were mapped. DBH, age, diameters at various points along the stem, merchantable height, and total height were measured. Infections on all trees were flagged and their heights above ground were measured. All branches in each infected third of live crown were recorded. Disks were removed at the 4-inch tops. Radial growth in inches for the last 5 and 10 years was measured along two randomly-selected radii on these disks.

Total cubic foot volume of each tree was determined by using standard cubic foot tables for total and merchantable stem volumes and tariff access for western hemlock in Washington and Oregon developed and published by Weyerhaeuser (3).

Data were tabulated and statistically analyzed with a one-way analysis of variance with unequal replications. Analyses were made to determine if there were significant differences between stocking and total cubic foot volume between infected and uninfected plots. Using only data from the infected plots, I compared volumes of trees with varying intensities of dwarf mistletoe infection to determine if trees with higher infection ratings were smaller than those with low ratings. For this and other analyses, trees rated 0, 1, and 2 were combined into one group and trees rated 3, 4, and 5 were combined into another. This was done because of the small numbers of trees rated 4 and 5. No 6-rated trees were found. Radial growth at the 4-inch tops for the last 5 years was compared for trees with varying mistletoe infection intensities. Volumes of trees 0 to 10, 11 to 20, and 21 feet and further from the infected residual were compared to determine if the residual tree had contributed to slower growth. Mistletoe infection intensity was plotted against distance from the residual tree to determine spread patterns.

Results of this evaluation revealed there were no statistically significant differences in stocking or total cubic foot volume between infected and uninfected plots. Infected plots contained a total volume of 2,300 cubic feet versus 2,076 for uninfected plots.

In infected plots, volumes of trees rated 0 to 2 were not statistically different from those rated 3 to 5 at the 95 percent level. Also, trees rated 0 to 2 showed no statistically significant greater amounts of radial growth at the 4-inch top for the last 5 years than did those rated 3 to 5. A tree rated 3 had more radial growth (1.25 inches) than all others.

I was surprised to learn there were no statistically significant volume differences between trees growing 0 to 10, 11 to 20, and 21 feet and further from the residual trees on infected plots. The two largest trees, 65 and 76 cubic feet, were growing within 12 feet of the residuals.

Range of infected tree ratings on infected plots is shown in Table 1. Few trees rated higher than 3.

Table 1.--Range of Dwarf Mistletoe Infection Severity on Five One-Thirtieth-Acre Infected Plots on the Quinault Indian Reservation

	Dwarf Mistletoe Infection Severity						
	0	1	2	3	4	5	6
Percent Trees	45	21	14	11	6	3	0
	Total 65 trees						

Infection pattern around infected residuals is displayed in Table 2.

Table 2.--Percent of trees infected by severity classes in relation to distance from the infected residual tree.

Distance (ft)	Dwarf Mistletoe Severity		
	0	1-2	3-5
	Percent Trees		
0-10	14	4	31
11-20	21	48	38
21+	65	48	31

Trees farthest from infected residual trees tended to be less severely infected. Only six trees rated 4 or 5 were found, all were within 20 feet of residuals.

In this 43-year-old stand, dwarf mistletoe had not caused any significant growth impact under conditions that I feel should have been ideal for spread. Tall, obviously infected residuals had been left to infect a pure stand of developing hemlock. It was surprising to see how limited mistletoe spread was around the infected residuals. Possibly the dense stocking of the developing stand restricted movement of mistletoe from second-growth tree to second-growth tree. Dense stocking would not have prevented spread from residuals to second-growth. Apparently, much spread did result from the residuals and not from second-growth to second-growth because on three of the five infected plots, uninfected trees were positioned between the residuals and infected second-growth.

I believe that dwarf mistletoe had not caused significant growth reductions in this stand for two reasons. Most infections were low in the crown where light was poor because of dense stocking. As a result, vigor of mistletoe plants was probably poor. In fact, few live shoots were seen in the lower crowns of infected trees. As a result, seed production was probably not copious. Secondly, tree height growth averaged slightly over 2 feet per year in this stand. It was probably greater earlier in the life of the stand. I believe annual tree height growth substantially exceeded annual vertical extension of female mistletoe plants. Trees were not infected severely enough for noticeable growth rate reductions. It is generally thought that dwarf mistletoe does not cause significant growth loss until trees have infection ratings greater than 3. Average time interval for hemlock dwarf mistletoe to increase one infection class is not known; however, I believe it would take at least 10 years, since tree height growth is rapid early in stand development and available light is poor because of rapid crown canopy closure. For these reasons, it is unlikely that many trees would develop serious infections in a 40- to 50-year period on coastal sites unless there are large numbers of infected residuals left.

Another area used to measure mistletoe impact was the Hemlock Experimental Forest, located approximately 13 miles north of Hoquiam, Washington. This 73-year-old stand has been the site of many experimental thinnings, the first being instituted in 1952.

This stand provided a distinct contrast to the Quinault stand because it was 30 years older and portions had been thinned 26 years before the evaluation. Twenty trees selected on a grid pattern had been cut for another disease evaluation in each of four separate areas in the stand. Two of these areas had been thinned, two had not. Total cubic foot volume, position and number of mistletoe-infected branches, and age were measured. Three trees were excluded from data analysis because they were residuals from the previous stand.

Data from the two unthinned areas were combined after no statistically significant differences in tree volumes were found. Data from the thinned areas were combined for the same reason.

Range of dwarf mistletoe ratings encountered on sample trees is shown in Table 3. In thinned portions of the stand, 17 percent of the infected trees rated higher than 3. If the 3 residuals from the former stand are dropped from the analysis, then 56 percent of the infected trees in the unthinned portions rate higher than 3. It is likely some of the more severely-infected trees were removed from the thinned portions during previous logging operations.

Table 3.--Range of dwarf mistletoe ratings in the Hemlock Experimental Forest

	Dwarf Mistletoe Rating						
	0	1	2	3	4	5	6
	Percent Trees						
Thinned[1/]	35	12.5	22.5	17.5	5	7.5	0
Unthinned[2/]	51.3	2.5	12.8	2.5	7.7	12.8[3/]	10.3[4/]

1/ 40 trees
2/ 39 trees
3/ 1 tree was a residual from the former stand
4/ 2 trees were residuals

Data from the two combined areas were analyzed by a one-way analysis of variance with unequal replications to determine if mistletoe had caused differences in growth. Trees rated 0, 1, and 2 were grouped, as were those rated 3, 4, 5, and 6.

In unthinned portions of the stand, there were no statistically significant differences in total cubic foot volume between the two infection groups. One of the largest trees (172 cubic feet) rated 6. A separate analysis showed no significant volume differences when trees with ratings 0 and 1, 2 and 3, and 4, 5 and 6 were grouped and compared.

There was a statistically significant difference ($P = .05$) in tree volumes in the thinned areas. Trees rated 3 to 6 were larger than those rated 0 to 2. The largest tree (403 cubic feet) rated 5. Volume of 0 to 2-rated trees averaged 104 cubic feet, 3- to 6-rated trees averaged 159 cubic feet. Measurements taken of the last 10 years' radial growth at the stumps and 4-inch tops were analyzed to determine if there were differences. Radial growth for the last 10 years measured at stumps was significantly greater ($P = .05$) for trees rated 3 to 6 than trees rated 0 to 2. There were no differences between radial growth of the two groups at the 4-inch tops or in 10-year leader growth.

Forty-two different trees were selected at the Hemlock Experimental Forest to determine the possible influence of dwarf mistletoe on log grades. I repeatedly observed that infected hemlocks had live branches below the main portion of the live crowns. These lower branches were found to almost always be infected. Mistletoe is clearly prolonging the life of these branches. I suspected that dwarf mistletoe was lowering log grades because branches were retained that normally would have died and fallen off. Trees that displayed a wide range of mistletoe infection were nonrandomly chosen for sampling.

Cutters felled the trees and bucked them into standard log lengths. Number and location of live infections were recorded for each tree. All branches were cut off after they were examined.

An experienced log grader from the Grays Harbor Log Grading and Scaling Bureau was hired to grade each log. A total of 136 logs were produced from 42 trees.

Data were tabulated and analyzed to determine if mistletoe had lowered log grades by causing branch retention or stem swellings.

Of the 136 graded, only one log was obviously graded lower than its potential because of the effects of mistletoe. The log was cut from a residual of the previous stand. It contained a large stem swelling about 6 feet above ground. Had this swelling not been present, the log potentially could have been graded special mill.

The major factor influencing hemlock log grades is diameter of the logs. For example, any log less than 12 inches in diameter at the small end cannot be graded higher than 3 and any log less than 6 inches is a 4. Influence of dwarf mistletoe infections on diameter growth of these trees was not investigated because the trees were not randomly selected. In the previously described evaluation at the Hemlock Experimental Forest, dwarf mistletoe did not reduce growth of trees; therefore, I conclude that for that site it was not a significant factor in lowering log grades.

The reason for trees rated 3 to 6 being larger than those rated 0 to 2 in the thinned areas is not clear. One possible explanation is that the first trees to become infected in a developing stand are those which are growing most rapidly. Such trees present more target area to receive mistletoe seeds than smaller trees. These larger trees may have maintained their competitive advantage in spite of mistletoe infections, particularly since infections tend to be concentrated in the lower portions of crowns in young, dense hemlock stands. When the stand was thinned, these trees may have responded better than did smaller trees. It is also likely that the mistletoe responded to the thinning and increased one or more infection classes. There are indications that recent volume increment by the 3 to 6-rated trees is slowing more than volume increment in less severely infected trees. I would anticipate that if the stand was to be left, eventually trees which are now less severely infected will exceed the size of trees presently rated 3 to 6. This will not happen however, because the stand is already past common hemlock rotation age for coastal sites and is scheduled for clearcutting in about 2 years.

In summary, hemlock dwarf mistletoe has not caused significant damage in the 43- and 73-year-old stands examined. Rapid tree height growth and crown closure, in particular, seem to be responsible for preventing serious mistletoe damage in stands growing close to their maximum biological potential.

If even-age management is practiced and rotations do not exceed 100 years, I believe hemlock dwarf mistletoe will not cause large losses on coastal sites in Oregon and Washington. This should be particularly true in 40- to 60-year rotations because periodic annual increment does not reach its maximum on coastal sites until 35 to 65 years (4).

Hemlock dwarf mistletoe should not be ignored. We should still encourage foresters to remove all hemlocks when infected hemlock stands are clearcut. However, the value of residual removal as a separate operation following logging is questionable if the following rotation is planned for less than 100 years. There may, however, be some point at which so many infected residuals are left that control may be desirable. Foresters should still be encouraged to discriminate against severely-infected trees during thinnings, but mere presence of infection should not make a tree an automatic target for the chainsaw. I feel that presence of hemlock dwarf mistletoe should not be used as the sole reason for delaying or avoiding thinning in coastal sites. In fact, thinning may be just what is required to increase the growth rates of infected trees in overstocked stands. Thinning, in all likelihood, will cause an increase in the mistletoe population, but if the trees have reasonable crowns, their growth rates will accelerate or, at a minimum, not decrease. These trees could be grown to merchantable size and the stand clearcut, at which time the parasite can be eliminated from the site. If no treatment is undertaken in infected overstocked stands, tree growth rates will naturally slow and it will take much longer for the trees to reach merchantable size.

Additional stand sampling is planned for Coastal and Cascade Mountain sites to help clarify the impact of hemlock dwarf mistletoe in managed stands.

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THE IMPACT OF FOMES ANNOSUS INFECTION IN YOUNG-GROWTH WESTERN HEMLOCK 11 YEARS AFTER PRECOMMERCIAL THINNING

Theodore D. Chavez, Jr.
Charles H. Driver
Robert L. Edmonds

Introduction

In 1967, the University of Washington, College of Forest Resources with the state Department of Natural Resources, set up a series of 0.1 acre study plots on DNR land near Clallam Bay, Wa. (Edmonds, 1968). The intent of the research on these plots was to measure the occurrence of stump infection by Fomes annosus (Fr.) Karst in a 15 year old precommercially thinned stand of western hemlock. Study plots were thinned from an estimated 2500 stems/acre to approximately 300 stems/acre.

Stump infection ranged from 60-100% with highest infection in June. The stand was slightly infected prior to thinning, but no more than 12% of the trees were infected. In addition, stump colonization by F. annosus in relation to stump diameter class from 1.5 to greater than 2.5 in. was recorded. All classes were similarly infected with a slightly higher infection in the larger stumps.

After 11 years the stand was re-visited in order to determine the extent of infection in the remaining crop trees in two untreated naturally infected plots, thinned in February and May, 1967, respectively. The specific objectives of this study were to determine: (1) the extent of infection by F. annosus in precommercially thinned young-growth western hemlock residuals, 11 years after documented stump infection (Edmonds, 1968), (2) approximate rate of stand infection and course of spread of F. annosus, and (3) if infection by F. annosus suppresses height or diameter growth of western hemlock.

Materials and Methods

Two 0.1 acre study plots, consisting of precommercially thinned 26 year old, naturally regenerated western hemlock, were selected for intensive examination. Initially, detailed stand maps were prepared to aid in pinpointing location of stumps in relation to live trees. A grid system was used for map construction. Selected trees were felled and 2 in. disk samples, collected at 2 ft. intervals from ground level to incipient decay extent when necessary, were taken to the laboratory infection determination. The incubation method was used for detection of F. annosus (Rishbeth, 1950). In addition, on selected trees internode length and annual ringwidth measurements were

recorded for later use in assessing both height and diameter growth impact. Selected tree/stump groups were then hydraulically excavated to a depth of approximately 18 in. using portable fire pumps and water from a nearby creek. A series of sections were then made on exposed roots of residual trees, tracing the fungus to source using incipient decay as a tracer guide.

Results and Conclusions

A majority of the crop trees in the two plots examined were found to be infected by F. annosus. Infection levels of 95 and 85% were detected for plots thinned in February and May, 1967, respectively. In contrast, only 12% of the trees on the February-thinned plot, and 4% of the trees on the plot thinned in May, were infected prior to thinning. Incipient decay stain data is summarized in Table 1. Source of tree infection in percent is shown in Table 2. Of particular interest here is that, with the two plots combined, 61% of the total infection was traced directly to stumps.

In conclusion, it was determined that: (1) untreated stumps resulting from precommercial thinning provide a significant infection court to remaining crop trees in young-growth western hemlock stand, (2) in all cases where infection originated from a stump, the infection source or stump was in close proximity (less than 2 ft.) to the live tree, (3) Fomes annosus spreads from infected stumps to surrounding trees at a mean rate of approximately 74.6 cm per year, with a range of 23-128 cm per year, (4) stain volume/total volume to a 4 in. top ranged from a minimum of 0.003% to a maximum of 19.9%, and (5) F. annosus has no appreciable effect on height or diameter growth of western hemlock.

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Table 1. Incipient decay stain height and volume from trees infected with with Fomes annosus.

Plot No.	Stain Height (ft.) Max.	Ave.	Min.	Stain volume/Total to 4" top Max.	Ave.	Min.
21 (thinned Feb. 1967)	38.7	11.3	0.6	4.8	1.0	0.003
15 (thinned May 1967)	36.2	13.5	5.7	19.9	2.3	0.1

Table 2. Source of tree infection after thinning (percent).

Plot. No.	Stump	Animal Damage/Wounding	Both	Uncertain
15 *	70	6	6	18
21 **	73	42	0	5

* 17 of 20 trees infected
** 19 of 20 trees infected

LOSS DUE TO ROOT AND BUTT DECAY IN MANAGED WESTERN HEMLOCK STANDS

Don Goheen

Western hemlock (Tsuga heterophylla (RAF.) Sarg.) has a reputation for proneness to infection by decay fungi, especially Fomes annosus (Fr.) Cke. In the past, decay losses have been substantial. Studies in old growth stands have shown 25 to 52 percent of the cubic foot volume lost due to decay.

In recent years, there has been a shift in hemlock management. We are now using rotations of 40-125 years rather than 150-300 years. What kind of decay losses can we expect in these younger stands? Also, what effects will the more intensive management practices that accompany these shorter rotations (precommercial and commercial thinning) have? To begin to answer these questions, Forest Insect and Disease Management, Region 6, U.S.D.A. Forest Service carried out an evaluation during the summer and fall of 1978.

METHODS

We examined eight pairs of hemlock stands in western Washington and northwestern Oregon. Each pair consisted of a commercially thinned stand that had been thinned at least 10 years before the evaluation and an adjacent unthinned stand of similar age and character. All stands were between 40 and 125 years old.

In each of the 16 sample stands, a grid of sample points was established. The grids were composed of four lines equally spaced through the stand with 5 plot centers at 100 foot intervals along each. Basal area data was taken at each of the 20 plot centers in each stand. A 20-factor prism was used in thinned and a 40-factor prism was used in unthinned stands. Diameters of all "in" trees were measured and the first "in" tree to the north of each plot center was singled out for intensive sampling.

On each intensively sampled tree, four roots corresponding to the cardinal directions were excavated to a distance of 3 feet from the root crown, 6-inch-long sections were excised from each, and these samples were returned to the laboratory for isolation. Following root sampling, each sample tree was felled. Total height and height to a merchantable top (4 inch diameter) as well as diameters inside bark at the stump and at 40 feet were measured and used to calculate tree volume. Leader length for the last 10 years was also measured. If there was evidence of decay at the stump, the sample tree stem was dissected in 1-foot sections as high as decay extended. Decayed areas were outlined on all sections and photographed with a polaroid camera on a calibrated stand. Photos were planimetered in the laboratory and volume of decay was calculated using Smalian's formula. Size and age of all wounds on the stems of sample trees were determined in the field and volume of decay associated with each was estimated using the regression formula developed by Wright and Isaacs.

Sections cut from stump level, ground level, and the 4-inch top were returned to the laboratory for growth measurements and isolations. Ten-year radial growth measurements were taken across 4 randomly chosen radii on the stump and 4 inch top discs. Stump and ground level discs had isolations made from any decay or stain onto selective media. They were then washed in a 10 percent chlorox bath, wrapped in newspaper and incubated for 2 weeks, after which time they were examined for oediocephela of F. annosus. Fomes annosus fruited luxuriantly on infected material, and we got nearly perfect correlation between incubation and isolation results.

RESULTS AND DISCUSSION

We found high levels of infection by decay fungi in all stands examined (Table 1). However, amount of actual decay loss in these young stands was relatively low (Table 2), averaging about 2.8 percent of the cubic foot volume in all stands. Most of the decay that was encountered was associated with wounds and F. annosus was by far the most frequently associated fungus. We also found no statistically significant differences in growth between infected and uninfected trees. Our results suggest that, though young western hemlock are very susceptible to infection by decay fungi, especially in stands where there is damage associated with thinning, considerable time is required before substantial amounts of wood are actually destroyed or growth is affected.

Apparently, the short rotations now being employed do not allow the fungi sufficient time to cause the kind or magnitude of damage observed in old growth stands.

Table 1: Average decay fungi infection levels found in eight thinned and eight unthinned western hemlock stands

	% Trees Infected by:		
	Fomes annosus	Armillaria mellea	Any Decay Fungi
Thinned	26.2	11.2	41.9
Unthinned	13.1	5.6	23.1

Table 2: Average percent of cubic foot volume lost to decay in eight thinned and eight unthinned western hemlock stands

	% Ft^3 Vol. Lost to Decay		
	In Butt	In Stem	Total
Thinned	0.9	1.3	2.2
Unthinned	0.6	2.8	3.4

Panel: Integrated Management of White Pine Blister Rust
G. I. McDonald, Moderator

Panel Introduction

White pine blister rust has been a major forest disease problem in North America for nearly 80 years. The absence of a workable management strategy stands as one of the major failures of our discipline. All the elements are now available to develop and use a cost effective management strategy. Forest managers need no longer be handicapped by the unavailability of key white pine species.

The objective of the rust management plan should be to balance risk, cost and benefit so as to maximize genetic breadth of the pine hosts and to minimize the biologic and dollar costs of control. White pines are a necessary part of the mix of species in Western North America because of their excellent growth rate, ease of regeneration, and resistance to frost and root pathogens. Two principal risks of the proposed program are maladaptation of genetically manipulated populations to physical and biological (pests) factors of the environment and future susceptibility of blister rust resistant materials to variation in the rust. The major cost of an integrated program relates to reduction of these risks. The first line of defense should be maintenance of the largest possible genetic breadth of the pine population. This means use of highly resistant materials only where they are needed (resistance-hazard alignment) and the widespread and judicious use of silvicultural, biologic, and chemical control means on a need basis. This panel will discuss some of the pertinent elements of integrated plans as well as the skeleton of three plans currently under development. Ray Hoff provides a rundown on the development of blister rust resistance up to establishment of seed orchards and seed production areas in north Idaho. Bill Randall will provide a summary of the U.S.F.S Region 6 resistant white pine and sugar pine program. Ray Steinhoff will discuss new findings about the patterns of genetic diversity in western white pine. Neil Martin will examine new and past findings about the influence of pruning and thinning on rust development in young stands and Everett Hansen will take a look at racial variation in Cronartium ribicola and the genus Cronartium. Finally, I will try to knit these pieces together into three specific plans directed toward three different management situations.

PROGRESS IN BREEDING BLISTER RUST RESISTANT WESTERN WHITE PINE

by

R. J. Hoff

In 1959 Bingham presented a paper to WIFDWC on the progress in Breeding Blister Rust Resistant Western White Pine. He described the level of resistance and predicted a genetic gain of 21% per generation. He also predicted that large-scale planting of resistant stock would be possible in 20-30 years.

In 1969 Bingham presented a paper describing the development of seed orchards that would provide the seed for production planting. He also discussed research on the resistance system, the observations of races of blister rust and touched on philosophies of plant breeding that would have the highest likelihood for producing a stable variety.

Now, in 1979, I would like to describe what happened. The time span in the field of forestry nearly always is greater than one person's working career. I will describe two phases of work. Phase one is the work that was started by Bingham in 1950 and ended in 1974, when he retired, with the production of four seed orchards, one breeding arboretum, and seven research outplantings, with over 20,000 trees, half of which are pedigreed stock.

PHASE I PROGRAM

The base population includes the western white pine stands of northern Idaho, northwestern Montana, and northeastern Washington. Blister rust entered the area in or around 1923, was generally distributed over the area by 1937-41, and has been causing severe damage (presently up to 95% mortality) from 1945 to present.

400 rust-free phenotypically resistant candidate trees were located in heavily infected natural stands, 1950-55.

Preliminary research based on mating many pairs of candidates, artificially inoculating the progeny, indicated an average resistance of about 30%, a heritability of .60 and an estimated gain of 20% by selecting the best 25%, 1953-65.

The best 24 trees that had high resistance over several crosses (GCA - General Combining Ability) were selected for each of three elevational zones (less than 3300', 3301'-4100'; 4101' +) The 12 within-zone pairs were mated and 36 F_1 progenies artificially inoculated. Healthy F_1 seedlings were selected for each mated pair for seed orchards, 1963-73.

Meanwhile more preliminary research showed that there were several mechanisms of resistance involved, 1964-present (see table 1). This resulted in a change in the seedlings selected for the seed orchards. Although it was too late to select for most of the resistance mechanisms observed, selection was made for two foliage and one bark resistant type.

3 seed orchards established, 13 acres low-elevation with 1,440 trees, 21 acres mid-elevation with 2,160 trees and 7 acres high-elevation with 756 trees.

The level of resistance in the seed orchards is expected to be between 60 and 70%, made up of four of the mechanisms listed in table 1 - type 5, 8, 9 and 10.

Production of seed in the seed orchards is just starting. However, the "Moscow Arboretum" which is a collection of resistant F_1 seedlings established for breeding purposes has produced over 400 pounds of seed from 1974 to 1979.

Cost of the Phase I program was about $1.8 million, including research, development and seed orchard establishment.

PHASE II PROGRAM

This program was started in 1966 by Bingham when it was recognized that with only the 400 candidates to choose from (this boiled down to 73 after selection) there would not be enough of a genetic base to select for other traits, e.g., volume production and form - especially so if one had to work within each of the elevation zones. Later it was found that there were many mechanisms of resistance, some that had much higher probability of being more stable than others. And that the resistance that Bingham had selected for using the GCA method were largely controlled by single genes.

The base population is the same as Phase I.

3100 phenotypically resistant candidate trees were located in heavily infected natural stands, 1966-72. Notice that these need not have been rust-free. By this time we also knew that surviving but infected trees within very high infection centers also carried some resistance genes. If trees within a stand had 10-20 cankers per tree, no cankers were allowed on the selected tree versus if 150 cankers or more were found, the selected trees could have 4 or 5 cankers. Our data really isn't good enough to get that specific but you get the idea.

Open pollinated seed is being used in the selection tests. Because of the high amount of resistance, and because of the difficult task of making two parent crosses, it was decided to base this program entirely on open pollinated seed, 1968 - to date.

Meanwhile, still more research by R. J. Steinhoff and G. E. Rehfeldt at the Moscow lab has shown that there is essentially one seed zone for inland western white pine. This was surprising because we are quite used to breaking up genetic populations of forest trees in fairly small adaptive units. The only difference found was that the high elevation trees performed less well than the low elevation trees - still this amounted to only 2% of the total variation.

One seed orchard is all that is really needed for the entire Inland Empire -- that is what the data says. However, that is an awfully big area and so we are planning on three, one for high elevations (4500'+) for the entire area and two for elevations less than 4500'.

Artificial inoculation was completed on over 100,000 seedlings in 1978. The next test is planned for inoculation in 1980, and etc. until all candidates have been tested.

Seed orchard establishment will begin in 1983. Hopefully, all seed orchards will be completed by 1988.

Selection criteria include getting rid of slow growing trees - 15 to 20% slowest, and for resistance to blister rust as follows:

1. Individual and family selection for reduced frequency of secondary needle spots (type 6, table 1).

2. Family selection for slow fungus growth in the secondary needles (type 7, table 1).

3. Family selection for slow fungus growth or tolerance in the stem. At the seedling stage it is impossible to separate these mechanisms (types 11 and 12, table 1).

4. Individual selections for prevention of needle infections (type 5, table 1), premature shedding of infected needles (type 8, table 1), short shoot reaction (type 9, table 1), and stem reactions (type 10, table 1).

The level of resistance, i.e., number of uninfected seedlings, that we hope to produce in this new variety would be 50-60%. Nature can adjust it to the required balanced condition.

Philosophic base for selection for blister rust resistance we feel must contain these elements.

1. Never base resistance on only one gene. A variety of western white pine could be produced that would be 100% clean (no cankers at all), but how long would it last?

2. Use combinations of resistance types - as many genes as possible - diversity is essential.

3. Incorporate as many horizontal resistance types as possible. Although these don't stop the life cycle of the rust, they can give the whole system stability.

4. Maintain a fairly low gene frequency for all the critical resistance types 0.3 to 0.4 or less.

Natural Selection - An Alternate Breeding Method

When the fungus first came to the inland forest the level of resistance, i.e., the number of white pine seedlings that were alive after artifical inoculation, was less than 1%. But now that has changed. In stands that have sustained high mortality due to blister rust (80-90%) that resistance is near 20% (table 2).

With this level of resistance, nature can do the job. The only real drawback is that nature's way is a little too slow for us. So let's speed it up. Stands like those in table 3 could be selected, all the dead white pine and other species removed (if desired), a seed bed prepared and the stand regenerated with partially resistant stock. The number game for the reproduction capacity for white pine in such a situation is shown in table 4. We did this for Pete's Creek (table 3). At the time the site was ready for seed (fall 1974) there were 26 trees per acre left. Some trees were removed because they were almost dead, others because they were in a good site for a landing, etc.

The real benefits of using natural selection is that nature selects for all the mechanisms of resistance (we can select only for those we see). Nature uses all races of blister rust over many inoculations years (we inoculate once with a mixture from several sites). We are going to continue to produce seed orchards mainly because there are many sites that need to be planted and also because improvement can be made for growth and form. Nonetheless, over the long tern, natural selection is an ace in the hole. If everything else fails, this method probably will not.

More preliminary research has shown the possibilities of using a computer disease model to determine the effectiveness of each mechanism of resistance and for determining the possibilities of integrated control.

Table 1.--Observed resistance mechanisms in *Pinus monticola: Cronartium ribicola* system

	Mechanism of resistance	Resistance type	Hypothesized inheritance	h2
1.	Resistance in secondary needles to a yellow-spot forming race	Vertical[1]	Recessive gene	
2.	Resistance in secondary needles to a red-spot forming race	Vertical	Dominant	
3.	Resistance in secondary needles to a yellow-green-island spot forming race	Vertical	Dominant gene ?	
4.	Resistance in secondary needles to a red-green-island spot forming race	Vertical	Dominant gene ?	
5.	Resistance in secondary needles that prevents spot formation	Vertical	?	
6.	Reduced frequency of secondary needle infections	Horizontal[2]	Nondominant gene ?	0.37
7.	Slow fungus growth in secondary needles	Horizontal	Polygenic	0.46
8.	Premature shedding infected secondary needles	Vertical	Recessive gene	
9.	Fungicidal reaction in short shoot	Vertical	Recessive gene	
10.	Fungicidal reaction in stem	Vertical	Oligogenic ?	0.367
11.	Slow fungus growth in stem	Horizontal	Polygenic ?	0.21-0.46
12.	Tolerance to infection	Horizontal	?	

[1]Vertical resistance types. This type is most often inherited as major genes and it often has a presence or absence effect. Also called specific and differential resistance.

[2]Horizontal resistance. This type is most often inherited by polygenes or at least it is oligogenic and differences are therefore usually quantitative. Also called field, general, and uniform resistance.

Table 2.--The level of resistance of seed lots collected from rust-infected western white pine stands with high mortality

Control lot	PROGENY TEST 1964 (4 yr after inoc.) Uninfected plants	Total plants	Uninfected plants	1965 (3 yr after inoc.) Uninfected plants	Total plants	Uninfected plants	1966 (2.5 yr after inoc.) Uninfected plants	Total plants	Uninfected plants
			Percent			*Percent*			*Percent*
DD[a]	12	85	14.1						
NN[a]	22	78	28.2						
OO[a]	10	88	11.4				4	40	10.0
SS[b]	17	69	24.6						
WW[b]	34	81	42.0	38	204	18.6	29	138	21.0
QQ[b]	11	37	29.7						
RR[a]	11	60	18.3	43	195	22.1	0	41	0
VV[b]	27	82	32.9				5	49	10.2
YY[b]	6	78	7.7						
XX[b]	14	61	23.0	13	121	10.7	36	145	24.8
TT[a]				23	150	15.3	29	133	21.8
ZZ[b]				28	65	43.1			
NR11 x Mix[c]				9	64	14.1			
NR12 x Mix[c]				1	10	10.1			
NR13 x Mix[c]				2	64	3.1			
NR14 x Mix[c]				3	17	17.6			
Total	164	719	22.8	160	890	18.0	103	546	19.0
Grand total	427	2,155	19.8						

[a] Collections from five wind-pollinated, rust-infected trees.
[b] Collections from squirrel caches.
[c] Crosses made on specific trees with a mix of pollen from 10 rust-infected trees.

Table 3 shows the frequencies of these three mechanisms of resistance. For comparative purposes, we have included data from the F_1 and F_2 crosses of resistant candidates and their progeny. Compared to the level of resistance in the original populations, considerable selection for these resistance into three categories, two of which are presumably controlled by single genes, we have no data concerning the total number of resistance mechanisms nor of genes present in these surviving trees. An extensive study is underway that is expected to provide answers to these questions.

Table 3.--Total living western white pines per acre in four high-mortality stands

Stand	Tree total	Total living	Mortality
			Percent
Bertha Hill	390	70	82
White Rock Springs	190	48	79
Elk Creek	182	20	89
Pete's Creek	434	46	89
Average	299	46	88

Table 4.--Potential numbers of resistant white pine seedlings that could be produced per acre through mass selection in stands with high mortality

Cones per tree	28
Seeds per cone	120
Seeds per tree	3,360
Established seedlings (2 percent of seed in 6 years)	67
Years for natural regeneration, 5 to 10; choose 5 years	5
Established seedlings per tree	335
Average living trees per acre	46
Total established seedlings per acre	15,410
Percent resistance	19.8
Established resistant seedlings per acre	3,051

VARIATION IN WESTERN WHITE PINE

R. J. Steinhoff

In any tree planting operation, whether routine reforestation or as part of an improvement program for either pest resistance or growth, one must be concerned with maintaining adaptation and growth potential. This is the reason for seed zones and seed transfer rules and research into geographic variation patterns and efforts to find or develop trees which are widely adapted. In an improvement program the extent and patterns of variation are major factors in determining how many seed orchards are needed.

VARIATION IN INTERIOR WHITE PINE

For northern Idaho, and adjacent parts of Washington and Montana, there is lots of variation in the growth of western white pine but very little pattern to the variation (table 1). That is, most of the variation is among saplings of the same family or among families from the same stand. Differences among the means for larger areas appear to be minimal.

Saplings from two areas, Trestle Creek and the Clearwater National Forest, give some indication of growth differences related to elevation of seed source. However, for one area the higher elevation saplings grow faster than those from lower elevations while for the other area the reverse is true. In other tests there is an indication that saplings from high elevation seed collections may generally be a little slower growing but the difference is probably not much larger than 5 percent of the mean.

COMPARISON OF OLYMPIC PENINSULA, CASCADE, AND INTERIOR WHITE PINES

Along with the north Idaho white pine seedlings we also planted a few from the east and west sides of the Olympic Peninsula, some from the Cascades near Mt. Rainier, and some from the Blue Mountains of southeastern Washington. Although most of these non-local seedlings are slightly shorter than those from north Idaho (table 1) most of the difference can be attributed to the fact that many of the non-local seedlings are a year younger than the local ones. This age difference arose because of insufficient stratification of the non-local seed which were added to the main study as an afterthought.

Some north Idaho seedlings have been planted on Vancouver Island by MacMillan Bloedel along with local seedlings. After 5 years in the field the north Idaho seedlings were slightly taller but the difference was not significant statistically.

No differences have been detected in the survival rates of local and non-local trees in north Idaho. There has been no damage to any trees from late spring or early fall frosts nor has there been any mortality directly related to winter cold even though temperatures reached -30°F last winter. Some sunscald damage to foliage occurred on both local and non-local trees last winter.

These results are very different from those we've had with coastal Douglas-fir and grand fir when grown in north Idaho. Coastal Douglas-fir seedlings initially grow much faster than interior ones but cannot tolerate our winter weather. Coastal grand fir grows faster than interior but appears to have less cold hardiness than interior seedlings. Coastal white pine grows at about the same rate as interior and appears completely cold hardy.

USE OF FINDINGS

The implication for the Region 1 blister rust resistance breeding program is that a single seed orchard can supply seed for most if not all of the intended planting area with little if any growth loss or survival problems.

On a broader scale there appears to be little advantage to moving seed from the north coast to the interior or vice versa from a growth standpoint. However, the lack of differences should allow transfer of seeds, seedlings, or pollens that have unique rust resistance mechanisms without having to carry out long breeding programs to maintain growth or adaptability.

FUTURE RESEARCH

We are currently accumulating seed from additional stands throughout the Cascades and Washington and Oregon Coast Ranges as well as a few Sierra Nevada stands to verify the preliminary findings and extend the sampling area.

Table 1. Heights of 12-year-old western white pine saplings at the Priest River Experimental Forest [1/]

Seed Collection Elevation	Seed Collection Area: Kaniksu N.F. Lat.48°45'	Trestle Cr. Lat.48°15'	St. Joe N.F. Lat.47°	Clearwater N.F. Lat.46°30'	West Side Olympic Peninsula	East Side Olympic Peninsula	Cascades Near Mt.Rainier	Blue Mts. Southeast Washington
Feet	Height in feet and tenths							
2000		8.2			7.5[2/]	7.6		
2500	8.9	8.3		7.8				
3000	7.9	8.2	8.2	7.8				
3500	7.5	8.2	8.4	7.9				
4000	8.0	7.9	7.8	8.2				
4500		7.8		8.3			7.0	
5200		7.9		8.4				7.3
$\bar{x}$	8.0	8.1	8.1	8.1	7.5	7.6	7.0	7.3

[1/] Plantation elevation 2400'

[2/] Elevation less than 1000'

PRUNING - AN APPROACH TO MANAGEMENT OF BLISTER RUST

Neil E. Martin

Most problems have as many solutions as there are observers because each observer has formulated a solution in reference to the problem as defined, to the results of research, and to their experiences. My hope is that the information presented by each panel member will complement each other in such a way that a holistic perspective on the management of blister rust through silviculture of western white pine will be evident and logical. Western white pine is one of three ingredients in disease establishment and development. This tree host together with a supportive environment and a compatible strain of Cronartium ribicola result in a pathological condition termed Blister Rust. It is my privilege to contribute a silviculture approach to managing the blister rust problem through manipulation of the pine host.

The life cycle of C. ribicola is known in considerable detail and illustrates the heteroecious macrocyclic type. Three spore forms, urediospores, teliospores, and basidiospores develop on the leaves of the deciduous host, Ribes spp. The other two spore forms, pycniospores and aeciospores, are produced by the perennial infections of five-needled pines. The most effective means of permanently interrupting the life cycle would be to rid our forests of the perennial source, namely infected western white pine, and let the infected ribes leaves succumb to nature. Fortunately for this obligatory fungal parasite, the economic importance of western white pine pitted the eradication program against the ribes host. The ribes eradication program is now history and the involvement of ribes in the blister rust disease cycle remains a vital element.

Rogueing of diseased plants or its more sophisticated application, namely pruning of infected plant parts, is a fundamental principle vital to disease management. The removal of blister rust diseased branches as a means of protecting eastern five needle pines from imminent death is recorded at least as early as 1939. In 1969 Roger Hungerford, Intermountain Forest and Range Experiment Station, began installing research field plots designed to measure the benefits of pruning potential crop trees in western white pine plantations in northern Idaho. Pruning can achieve a number of important effects that impact the blister rust disease cycle. First, removal of branches, together with thinning a stand, opens the canopy to air movement and consequently drying of the foliage, a detriment to spore germination. Secondly, blister rust infections, which begin in the needles and then progress down the branches toward the bole, are susceptible to pruning for a number of years and, therefore, may be removed during any one of several inspections. Thirdly, removing branches reduces the target area for new infections.

Pruning treatments were applied only where thinning was done. Trees with bole cankers or branch cankers closer than 4 inches to the bole were removed in thinning. On one-half of the pruned plots only white pine were pruned as opposed to all species being pruned on the remainder. In all cases, all branches were removed from the ground upward to 1/3 of the tree height. However, pruning could not exceed one-half of the tree height or 17 feet. (In instances where infected and prunable branches existed above this limit select pruning was done.) Branches were pruned flush with the bole with hand pruners or small chainsaws. Other treatments consisted of no treatment at all (control) and thinning only.

Although six plantations were treated, this report will use the Johnson Draw plantation in the Clearwater National Forest, near Pierce, Idaho, to illustrate the trends in the results. Characterization of the Johnson Draw plantation at the beginning of the study is recorded in table 1.

Table 1.--Characteristics of the Johnson Draw study area in 1969.

Habitat type.	Tsuga/pachistima		
Elevation.	3,300	WWP regeneration. . .	planted 1951
			natural
Aspect.	N,E&S	WWP d.b.h..	2.5 inches
Slope.	10-35%	WWP height.	15.0 feet
Site index.	76-85	WWP/acre.	148
Acres.	105	Other/acre.	257
Harvest cuts.	Several to 1950		
Volume/acre.	40M		

The Johnson Draw area is considered a heavy infection area as illustrated in table 2. Data in table 2 also show that conditions for successful infection continue to exist in that on the average for the entire area during the recent 5 years 13 new infections were added to each tree.

Table 2.--New blister rust infections per plot tree.

	Cankers/tree			Lethal canker/tree		
	1969	1974	1979	1969	1974	1979
Johnson Draw	3.6	11.9	13.1	0.9	7.1	6.6
Greenhood	0.7	4.8	9.0	0.2	3.1	3.0
Blickensderfer	0.3	3.0	*	0.2	1.5	*
Snyder Creek	0.2	0.4	*	0.2	0.4	*
Two Cut Draw	2.4	3.0	*	1.0	1.9	*
Potter Creek	0.6	1.1	*	0.4	0.9	*

* Data not available

Not shown in table 3 but of significance is that in 1969 only 24-38 percent of the trees were free of infection. New infections reduced the number of rust free trees to 6-12 percent by 1974 and by 1979 C. ribicola had established itself in every study tree. By 1974, 44% of the pruned and 57% of the non-pruned study trees had non-prunable cankers, i.e, either in the bole or within 4 inches of the bole. This year, 1979, the percent of trees with non-prunable cankers in all plots ranged from 39 in the pruned to 78 in the non-pruned (table 3).

Table 3.--Current status of crop trees, Johnson Draw, 1979.

	Dead total	B.R. caused death	Living WWP	Bole infected	Percent non-treatable
All species pruned	8	1	109	48	44
WWP pruned	7	1	97	38	39
Thinned only	13	2	98	67	68
No treatment	12	7	94	73	78

Treatments had an effect on the number of blister rust infections and such trends implied in 1974 are becoming more clear in the data of 1979. The most effective was the pruning treatment (table 4). In this treatment the target area of the WWP is progressively reduced by pruning. Consequently, the number of accumulated infections in 1979 are about one-half of the totals in non-pruned trees. The concept of available target area impacting frequency of infection is further verified in thinned only vs. no treatment. Lower branches in thinned only plots are exposed to spore loads and sunshine due to absence of filtering (screening) by neighboring trees. In non-treated plots the trees are shading each others' lower crown which results in death of the lower branches and consequently, few accumulated infections as shown in the data of table 4, 34 infections with no treatment vs. 43 infections per tree in thinned plots.

Table 4.--Infections per tree, 1979, Johnson Draw.

	All species pruned	Pruned WWP	Thinned only	No Treatment
Rep 1	27.85	23.33	69.16	49.43
Rep 2	11.38	16.51	34.78	31.27
Rep 3	13.16	17.53	25.88	22.32
Ave.	17.46	19.12	43.27	34.34

Imposing a pruning treatment on the pine host is illustrated in this report as being biologically sound in that 68-78 percent of non-pruned trees are fatally infected whereas only 39-44 percent of pruned trees are so afflicted. The data in table 4 also contains a warning that is biologically sound. That warning is that the environmental ingredient contributing to this disease must be considered before pruning is indiscriminately applied to all stands. Numbers of cankers per tree in table 4 shows that replication 1 apparently has characteristics different from replication 2 and 3 due to location although they exist within a 105-acre plantation in the same drainage. Further analysis of the data in regard to micro-habitats, aspects, and moisture regimes may yield some insights into identifying environments that are compatible with the principle of pruning.

PATHOGENIC VARIATION IN CRONARTIUM RIBICOLA

E.M. Hansen

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What is this term "pathogenic variation?" Why don't I title this "Races of Cronartium ribicola" like I was asked? I started out that way, but quickly got lost in the many and ambiguous uses of the term "race" in the forest pathology literature. Hence, my retreat is to the more general topic of pathogenic variation. The existence of vertical pathotypes (Robinson 1969), or "races" of Cronartium ribicola is certainly pertinent, but it is only one aspect of the larger concern among blister rust workers for the ability of the fungus to overcome host resistance mechanisms.

Even though variation in pathogenicity is a fact of life in virtually every host-parasite system studied, it is still necessary to include the topic in a panel such as this. Plant pathologists and fungal geneticists may well get impatient and ask: "Why in the world should the Cronartium-white pine system be any less subject to rapid shifts in fungus virulence or aggressiveness than have been so thoroughly documented for pathogens of agricultural crops?"

Forest pathologists have elaborated their answer for the past 50 years. 1) It is the haploid stage that attacks the economic host in white pine blister rust, not the dikaryotic stage as in wheat stem rust. "Physiologic races are less likely in the haploid mycelium than in the dikaryotic form (Czabator 1971)." 2) The repeating, conidial stage is on the non-economic host (Snow et al. 1976). 3) The fungus is probably homothallic, with less chance for variation (Hirt 1964). 4) The introduction of the fungus to North America was probably limited to two incidents, consequently the gene pool must be limited. 5) There has been no evidence for pathogenic races in ribes or pine (Hahn 1949). I will address the theoretical arguments based on the life cycle of the fungus first, and conclude with a summation of the evidence for pathogenic variation in the genus Cronartium and C. ribicola in particular.

Because pines are infected by basidiospores, products of meiosis, it is argued that virulent genotypes will likely be altered before they reinfect pines. Not being a geneticist, I won't attempt the logical rejoinder, but will rely on the observation that, likely or not, vertical pathotypes do occur on the haploid host in at least four rusts including Puccinia graminis (Day 1972). More generally, meiosis with recombination and crossing over is more likely to create new pathogenic variants than to reduce their frequency.

Lack of a secondary infection cycle on pine will surely slow the buildup of new virulent strains adapted to the resistant pines of the future. The current epidemic should demonstrate to the most skeptical, however, the ability of a virulent pathotype to establish itself and spread destructively, without a conidial stage on pine. It is also argued that the obligate alternate host will exert a stabilizing selection on the fungus, but there is no evidence to support this. The possible existence of short cycled, pine-to-pine forms of C. ribicola (Peridermium strobi) (Saho and Takahashi 1973), renders the argument tentative, at best.

Homothallism versus heterothallism has been argued for years for the Cronartium rusts, but could not be resolved without genetic markers. The identification by McDonald and Hoff (1975) of needle spot color pathotypes finally allowed genetic analysis (McDonald 1978). C. ribicola is heterothallic, and it is possible that aeciospores and urediospores, as well as spermatia can effect fertilization.

An introduced fungus does likely start with a reduced gene pool. Most pathogens of agricultural crops started as introductions, however. The ability of fungi to respond to the selection pressure of new genes for resistance has been amply demonstrated.

Finally, this lengthy introduction leads to the crux of the matter, the evidence for pathogenic variation in Cronartium and particularly in C. ribicola. Albino strains have been reported for several Cronartium rusts (Kais 1966). While they do not differ in pathogenicity from wild-type yellow or orange-spored strains, their stability through segregation at meiosis demonstrates that a simply inherited character such as virulence, can persist through the complex rust life cycle. Perhaps careful single "aeciospore" inoculations with albino P. harknessii could provide evidence for the hypothesized meiosis in autoecious strains of Cronartium (Hiratsuka 1969).

Morphological similarities (Peterson 1967, 1973) suggest the relatedness of the Cronartium rusts, including those with only a Peridermium stage. Recent reports of new alternate hosts common to several rust species support old rumors, and open the possibility that the Cronartium gene pool is very large indeed. Cronartium quercuum and C. fusiforme are finally accepted as one species (Burdsall and Snow 1977). You can't tell P. harknessii from C. quercuum without a spore germination chamber, and legend persists that P. harknessii occasionally infects oaks in California (Peterson 1967). The demonstration of western gall rust infecting Castillija spp. has been repeated enough times that it has to be believed (Peterson 1967, 1973). Recent inoculations have shown that the C. ribicola, P. strobi, P. kamtschaticum complex infects both ribes and at least two genera of Scrophulareaceae, the common hosts of the C. coleosporioides complex (Hiratsuka and Maruyama 1976, Yokota et al. 1975, Wicker and Yokota 1976). Unrepeated inoculation experiments even link the European C. flaccidum complex to C. ribicola (Ericksson 1922). Common hosts suggest relatedness, but do

not necessarily imply a common gene pool, except in the evolutionary sense. This evidence does suggest the capacity of these fungi for pathogenic specialization, however. Fusiform rust is considered a forma speciales of C. quercuum; a horizontal pathotype adapted to a particular group of tree species (Burdsall and Snow 1977, Kais and Snow 1972, Powers 1972). Host specialization is evident even within eastern gall rust, with three forma speciales named for the host specific isolates on jack, virginia, and shortleaf pines. It seems a testable hypothesis that most of the Cronartium species are but pathogenic variants, forma speciales, of a single species, with pine-to-pine strains liberally scattered throughout.

Pathogenic variation has been documented more thoroughly for fusiform rust on both loblolly and slash pines than for any other Cronartium group (Powers et al. 1977, Snow and Kais 1970, Snow et al. 1972, 1975, 1976). A series of open-pollinated families of low, moderate, and high resistance were inoculated with C. fusiforme collections selected to test pathogenic variability within the fungus. Briefly stated, the results of repeated inoculations showed extensive variation between rust isolates from widely separated geographic areas, between several galls from single geographic areas, and between cultures derived from single aeciospores from a single gall. There was as much variation between single spore isolates from a single gall (Powers, Forest Science, in press) as between galls from widely separated geographic areas. In each case from 30 to 80% or more of slash pine seedlings of the nominally resistant family were galled by the range of isolates. One extensive test with loblolly pine had similar results (Powers et al. 1977). Mass selected loblolly pine from Livingston Parish, Louisiana, are reported to carry a moderate level of resistance to local fusiform rust isolates. However, in an extensive trial where these progeny were inoculated with 72 fusiform rust isolates from throughout the south, the resistance did not hold. An average of 54% of the seedlings were galled, but six individual rust isolates galled > 70% of the seedlings. Widespread planting of resistant pines exerts selection pressure on the fungus for virulence. Progeny of a slash pine family selected for resistance were outplanted in a block in a plantation with unselected, susceptible pines (Snow et al. 1976). After 11 years in the field, rust isolates were collected from the few galls that had formed on the resistant trees (4% infected) and from the heavily infected susceptible stand surrounding (44% infected). New seedlings from the same resistant family were inoculated with 9 rust isolates from each source. Rust isolates collected from trees of the resistant family infected an average of 77% of the seedlings, compared to 17% infection by "wild", unselected isolates.

The strategy of breeders for resistance to fusiform rust in the South seems to be to select for the highest possible resistance to a wide range of fungus isolates (Snow et al. 1975, Dinus and Schmidt 1977). Since parent trees are selected and progeny are evaluated only for presence or absence of active galls, however, there is a good chance that selection is for hypersensitive reactions (Miller et al. 1976). In

most host-pathogen systems these are simply inherited, and simply overcome.

In contrast, much is known of the resistance mechanisms in five-needled pines to Cronartium ribicola (Hoff and McDonald 1972; Hoff, these proceedings), but relatively little is known of pathogenic variation in the fungus. Anderson and French (1955) inoculated two Ribes varieties with aeciospores from several locations, and found that isolates differed in their pathogenicity to the Ribes. Geral McDonald (personal communication) has also shown a differential host-pathogen interaction when single aeciospore lines were inoculated to several clones of Ribes.

Evidence for variation in the fungus attack on pine began to accumulate in 1964 with the observation that red needle spots as well as the long-described yellow spots formed on white pine seedlings inoculated with blister rust at Moscow, Idaho. Subsequent careful work showed that some seedlings had only yellow spots, some had only red spots, while some had both types (Hoff and McDonald 1972). Furthermore, the frequency of spots/meter of needle length on seedlings with both yellow and red spots was nearly identical to the combined spot frequencies on the yellow-spots-only seedlings and the red-spots-only seedlings. This suggested a classical gene-for-gene relationship, with some seedlings resistant to the red spotting pathotype, some resistant to the yellow-spotting pathotype, and some susceptible to both (McDonald and Hoff 1975). This hypothesis was supported by single aeciospore cultures from yellow-spots-only and red-spots-only trees, grown on Ribes and reinoculated to pine (McDonald 1978). Unexplained, however, is the widespread occurrence of such a seemingly specific reaction both in sugar pine with C. ribicola (Kinloch and Littlefield 1977), and in maritime pine with C. flaccidum (Raddi 1976). Even more puzzling is the increased frequency of occurrence of the red spot type over time. A corresponding change in tree resistance has not been noted, and preliminary work showed no epidemiological fitness advantage for the red strain (McDonald, personal communication). Both color types appear equally aggressive on white pine.

A new, virulent race of C. ribicola has just been demonstrated at the Dorena Tree Improvement Center, Forest Service Region 6 (McDonald and Hansen, unpublished data). The work of the Dorena Center was described in an earlier paper from this panel. Phenotypically resistant trees from the Champion Mine vicinity of the Umpqua National Forest were identified, tested, and incorporated into the rust resistance selection program in the 1960s. In 1970, however, many new cankers were noted on these trees. Cankers were dated, revealing that $< 1.4\%$ of the infections were more than 10 years old, while more than half had occurred in the last three years. In 1972 inoculum on Ribes from the Champion Mine locale was first included in the screening at Dorena, with an alarming increase in pine infection and subsequent mortality. In 1977 an inoculation experiment was designed to establish whether C. ribicola from Champion Mine did indeed differ in virulence from inoculum

from other sources and to provide preliminary data on the genetics of the hypothesized new strain (McDonald and Hansen 1977). Six seedlings from each of 20 families were inoculated in each treatment of a twice replicated design. Inoculum came from Champion Mine, an adjacent drainage, and the Mt. Hood National Forest. Data analysis is not complete, but preliminary results, based only on the percentage of healthy seedlings after 2 years, clearly indicate that a new, virulent strain of C. ribicola exists at Champion Mine. In Table 1, treatments are grouped by inoculum source, either Champion Mine or "wild" type.

Table 1. Healthy seedlings from families of western white pine 2 years after inoculation with Cronartium ribicola from Champion Mine and other locales.

	Inoculum Source	
Families	Champion	"Wild" type
	.....% healthy.....	
All 20 families	3	20
6 Dorena families	6	50
4 North Idaho families	2	8

Analysis of needle and bark reactions is not complete, but will likely make the results still more dramatic.

Pathogenic variation, including vertical pathotypes, or races, is present in the C. ribicola population. This should come as no surprise, considering what we have seen of other host-rust systems. Neither should it cause dismay. A new, virulent race of C. ribicola should pose no particular threat to a well-balanced program for management of white pine unless we give it some selective advantage through a narrowly based breeding program or through poorly conceived deployment of resistant stock. Jim Byler has described to this meeting his observations on sugar pine at Happy Camp, California. Progeny of parent trees with a simply inherited resistance to C. ribicola (Kinloch et al. 1970) were uncankered for a number of years, but now, 10 years after outplanting, are heavily infected. The fungus' potential to change has been amply demonstrated; it will require enlightened, comprehensive strategy to confine the effects to experimental plots.

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Development of Integrated Management Plans

G. I. McDonald

The key to successful management of all infectious diseases lies in understanding the populational interaction of the antagonists in any given environment. Epidemiologic understanding points the way (see Zadoki and Schien, 1979) by forcing a wholistic view of a system. Thus, available knowledge about blister rust epidemiology has been accumulated, hopefully in a manageable fashion, in a computer simulation (McDonald, Hoff, and Wykoff, in preparation; McDonald, 1979). Today, I wish to present three skeleton plans that could utilize the simulator in a decisionmaking role. Please remember, however, that full application of these plans would require verification of the simulator. This step has not yet been completed. In the meantime, the plans provide goals and some food for thought.

The first suggested plan called REHAL (Resistance-Hazard Alignment) is diagrammed in figure 1 and its objective is to match site connected risk and resistance quality and quantity to minimize the need for added controls and to maximize genetic breadth. Factors such as Ribes distribution, species, and resistance patterns, and topographic variables like slope, aspect, and canyon physiography interact under the influence of moisture, wind, and temperature to yield a site-determined level of risk. A version of the simulator could be used to aid in making these site by site forecasts of risk.

The second plan, RUSDAM (figure 2) would monitor temperature and moisture to predict stand-specific rust dammage so that backup controls could be applied as needed. This plan would take various aspects of ribes biology (species, bush size, distribution patterns, and resistance patterns), pine biology (growth rate, size, resistance patterns, species, and stocking diversities) and rust biology (growth rate, aeciospore loads, and genetic variation of epidemological fitness traits) in combination with physical parameters (temperature, moisture, and time) to provide a real time estimate of damage while the infections are still in the needles.

This would provide some time to plan and conduct a control operation. Since high rust years occur sporadically and stand damage is highly related to stand age, and growth rate as well as spore load and pine resistance patterns, the damage forecast would let us be very selective in our application of these expensive backup controls.

The third plan, ESPROG (figure 3) was designed to employ infection and growth history of existing stands to pick an optimum alternative for that established stand. This approach would use parameters such as canker-per-tree, cankers-per-year-per-unit-target, mortality rate, and lethal-canker-accumulation rate to combine with ribes, pine, and rust biologic parameters. The resulting composite data would then be used to develop management plans.

These management schemes were presented at their highest level of sophistication, which includes three different verified forms of the simulator. However, the REHAL option could be used in many situations without simulation assist. Evidence to date indicates that long-distance spread is probably not as important as it was once thought to be and that distribution of upland ribes species is probably the most important risk factor (McDonald 1979).

The white pines are a necessary component of the forest management picture for the west. The most important rust management principal is maintenance of genetic breadth. All levels of resistance from susceptible to highly resistant should be used, and large investments of money and resources are not required for us to learn if a few simple strategies will work. We can begin by planting seedlings obtained from readily available susceptible or low level resistance seed on sites that have proven over the last 50 years to have low blister-rust risk.

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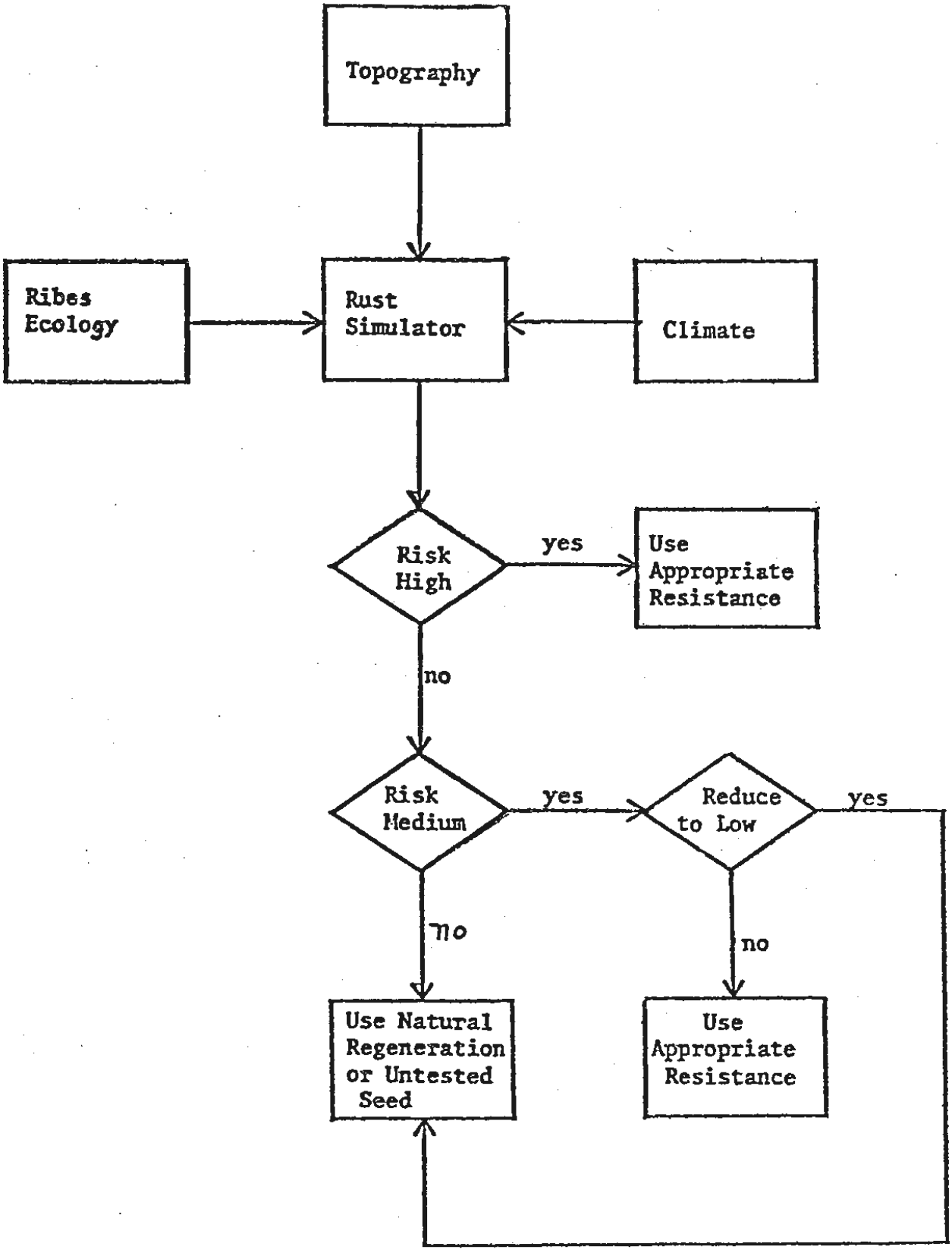

Figure 1.--Flow diagram of blister rust management plan REHAL: resistance -hazard alignment.

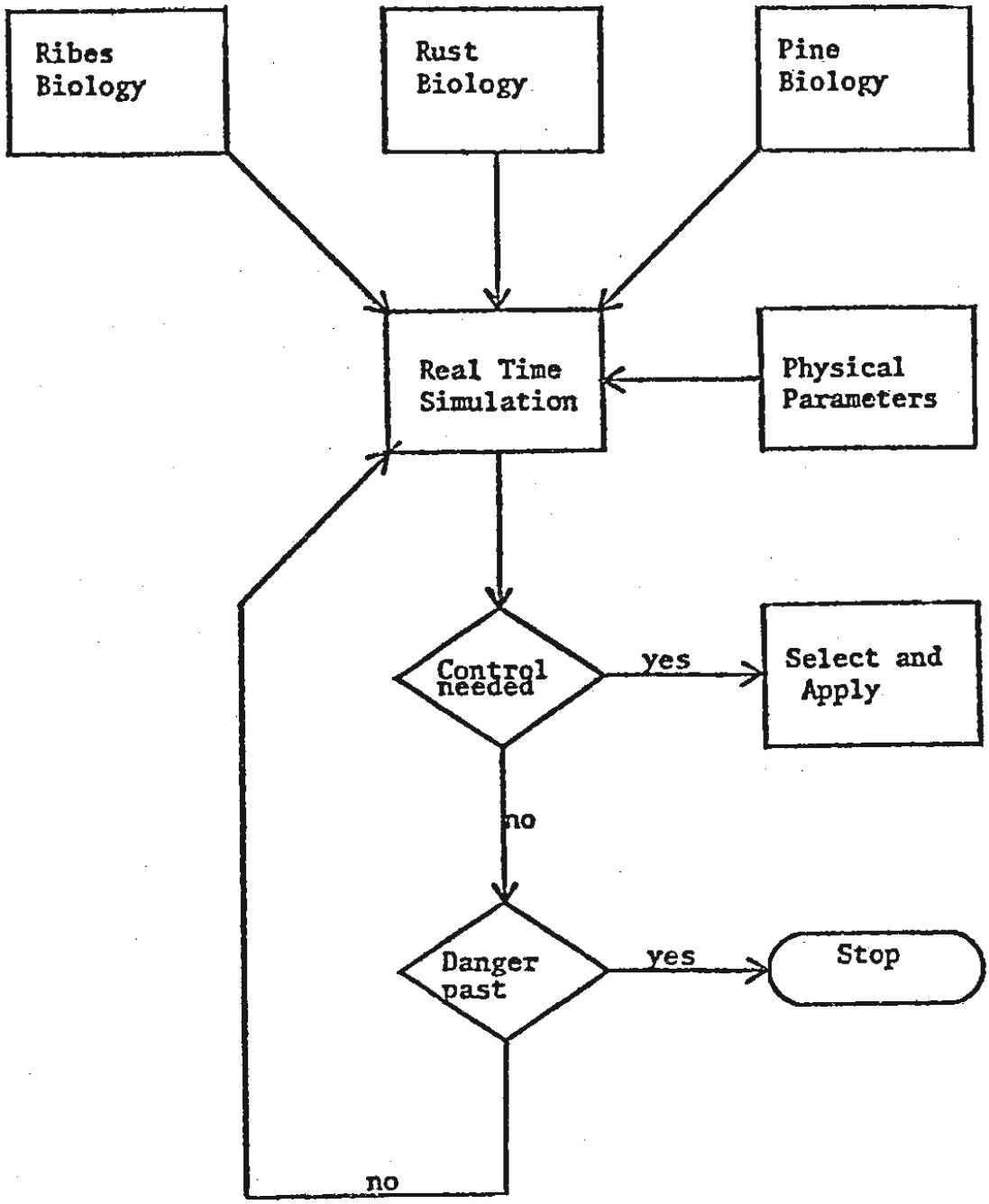

Figure 2.--Flow diagram of blister rust management plan. RUSDAM: rust damage forecast.

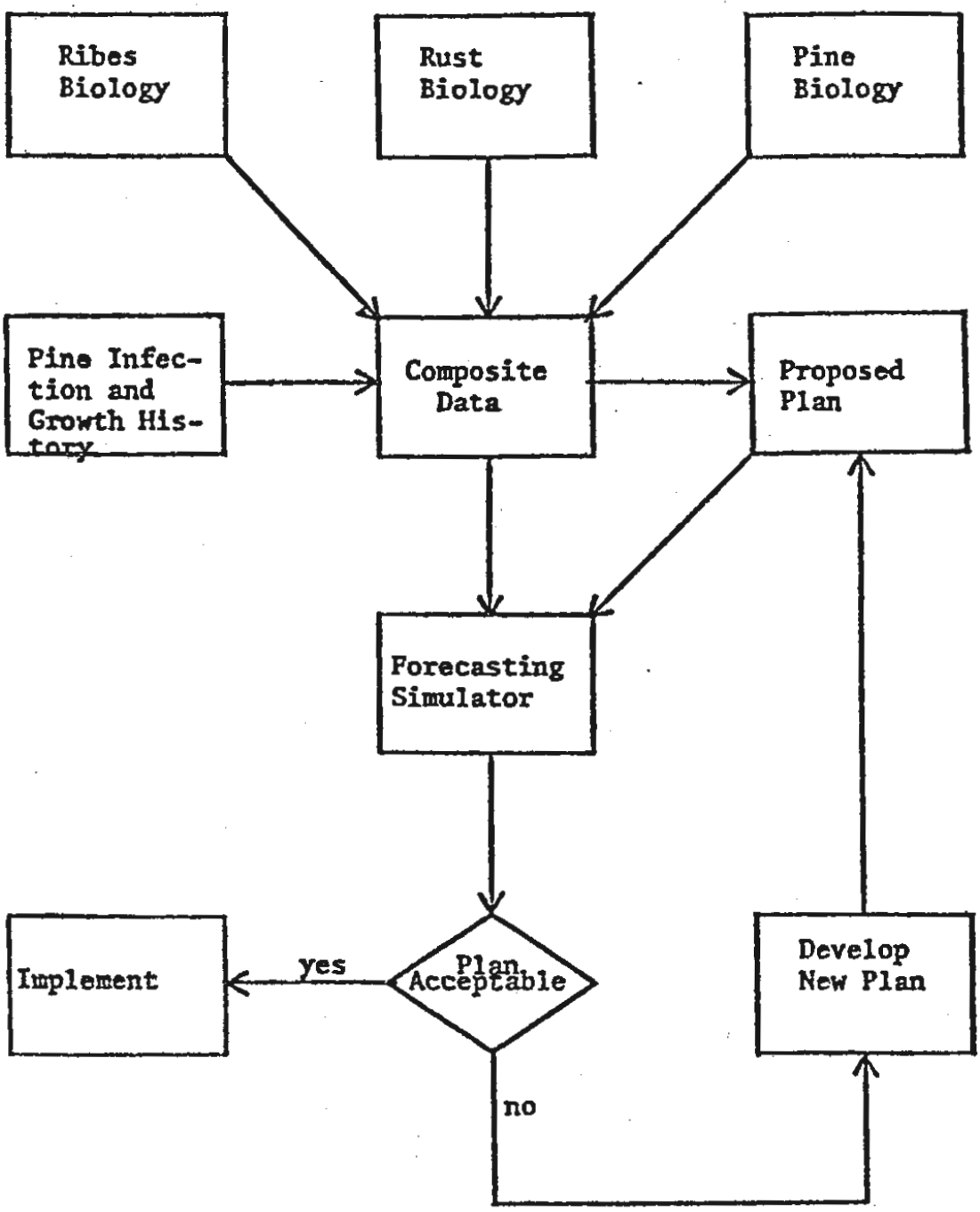

Figure 3.--Flow diagram of blister rust management plan ESPROG: established stand prognosis.

LOPHODERMIUM CONTROL IN PACIFIC NORTHWEST SCOT'S PINE

Filmed by John M. Staley

Narrated by Ken Russell

This film begins with the expulsion of ascospores from a fruit body of Lophodermium seditiosum Minter, Staley & Millar (1), and shows, in addition to the means by which the inoculum is dispersed in nature, how spores are manipulated to obtain single or multispore isolates of Lophodermium species for comparative studies in pure culture.

The differences in symptom expression among L. seditiosum, L. pini-excelsae Ahmad, and L. pinastri (Schrad. ex Hook.) Chev. are next illustrated. Although L. seditiosum causes the most conspicuous spring reddening, in commercial practice plantings infested by it can be spotted long before harvest, and losses associated with shipment avoided. While losses due to L. seditiosum are severe, L. pini-excelsae is perhaps more damaging due to its insidious yellowing of 2-year-old needles that makes it hard to spot and its damage frequently not apparent until after the trees have arrived at their destination. Buyer dissatisfaction and rebate costs combined can be a more serious concern than any damage to the investment in plantations prior to harvest.

L. pinastri (in the strict sense of Staley (2)) yellows and casts only 3-year-old and older needles (of otherwise normal trees). It does little or no damage and is perhaps beneficial under normal forest or hedgerow growing conditions. In Christmas tree plantings, the loss and lodging of 3-year-old and older needles is best described as an "expensive nuisance," adding several cents per tree to the cost of marketing. This cost is offset to some degree by reduced shipping weight after old dead needles have been removed. L. pinastri was present at Mike Webster Nursery and in outplantings from it many years before the more recent and more serious Lophodermium problems were introduced.

Foliar infection in nurseries is shown and the point emphasized that nursery infection is the origin of the problem. The trouble does not come from surrounding native conifers. Thus, nursery sanitation is the logical and necessary practice to minimize Lophodermium damage in outplantings. Lophodermium damage can be recognized and traced from infested nurseries into successively older outplantings, and finally into plantations that are of harvestable size. Intensification of infection in outplantings is most visible after 5-7 years of growth.

Plantations of Scot's pine are frequently infected with all three Lophodermium species. Under these conditions the 3 fungi combine to produce more severe damage and can pose an almost hopeless problem for taxonomic analysis unless each of the 3 species has been studied where it, by chance, exists in isolation from the other species.

Some differences in susceptibility of individual Scot's pine are illustrated. Resistance to L. seditiosum is apparently not related to L. pini-excelsae resistance (or at least resistance associated with current year foliage does not hold up as that foliage becomes older). Certain trees are extremely susceptible to L. seditiosum and these are (where other causal factors appear to be absent) dwarfed and killed by the damage to their foliage. Such trees frequently provide foci of infection that are most apparent in largely healthy plantings and are referred to as "focal trees".

Experimental and commercial control programs are shown to demonstrate that even severely injured trees can be restored to marketable quality after only 2 years of chemical application, and for a cost less than 1% of the value realized. The most cost effective fungicide was Dithane-M45. More expensive but also slightly more effective was a mixture of Bravo and Actidione. (Untested observation suggests the Actidione additive may also boost the effectiveness of Dithane.) Because of the mild winters in the maritime regions of the PNW, approximately monthly fungicidal sprays are required. Commercial experience over 6 years has shown that omission of one or more sprays results in unacceptably costly damage to the foliage.

The cost of the control program illustrated in this movie was surprisingly cheap. Such control can be implemented for 6 years at a cost of 5% of the value protected. However, the long term goal in Lophodermium control is avoidance of both foliar injury and control expense. To this end, nursery sanitation and the planting of disease-free seedlings in disease-free locations is the ideal course of action. We need field tests of the feasibility of this proposed course of action. The success or failure of such field trials will evaluate our present biological understanding on which this course of action is recommended.

Another film, suitable for a TV spot announcement is available Inquiries should be made through Ken Russell.

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SURVEY FOR LAMINATED ROOT ROT ON THE MAPLETON DISTRICT OF THE SIUSLAW NATIONAL FOREST

Don Goheen

Laminated root rot, caused by Phellinus (Poria) weirii (Murr.) Gilbertson, is the most serious disease of Douglas-fir on the western side of the Cascade Mountains in Oregon and Washington. The fungus decays roots causing growth loss, windthrow, and tree mortality. The pathogen persists on a site, surviving for long time periods in infected roots and stumps and colonizing roots of new tree generations as they contact old residues. Subsequent spread from tree to tree occurs via root grafts and contacts. In time, unstocked or poorly stocked centers develop in infected stands. It is estimated that laminated root rot causes annual losses of 32 million cubic feet of wood in Oregon and Washington. Precise information on losses in specific areas is lacking.

In 1977, Forest Insect and Disease Management, Region 6, U.S.D.A. Forest Service was requested by the Mapleton Ranger District, Siuslaw National Forest to provide them with an estimate of loss due to laminated root rot on their lands. The Mapleton District is located along the Oregon coast on the lower drainages of the Smith and Siuslaw Rivers. Forest cover is mainly Douglas-fir with some western hemlock, western redcedar and Sitka spruce. Site quality is quite good, but the terrain is very steep and broken, and brush cover is extremely heavy. Almost the entire area was burned in the 1850's, so the Forest is now composed mainly of 130-year-old stands and plantations aged 30 years or less. We decided to concentrate our survey efforts in the 130-year-old stands since they were at or very close to rotation age. We chose 20 of about 1,000 stand compartments using a random number table and surveyed them using the technique developed by Dr. W. J. Bloomberg of the Canadian Forestry Service.

Basically, the survey method involved establishing two transect grids of five lines each across forest compartments of known area. The starting points of the grids were determined by dividing the length of the compartment by six, thus determining the interval between lines, and multiplying the interval obtained by two random numbers between 0 and 1.

Lines were run on compass bearing, trailing a 200-foot tape. When a disease center was encountered along a transect line, the length of its intercept was measured, and area contained in root rot centers for each surveyed compartment was estimated by developing a ratio between the total length of transect line falling within infection centers and the total length of all transect line in that grid. Since two independent grids with randomly located starting points were run in each stand, a coefficient of variation could be calculated and unbiased confidence intervals obtained.

We arrived at a figure of 4.7% of the area currently affected by the disease on the District. Since we measured the area of infection centers based on the first ring of green trees with no detectable decay or ectotrophic mycelium of P. weirii at the root crown or 3 feet out on two roots, the area we measured was actually something between the total area infected and the area out of production. Our figure agrees surprisingly well with the 5% estimate of Childs'.

BRANCH MORTALITY OF TRUE FIRS IN WEST-CENTRAL OREGON[1/] ASSOCIATED WITH DWARF MISTLETOE AND CANKER FUNGI

Gregory M. Filip, James S. Hadfield, and Craig Schmitt

Dwarf mistletoes (Arceuthobium spp.) are among the most destructive pathogens in Oregon and Washington forests, annually causing 2 million meters^3 of mortality and 2 million meters^3 of growth loss (2). Several canker-causing fungi are associated with dwarf mistletoe infections (4). One of these fungus-mistletoe complexes has been reported to kill limbs, tops, and even entire trees (8, 9). Damage may reach epidemic proportions, especially when trees are weakened by environmental or other factors.

In 1977, excessive branch mortality was observed in several stands of true fir (Abies sp.) on the Willamette National Forest in west-central Oregon. Preliminary observations showed that most damaged stands were infected with dwarf mistletoe. Objectives of our investigation were to: 1) determine the cause of branch mortality, 2) determine relationships between damage intensity and tree size, age, and species, and 3) determine relationships between causal agents and individual branch characteristics.

MATERIALS AND METHODS

In September 1977, fifty-three trees of various size and damage classes were examined on a 60-ha tract on the Willamette National Forest. Because only noble fir (Abies procera) and Pacific silver fir (A. amabilis) had significant branch mortality, only these species were examined.

The following data were recorded for each tree: 1) Species, 2) diameter at 1.4 m above ground level (dbh), 3) age, and 4) damage class. Damage class was determined by dividing the crown into thirds and rating each third on a scale of zero to 2 based on amount of branch mortality. Damage class was the sum of the three ratings. This index was similar to that developed for rating dwarf mistletoe damage (5).

Lower crown branches were examined on randomly selected trees for condition (living, dying, or dead) and presence of dwarf mistletoe infections. An equal number of dead, dying, and living branches were collected to heights of 9 m above ground and returned to the laboratory for culturing.

Branch age and age of dwarf mistletoe infections were recorded in the laboratory after each branch was cut into one 8-cm length that included a swelling, canker, or mistletoe infection. Each branch section was then flamed in alcohol, outer bark was removed with a scalpel, and five wood chips (5 x 5 x 15 mm) were extracted from the inner bark and cambium with a wood gouge. Chips were plated on 2% water agar and incubated for 2 weeks at room temperatures. Fungal growth appearing from chips was hyphal-tipped and transferred to potato-dextrose agar (PDA)(8).

Cylindrocarpon cylindroides Wollen, and Cryptosporium pinicola Linder were identified from conidia produced on PDA in 4 weeks at room temperatures. Suspected isolates of Cytospora abietis Sacc. were transferred from PDA to alder (Alnus rubra) twigs partially embedded in 2% water

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agar. Cytospora abietis sporulated readily on twigs embedded in this medium and was identified easily (8). Fungi isolated infrequently were not identified.

All data were analyzed by stepwise regression or one-way analysis of variance.

RESULTS

Fifty-two of 53 trees examined with varying degrees of branch mortality were infected with hemlock dwarf mistletoe (Arceuthobium tsugense). True fir dwarf mistletoe (A. abietinum) was not detected.

Damage was significantly (P = 0.01) greater in noble fir than in Pacific silver fir, and increased significantly (P = 0.01) with tree diameter (Fig. 1). Tree age was nearly identical for all diameter classes (average age = 82 years). Twenty-seven percent of 626 lower crown branches examined were either dead or dying (Fig. 2). Ninety-seven percent of all dead or dying branches were infected with dwarf mistletoe (Fig. 3).

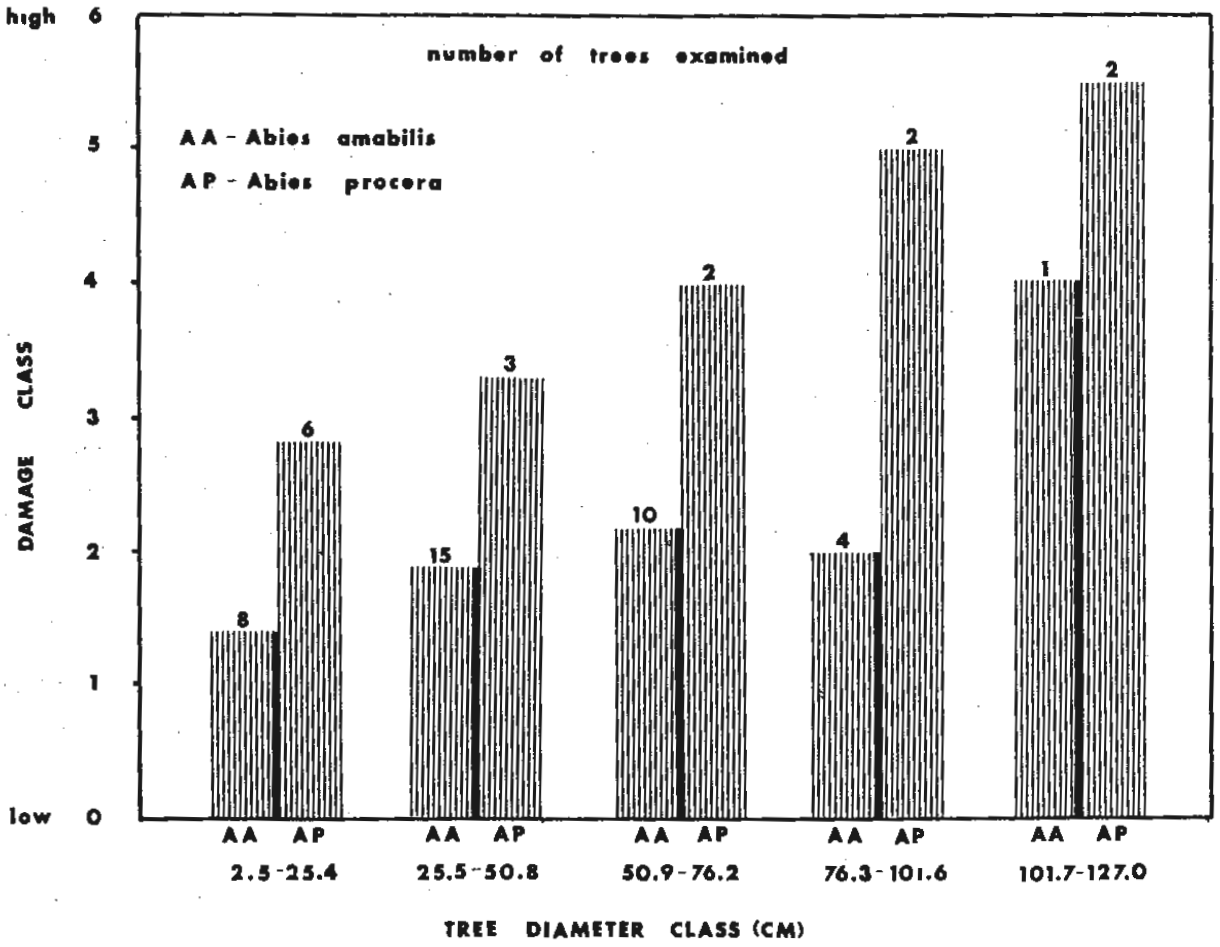

FIGURE 1. Damage class of Pacific silver fir (Abies amabilis) and noble fir (A. procera) by tree diameter class.

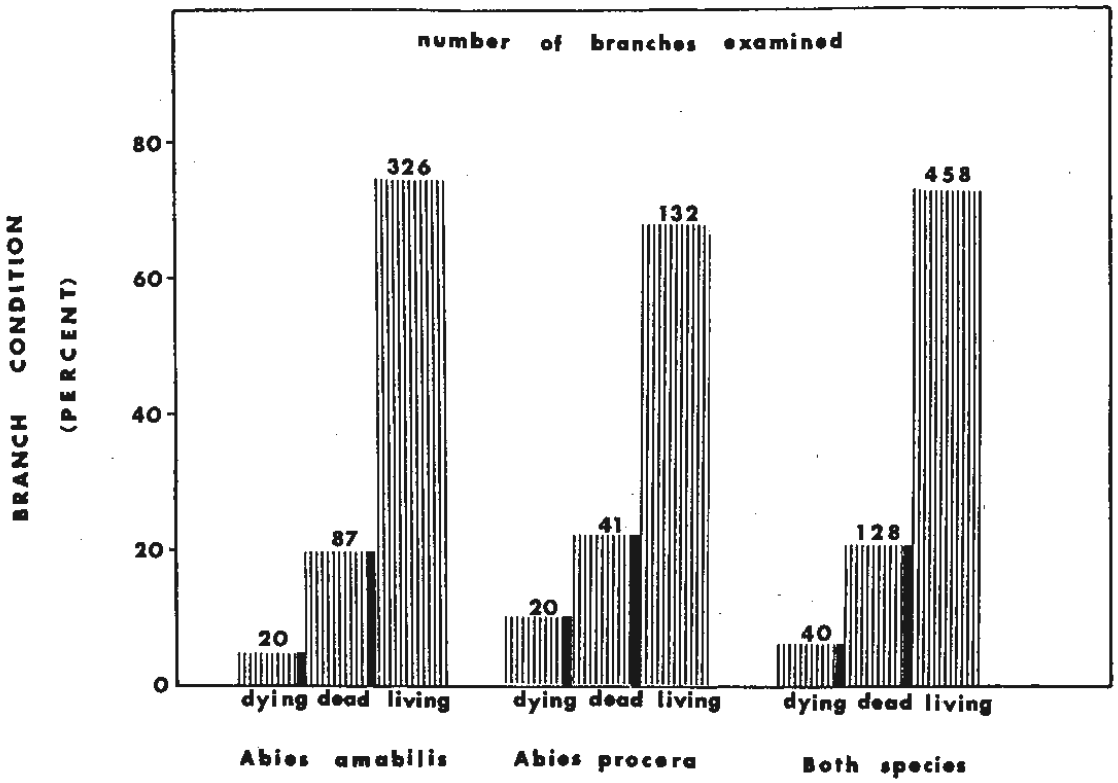

FIGURE 2. Percent of lower crown branches of Pacific silver fir (Abies amabilis) or noble fir (A. procera) that were living, dying, or dead.

Ninety percent of 154 branches examined in the laboratory were infected with dwarf mistletoe (Table 1). Average age of mistletoe infections was 8 years. Fifty-nine percent of all branches had open cankers, but there was no statistical correlation between branch condition and canker presence. It was not determined by Koch's postulates whether cankers were caused by dwarf mistletoe, by fungi, or by both.

C. cylindroides, C. pinicola, or C. abietis were isolated from 45 (29%), 50 (32%), and 11 (7%) of 154 branches, respectively (Table 1). Ninety-two percent of all branches with C. pinicola and all branches with C. cylindroides or C. abietis also had dwarf mistletoe infections. Sixty, 68, and 64% of C. cylindroides, C. pinicola, or C. abietis-infected branches, respectively, had cankers. Fruiting bodies of any fungus were not observed on infected branches. C. abietis was equally common in living, dying, or dead branches as noted by Scharpf (8).

Presence of either C. cylindroides, C. pinicola, or C. abietis in a branch was significantly ($P = 0.05$) correlated with presence of dwarf mistletoe or presence of cankers, but not with branch condition (Fig. 4), branch age, age of mistletoe infection, or tree species. Fifty-six percent of all branches examined were infected with one or more of the three fungi. Twenty-two percent of all fungus-infected branches contained more than one of the three fungi.

DISCUSSION AND CONCLUSIONS

True fir stands in west-central Oregon that exhibit extensive branch mortality are infected heavily with dwarf mistletoe. Similar damage has been observed in other true fir stands in southern Oregon. Severity of branch mortality, however, appears to be greater than normally would be caused by dwarf mistletoe alone. Cytospora abietis is known to infect and kill dwarf mistletoe-infected branches of true firs in California (8). Twenty percent of all branches bearing dwarf mistletoe were infected by C. abietis in California. Although C. abietis has been isolated from white fir (Abies concolor) and Shasta red fir (A. magnifica var. shastensis) in California (8,9), and from grand fir (A. grandis) in Idaho (7), it has never been reported infecting Pacific silver fir or noble fir in the United States.

A much lower incidence of infection by C. abietis (8% of dwarf mistletoe-infected branches) in our investigation prompted re-examination for other possible canker-causing fungi. Cryptosporium pinicola and Cylindrocarpon cylindroides were isolated from 32 and 29%, respectively, of all branches examined. These are the first reports of C. pinicola

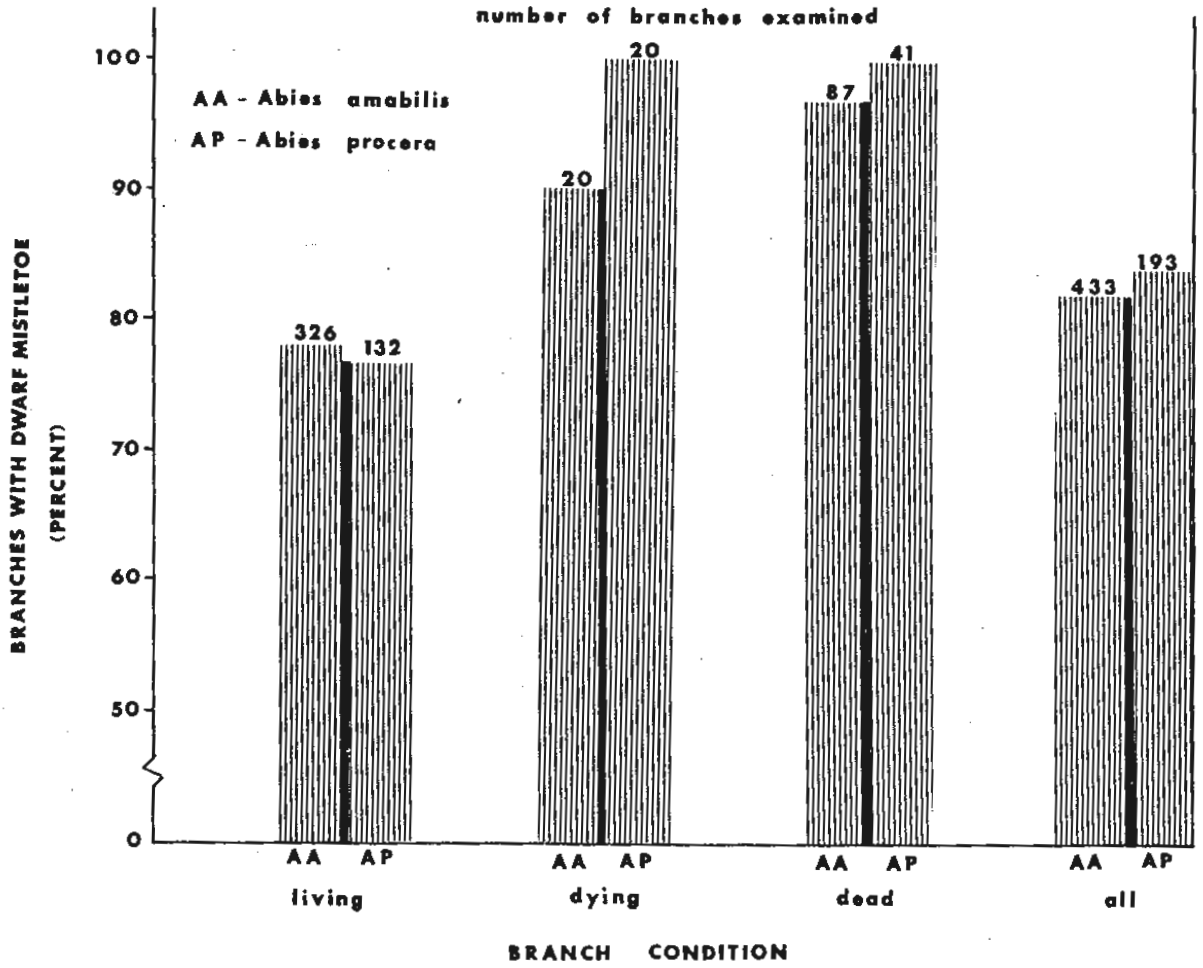

FIGURE 3. Percent of lower crown branches of Pacific silver fir (Abies amabilis) and noble fir (A. procera) infected with dwarf mistletoe.

Table 1. Incidence of dwarf mistletoe, open cankers, and fungi in branches of Pacific silver fir (AA) and noble fir (AP).

Fungal species isolated	No. Branches examined			% Dwarf mistletoe present			% Canker present		
	Total	AA	AP	Total	AA	AP	Total	AA	AP
Cytospora abietis	11	7	4	100	100	100	64	42	100
Cylindrocarpon cylindroides	45	31	14	100	100	100	60	52	79
Crytosporium pinicola	50	41	9	92	90	100	68	76	33
Any of the above	86	62	24	95	94	100	64	65	63
None or Others	68	48	20	84	88	75	53	56	45
Total	154	110	44	90	91	87	59	61	55

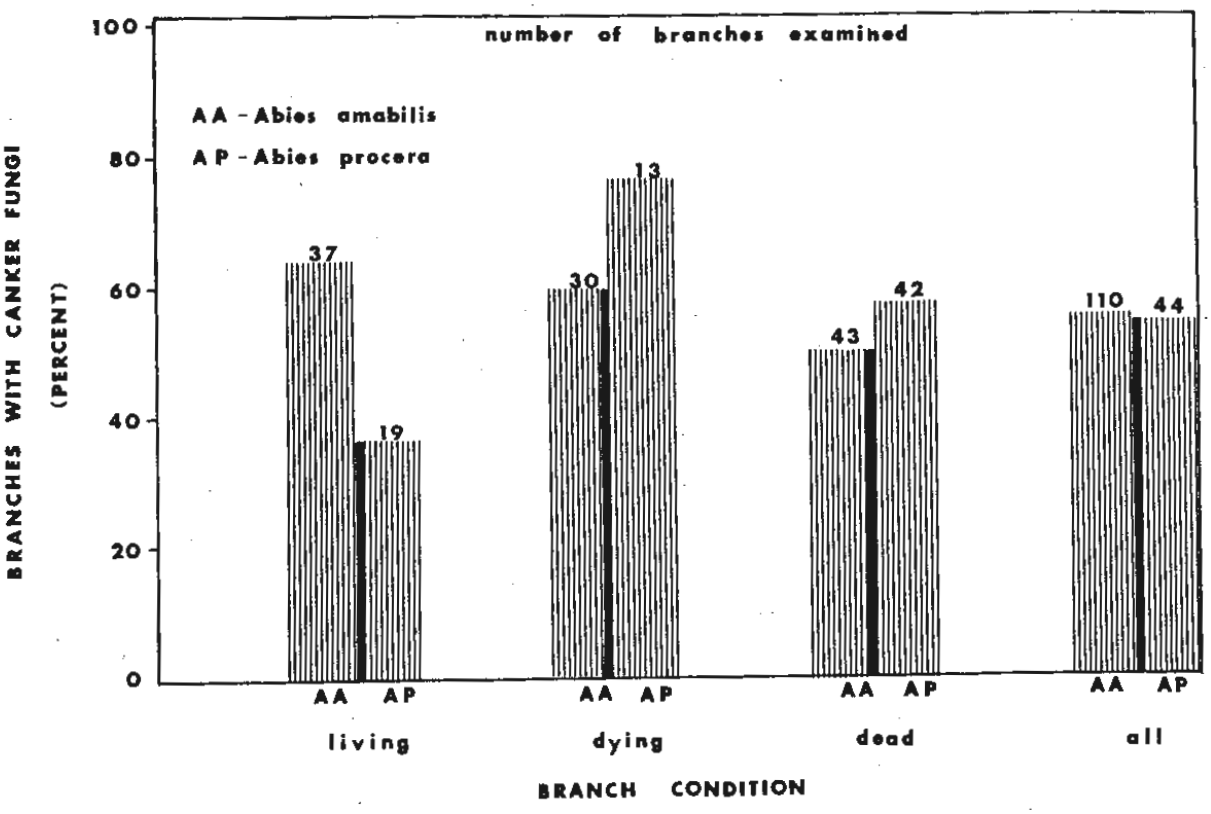

FIGURE 4. Percent of lower crown branches of Pacific silver fir (Abies amabilis) and noble fir (A. procera) infected with canker-associated fungi.

infecting mistletoe-infected branches and C. cylindroides infecting Pacific silver fir or noble fir in the United States.

Cylindrocarpon cylindroides is the imperfect state of Nectria fuckeliana Booth. Pathogenicity of this fungus has been demonstrated by others (1,3) and could, therefore, contribute to the branch mortality in our investigation.

Pathogenicity of Cryptosporium pinicola has not been reported, but its prevalence in our investigation suggests that it may be contributing to branch mortality. Cryptosporium pinicola has been isolated from white pine blister rust cankers in Idaho (6).

In our investigation, branch mortality was greatest in lower crowns where most dwarf mistletoe infections occur and in large trees that serve as larger targets to collect mistletoe seed than do smaller trees. Some of the lower crown branches undoubtedly are dying from natural suppression; however, because of the abundance of dwarf mistletoe and fungal infections in lower crown branches of all trees observed, it is impossible to identify any one factor as the cause of the branch mortality, and, indeed, a complex may in fact be involved.

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INFECTION OF TRUE FIRS BY SEVERAL ISOLATES OF CYTOSPORA ABIETIS

Robert F. Scharpf

Cytospora abietis is a fungus that causes branch cankers and tip dieback of true firs (Abies) in the West (4, 5). In California and Oregon, the fungus is commonly associated with dwarf mistletoe infected fir branches (1, 3). Along with dwarf mistletoe, it is responsible for frequent, and often severe branch flagging. In Idaho, on the other hand, C. abietis infests grand fir (A. grandis) without dwarf mistletoe and causes mainly a dieback of terminal and upper branches (2). To date, the fungus has been isolated from at least 5 different fir species in the West and ranges from California to Northern Idaho (1, 2, 4). However, little is known about host specificity of the various isolates, or of their virulence as pathogens on different hosts in different environments.

The study reported here was undertaken to test the pathogenicity and virulence of 3 isolates of C. abietis obtained from different fir species infected in Oregon and California.

Tests were conducted at the Institute of Forest Genetics, Placerville, California, in the spring of 1979 in a lathhouse using 2-year-old potted seedlings of white fir (A. concolor) and Noble fir (A. procera). The 3 isolates of the fungus tested in the inoculations were collected from 1) A. concolor (C), Stanislaus N.F., California, 2) A. procera (P), and 3) A. amabalis (A), Willamette N.F., Oregon. Pure cultures of each isolate were grown on potato-dextrose agar at 25° C for one week before being used for inoculation. Five trees of each host species were inoculated on March 22, 1979 with each of the 3 isolates by making a small slit in the main stem, placing a ½ X ½ X ¼ cm piece of agar with fungus mycelium in the slit and then wrapping the inoculation site with a 4 X 2 X 1 cm piece of polyurethane foam and grafting rubber.

For controls, five trees of each species were also slit and wrapped with foam pads only, and five were left unwounded and unwrapped. The test trees were then tagged and placed in a lathhouse and watered periodically with a automatic overhead sprinkling system. Two weeks after inoculation, all foam pads were removed from the trees.

Results

The test trees were examined in April, about 1 month after inoculation. Some resin accumulation was present on the stems at the wound site of both of the inoculated and uninoculated wounded trees but no canker development was observed. The trees were examined again in

June, approximately 3 months after inoculation. By this time, well developed cankers were present on some of the inoculated test trees (Table 1). In addition, some of the infested trees died above the site of inoculation. Dieback occurred on 4 A. procera; 3 inoculated with the procera isolate, and 1 with the concolor isolate. One A. concolor inoculated with the procera isolate also experienced dieback.

Nearly all infected trees showed pronounced resin production in association with canker development. Several cankers had girdled more than half the circumference of the stem. Some stems swelled and some became shrunken at the inoculation site. Shrinking was a result of drying of the dead, infected tissue. Swelling was caused by active callusing over of the cankered tissue by the host.

Canker length varied among the test trees (Table 2). In general, cankers were noticably longer on A. concolor than on A. procera.

In June, six of the infected trees, 3 of each species, were taken into the laboratory for fungus isolation in order to confirm the presence of C. abietis in the diseased tissue. C. abietis was isolated from all six trees.

The test trees were examined again on July 30, about 4 months after inoculation. No additional trees had become infected or developed cankers. Also, no more top dieback had occurred even among severely cankered trees. A few of the cankers had elongated 1-2 cm since the June reading, but most were essentially the same length as before. Thus, it appeared that the active canker growth and enlargment ceased in June with the onset of the host growing season. In addition, none of the cankers showed evidence of development of fruiting bodies of C. abietis. No cankers formed on any of the cut and uncut control trees after 4 months.

Conclusions

There was no evidence of host specialization or differences in virulence among the 3 isolates of C. abietis tested on 2 different host species growing under artificial, lathhouse conditions. All isolates appeared to be highly pathogenic on both hosts under the test conditions used.

Little evidence of canker development was present during the first month after inoculation, probably because the fungus was just infecting healthy tissue adjacent to the wound site and because no drying and shrinking of the infected tissue had yet taken place. Drying and shrinking of tissues, plus copious resin production, occurred 2 to 3 months after inoculation, after the fungus had invaded and killed host tissue, and as the host attempted to callus

out the infection. The onset of the warm summer growing conditions probably aided in the drying and shrinking of host tissues and also probably helped to prevent further canker development. Arrested canker development could be the result of changing temperature conditions per se on the fungus, or could be the influence of climatic conditions on growth of the host that allows the host to better resist further fungus invasion.

Table 1. Occurrence of cankers on Abies concolor and A. procera 3 months after inoculation with Cytospora abietis.

	Fungus isolate				
Species	C	P	A	cut	uncut
	----------% trees w/cankers----------				
A. concolor	100	100	40	0	0
A. procera	60	80	80	0	0

Table 2. Length of cankers caused by C. abietis on Abies concolor and A. procera 3 months after inoculation.

Host	Fungus isolate	Canker length	
		mean (cm)	range (cm)
A. concolor	A	3.7	2.1-5.2
	C	3.1	1.8-4.4
	P	4.2	2.0-6.5
A. procera	A	2.1	2.0-2.3
	C	2.0	1.9-2.2
	P	3.3	3.3----

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COMPARISON OF LAMINATED ROOT ROT DAMAGE IN A SECOND-GROWTH DOUGLAS-FIR STAND WITH DAMAGE IN THE PRECEDING OLD-GROWTH STAND

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Introduction

Laminated root rot, caused by the fungus Phellinus weirii (Murr.) Gilbertson, is one of the most damaging diseases of Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco in the Pacific Northwest (Childs and Shea 1967). The long-term survival of the pathogen as a saprophyte in stumps after the harvest of an infected stand, and its subsequent infection of regeneration on the site have led to speculation on the potential for increasing damage under intensive management (Shea 1958; Childs 1963, 1970). To date, all of the studies concerned with P. weirii damage in second-growth stands have dealt with estimation of damage wtihin the time frame of the current stand only. None of these attempted to directly relate the amount and distribution of damage in the current stand to the type and extent of infection in the preceding old-growth stand. The prime objective of this study was to directly compare the damage caused by Phellinus weirii root rot in a 60-year-old second-growth Douglas-fir stand with the reconstructed damage in the preceding stand on the same site.

Methods

The four plots (1 sq. ha. each) surveyed during this study were all located in the Chintimini Woods Creek area of Mary's Peak in the Oregon Coast Range. The current stand overstory was predominantly Douglas-fir about 60 years old (Site Index 165--100 yr base). The previous stand was also predominantly Douglas-fir and was approximately 300 years old at the time of its harvest. The area had burned prior to natural seeding-in of the current stand.

Comparisons of damage for these two stands were made from information gathered by mapping all current stand trees and previous stand residuals on the four plots. Damage in each of the two stands was assessed in terms of the reduction of density and volume by mortality, extent of infection in standing trees, and area covered by the disease. Maps (1:200 scale) of each plot were generated by computer. Damage in the current stand was estimated by noting the amounts and distribution of healthy, infected, and dead trees. Live infected trees were diagnosed by the presence of either surface mycelium or internal decay as evidenced by root collar excavation and increment boring of all trees around disease centers. Previous stand damage estimates were

based on amounts and distribution of P. weirii infected stumps, snags and old down trees. Setal hyphae were used as positive evidence of P. weirii decay in old growth residuals.

Results and Discussion

Comparisons of damage due to P. weirii to the two stands revealed that the disease was more destructive in the previous stand (at time of its harvest) than in the current 60-year-old stand. Estimates of the total loss due to P. weirii mortality from stand establishment to the present were made by comparing the Expected Stand Density and Expected Basal Area with the Actual Stand Density and Actual Basal Area found (Tables 1 and 3). Expected density and B.A. figures were calculated by extrapolating values from healthy areas of the plot to the whole plot. These corrected figures take into account trees which died in earlier stages of stand development and which have since decomposed beyond recognition.

The reduction of Expected Stand Density by P. weirii mortality was greater in the previous stand (75.5%) than in the current stand (41.7%) (Table 1). Of the living trees, 60.9% were infected in the previous stand and 35.1% were infected in the current stand (Table 2).

The reduction of Expected Basal Area by P. weirii mortality was also greater in the previous stand (56.6%) than in the current stand (22.5%) (Table 3). The proportion of the Actual Basal Area infected by P. weirii was also higher in the previous stand (64.8%) than in the current stand (41.1%) (Table 4).

Although both density and volume comparisons were effective in comparing total losses in the two succeeding stands, they do not give any indications of the spatial distribution and correlation of disease from one stand to the next. These can only be assessed by determining the areas affected by the disease in the two stands and how they overlap.

The Area of Concentrated Mortality represents all of the nonstocked area from a P. weirii kill out to the crown edge of the surrounding standing trees. Standing infected trees within 2 meters of P. weirii killed trees were also included in this area. The Area of Standing Infection includes all standing infected trees outside the Area of Concentrated Mortality. The Total Area of Infection is the sum of the first two areas.

On the average, 51.2% of the current and 82.5% of the previous stand areas were infected by P. weirii. This difference in the total areas affected is attributable for the most part, to greater amounts of Area of Concentrated Mortality in the previous stand (68.4%) than in the current stand (36.3%) (Table 5). Present differences in infection were related to past differences in the type of infection and not necesarily to past differences in amount of infection. The type of infection found

in the previous stand ranged from obvious centers with few standing trees to scattered openings with many standing infected trees. The lowest amount of diseased area in the current stand occurred on the plot with the largest open center in the previous stand. The highest amount of disease in the current stand occurred on a plot with a more scattered pattern of infection with many standing infected trees in the previous stand.

The Area of Potential Inoculum represents the combined tree areas of all possible sources of P. weirii inoculum to the succeeding stand and includes all previous stand killed and infected trees. Differences in the Total Area of Infection in the current stand were correlated with differences in the Area of Potential Inoculum from the previous stand and the composition of the inoculum sources (stumps, snags or old down trees). The previous stand Total Area of Infection did not effectively predict current stand damage since up to one half of this area was found to support healthy trees in the current stand. Most of this currently healthy area is found on the previous stand Area of Concentrated Mortality. This represents successful healthy regeneration on areas that were previously out of production due to infection. Spread of disease in the current stand beyond the boundaries of the previous stand infection was found to be minimal and varied. However, spread of disease in the current stand beyond the previous stand Area of Potential Inoculum was very consistant and accounted for 65% of the currently diseased area.

If the current stand was harvested at this time, the Area of Potential Inoculum for the "third" stand would be higher than that from the old growth stand (Table 6). This greater distribution of inoculum could lead to higher levels of damage in the succeeding stand.

Although damage levels were higher in the previous stand than those currently found, the average annual rate of damage increase was much higher for the current stand than the previous stand (Table 7). If damage continues at this rate the level of damage in the current stand at 300 years will be considerably higher than those found in the preceding stand. This prediction can only be a gross estimation without more precise data on damage rate fluctuation.

The effect of distance from inoculum source on probability of infection was studied by measuring distance from randomly selected living trees (both healthy and infected) to nearest P. weirii inoculum source by category. The categories of inoculum sources were: previous stand stumps, snags, and down trees, and current stand trees. The probability of infection decreases with increasing distance as expected for a pathogen spreading by root contacts. For all of the previous stand inoculum sources the probability of infection remained near 0.5 throughout the distance range measured. Current stand killed trees showed a much better relationship between distance and probability of infection. No infected trees were found beyond 9 meters from current stand killed trees.

Conclusions

Damage levels were higher in the previous 300-year-old stand than those found in the current 60-year-old stand. However, average annual rates of damage increase were four times higher in the current stand than in the previous stand. The amount of potential inoculum is also higher in the current stand than in the previous stand. These trends indicate that at a corresponding age damage in the second-growth stand would be greater than in the previous stand. The amount of inoculum awaiting the third stand will be higher than the amount for the second, indicating further intensification of damage. Predictions of damage to a stand are best made by considering the amount and distribution of inoculum sources from the previous stand. Infection in current stand trees is related to distance from current stand mortality.

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Table 1. Reduction of Stand Density by *Phellinus weirii* caused mortality.

	Expected Stand Density (trees/ha.)	Actual Stand Density (trees/ha.)	*P. weirii* Mortality (trees/ha.)
Current Stand	1031	601	430 (41.7)[a]
Previous Stand	375	92	283 (75.5)[a]

[a]Percent of Expected Stand Density.

Table 2. Proportion of Actual Stand infected by *Phellinus weirii*.

	Actual Stand (trees/ha.)[b]	Infected by *P. weirii* (trees/ha.)
Current Stand	601	211 (35.1)[a]
Previous Stand	92	56 (60.9)[a]

[a]Percent of Actual Stand Density

[b]Living trees at time of measurement (current stand) or time of harvest (previous stand).

Table 3. Reduction of Basal Area (B.A., m^2/ha) by Phellinus weirii caused mortality.

	Expected B.A.	Actual B.A.	P. weirii mortality
Current Stand	65.3	50.6	14.7 (22.5)[a]
Previous Stand	144.5	62.7	81.8 (56.6)[a]

[a]Percent of Expected B.A.

Table 4. Proportion of Actual Basal Area (B.A., m^2/ha) infected by Phellinus weirii.

	Actual B.A.	Infected B.A.
Current Stand	50.6	19.1 (37.7)[a]
Previous Stand	62.7	40.6 (64.8)[a]

[a]Percent of Actual B.A.

Table 5. Comparison of stand area covered by Phellinus weirii infection in the current and previous stands.

	Area of Concentrated Mortality	Area of Standing Infection	Total Area of Infection
	Percent of total area		
Current Stand	36.3	15.2	51.5
Previous Stand	68.4	14.1	82.5

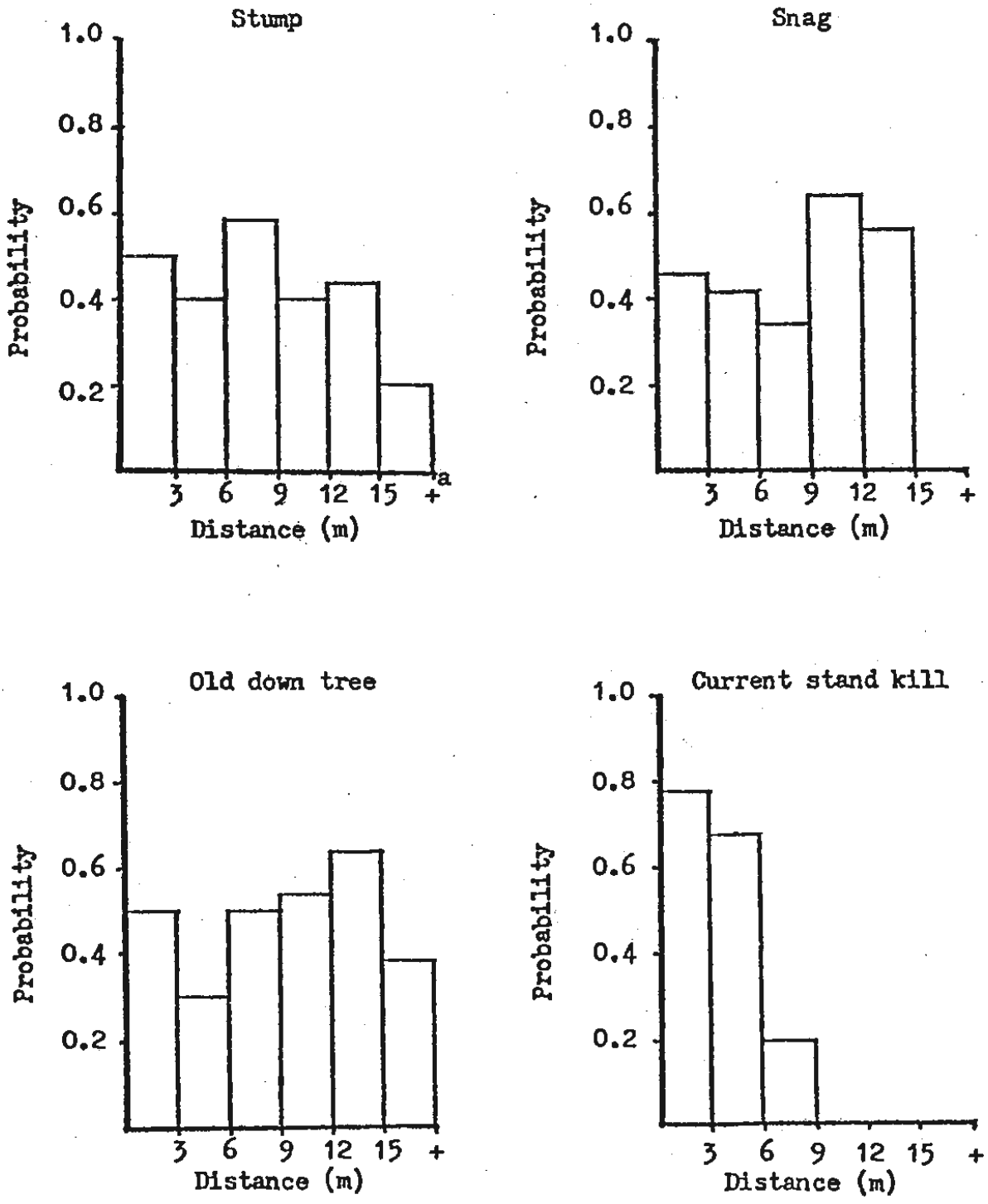
Table 6. Area of Potential Inoculum for current and previous stands.

	Area of Potential Inoculum (m^2/ha)	Percent of total area
Current Stand	2904.6	29.0
Previous Stand	2661.3	26.6

Table 7. Average annual rates of damage increase for the current and previous stands.

	Increase in Total Area of Infection	
	Cumulative	Annual
	Percent of total area	
Current Stand	51.5	0.86
Previous Stand	82.5	0.28

Probability of P. weirii infection with distance (m) from old infected stumps, snags, and down trees, and current stand killed trees.



[a]distance greater than 15 meters

Cryptosphaeria Canker of Aspen

Thomas E. Hinds

Cryptosphaeria populina (Pers.) Sacc., an ascomycete in the family Diatrypaceae of the Sphaeriales, has been consistently associated with a canker of western aspen (Populus tremuloides Michx.) over the past several years (Krebill 1972, Hinds 1976). A 1977 survey of 30 aspen sites in nine National Forests in Colorado revealed the cankers present on 83 percent of the sites and 4.3 percent of the 3,302 trees examined. Twenty-six percent of the tree mortality encountered was attributed to the canker (Juzwik et al. 1978). The canker has been collected as far south as the Santa Catalina mountains of Arizona, northward in the Rocky Mountains to British Columbia, the Yukon Territory, and north of Fairbanks, Alaska (Hinds and Laurent 1978). Other western poplar hosts include P. angustifolia James, P. balsamifera L., P. deltoides Marsh, and P. trichocarpa Torr. and Gray (Hinds and Laurent 1978).

The cankers, frequently associated with trunk wounds, are usually long and narrow. They may be only 5-10 cm wide, yet up to 3 m or more in length, following the grain of the underlying wood. Annual lateral extension of the canker margin may be only several milimeters per year but several centimeters or more in the vertical direction. Small trees die several years after infection and before the trunk is girdled. Branch cankers are not uncommon on large trees where they often girdle the branch and enlarge onto the trunk. Cytospora chrysosperma (Pers.) Fr. is frequently found along the canker perimeter and is quick to colonize the remaining bark after tree death.

The infected bark around the perimeter of a canker is discolored light brown to orange. Annual callus formation by the host in an attempt to limit the bark infection is obvious after two or more years. The dead bark adheres tightly to the sapwood. Bark that has been dead for more than one year is black, stringy, and sooty-like, similar to the sooty-bark canker caused by Cenangium singulare (Rehm.) Davidson and Cash. However, the dead bark contains small, lenticular, light colored areas, varying from 0.5-2.0 mm in size. Perithecia of the fungus are formed separately within an effused pseudostroma which may be 2-3 cm in diameter, up to 5 x 30 cm in size beneath the bark periderm which is slightly raised above the surface in the central portion of the bark that has been dead for at least one year. Light orange acervuli bearing filiform, nonseptate, curved conidia of the imperfect stage Libertella sp. are occasionally found in the advanced portion of the canker.

The fungus colonizes the heartwood and sapwood causing discoloration and decay before it penetrates the bark causing canker. This sapwood discoloration usually extends up to a meter or more beyond the vertical extent of the canker. Various hues of gray, brown, yellow, orange, and even pink are associated with the white-brown mottled trunk rot in larger trees from which Libertella sp. can be readily isolated. Under ultra-violet light, the incipient and advanced decay exhibit various patterns of yellow fluorescence which is more intense in the discoloration around the perimeter of infection.

Over 400 inoculations using various isolates of the fungus were made on aspen sprouts, trees of various sizes, and live branches between 1974 and 1977 to satisfy Koch's postulates that C. populina was the causative agent of the disease. The fungus caused branch, sprout and sapling mortality within one year. Trunk inoculations on larger trees resulted in cankers which extended up to 7.1 x 33.5 cm in 50 months. Sapwood discoloration and fluorescence behind these cankers extended a distance of up to 401 cm.

The ability of the fungus to cause decay was tested by the common soil block method. Average weight loss after 24 weeks incubation was: bark, 13.5 percent; sapwood, 27.0 percent; and heartwood, 19.1 percent. The infected wood showed fluorescence under ultraviolet light.

It was first thought that the yellow fluorescence associated with the Libertella saprot behind the canker might be diagnostic for identification purposes. However, upon inspecting other common aspen decays under ultraviolet light, they also showed the fluorescence, particularly in the discolored zones around the advanced decay. This fluorescent material is produced by the fungi. Concentrated water extracts of the various fungi grown in culture exhibit similar fluorescence. It is speculated that this fluorescent material contains polyphenol oxidases which oxidizes certain acids and phenols to more toxic quinones which are then polymerized to insoluble non-toxic melanins causing the discoloration around the perimeter of infection (Hare 1966).

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DWARF MISTLETOE CONTROL IN EASTERN WASHINGTON

TEN YEARS AFTER TREATMENT

Kenelm Russell

INTRODUCTION

The Department of Natural Resources (DNR) manages about 1.5 million acres in various categories in eastern Washington. Approximately 550,000 acres are designated commercial forest lands, mostly conifers of all ages, densities and species mix.

Portions of these forest lands are infected with dwarf mistletoes (DM); Arceuthobium campylopodum, A. douglasii, A. americanum and A. laricis being the principle species. In Washington and Oregon dwarf mistletoes are responsible for a loss of about 900,000,000 board feet annually. Many sapling stands exhibit poor growth because of above normal stocking and dwarf mistletoe infection. Certain of these stands with moderate to light infection offer good potential for simultaneous precommercial thinning and dwarf mistletoe control.

In 1968 the Forest Service offered financial assistance to the DNR for DM control in qualifying stands, at first sharing 25% then later 50% of the cost. Control was centered in three general areas of DNR ownership, Okanogan, Spokane and Glenwood. From 1969 to 1974, 7714 acres were treated at costs ranging from $22 to $32 per acre, averaging about $25 per acre.

CONTROL PROCEDURES

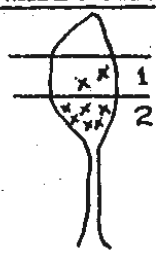
Early dwarf mistletoe control attempts were aimed at eradication of the parasite which proved to be quite impractical. The best management solution was to decide, "How much dwarf mistletoe could be tolerated in a stand without losing growth?" A simplified contract specified using the 6 class Hawksworth DM rating system (DMR) to decide cut and leave trees but with a slight variation. Crop trees larger than 6.5 inches d.b.h. allowed no more than a DMR-3 with the smaller crop trees allowed progressively lower DMR's. Growth impact by DMR is explained later. The DMR system with diameter limitations is explained below:

STEP 1: Divide live crown into thirds
STEP 2: Rate each third separately. Assign each third either 0, 1 or 2 as described below:
- (0) No visible infection
- (1) Light - Less than 1/2 of branches with DM plants
- (2) Heavy - More than 1/2 of branches with DM plants and/or brooms

STEP 3: Add ratings of thirds for total tree DMR.

CUT ALL TREES EXCEEDING THE RATINGS BELOW:

FOR THIS DBH CLASS (inches)	THIS IS MAXIMUM ALLOWABLE DMR
0 - 2.4	0
2.5 - 4.4	1
4.5 - 6.4	2
6.5 +	3



EXAMPLE: The drawing shows a DMR of 3.

Each tree cutter carried the above information on a waterproof wallet card. They were encouraged to use the rating system as a quick mental evaluation process which would take only seconds or fractions of seconds to decide cut or leave status. When no DM was present, standard tree spacing rules applied. When DM was encountered DMR rules prevailed. This type of cutting produced small clearcuts where infection was heavier and somewhat irregular spacing where infection was moderate. The appearance of the treated stand was very pleasing - a definite plus, both aesthetically and silviculturally. We preferred not to operate in heavily infected stagnated stands because growth response was much better on moderate to lightly infected stands. More DM free or nearly free trees could be left as crop trees in these stands.

After gaining experience, we found that certain contract clauses could be eliminated to reduce costs. We dropped the artificial tree free barrier requirement between adjoining properties with only a few exceptions. DM spread is slow enough laterally that subsequent management would minimize reinfection. We stressed the continued use of natural barriers for control unit boundaries such as roads, streams, meadows, and other small openings whenever possible.

INFECTION AND GROWTH IMPACT

Growth impact from dwarf mistletoe does not become significant on larger trees until they carry a DMR of 3+ (Table 1). A DMR-3 is the logical maximum rating to leave in the stand. It takes approximately 8 years for a five to six inch d.b.h. tree to go from a DMR-3 to 4 (Table 2).

TABLE 1. Dwarf mistletoe ratings and growth loss for single trees 1/ (ponderosa pine).

DM RATING	% GROWTH REDUCTION
0-3	0-9
4	10-34
5	35-64
6	65-100

1/ Hawksworth, F. G. 1974. Personal communication.

TABLE 2. Change in dwarf mistletoe ratings over time 2/

DMR FROM:	DMR TO:	YEARS TO NEXT DMR:
1	2	8
2	3	8
3	4	8
4	5	16
5	6	16

The next thinning (commercial) should occur approximately 10 to 15 years following initial treatment depending on site productivity. Assuming a DMR-3 tree (growth loss less than 9%) would change to DMR-4 during the interval, growth loss would increase to between 10 and 34 percent which is probably acceptable but only for the interval between treatments. Leaving trees with DMR-4+ at treatment time will result in individual tree growth losses within a few years of 35 - 100 percent which is not acceptable. For smaller trees, maximum allowable ratings must be lower because they will remain in the stand much longer.

Hawksworth 3/ has shown radial growth in heavily infected trees to be 71% less than healthy trees. Similarly infected trees were 36% shorter and 18% smaller in diameter than healthy trees. In general, trees with DMR-5 or 6 are losing approximately one to two-thirds or more of their potential growth. This growth data is for southwestern pine dwarf mistletoe A. vaginatum which is not found in the Pacific Northwest. Similar data is not available for A. campylopodum, the native species. Trends from A. vaginatum data are used and may be slightly greater than those for A. campylopodum.

WHAT DID WE MEASURE?

During summer 1974, dwarf mistletoe control units treated five years earlier were surveyed. We measured 832 trees on 34-1/10 acre permanent plots scattered throughout the units. We established 14 plots around Glenwood, 11 near Spokane and 9 west of Omak. We remeasured the plots in summer 1979 (ten years following treatment). Variables measured included: species, diameter, height, crown class, vigor, cut or leave status, DMR trunk canker presence and overstory-understory classification.

2/ Hawksworth, F. G. and C. A. Myers. 1973. Procedures for using yield simulations programs for dwarf mistletoe infected lodgepole and ponderosa pine stands. USFS Rocky Mountain Forest & Range Exp. Sta. Res. Note RM 237, 4 pp.

3/ Hawksworth, F. G. 1961. Dwarf mistletoe of ponderosa pine in the Southwest. USDA Technical Bulletin. 1246. 112 pp.

WHAT DID WE FIND?

Overall, visual inspection both five and ten years after treatment showed normally appearing thinned stands with trees free of DM brooms. At ten years, visual appearance of treated stands was definitely a plus when they were compared to nearby untreated stands. (We did not establish plots in untreated stands.) Dwarf mistletoe had not blossomed into growth robbing proportions except on a few plots where infection had been too severe prior to treatment. Proper follow-up commercial thinnings using DMR cutting rules in most instances will keep DM in check. Most of the stands will be re-treated 15 to 20 years later and DM would continue to be of minimum influence to growth. Waiting longer than twenty years between treatments would not be advisable.

Growth response could be measured on crop trees by an approximate doubling of annual ring widths except at the Glenwood area plots where it was slightly less. After treatment, the Omak and Deer Park plots averaged 203 and 212 stems per acre, respectively and the Glenwood plots averaged 288 stems. The higher stocking at Glenwood probably explains the reduced growth response there. Volume growth on most plots was very acceptable and will be reported in a later publication. Data is being computer analyzed and will be available in cubic foot yield tables.

We found DM infection on crop trees to be well within limits defined by the original cutting rules after ten years. Little overall change in DMR occurred between five and ten years although internal changes within the rating classes did occur which will be explained in detail later.

Table 3 shows percent of trees by DMR for 1974 and 1979. There was little overall change in most ratings. DMR-5 trees doubled, but involved only a few trees so is considered insignificant.

Five years after treatment, 69 percent of all crop trees were completely free of DM (had DMR-0) and after ten years 63 percent had DMR-0. The bulk of the change in the second 5 years occurred when DMR-0 trees became DMR-1 trees. The six percent drop in DM free trees after ten years is also insignificant and illustrates the relatively high number of infected trees (37 percent) which can be tolerated in a managed stand without suffering undue growth loss over a ten-year period. Of course, it is assumed that a very high proportion of these infected trees carry a DMR below allowable levels.

TABLE 3. Percent of trees by DMR 5 and 10 years following control indicates little change (Basis: 832 trees, 1974; 761 trees, 1979).

Years Since Treatment	Dwarf Mistletoe Ratings						
	0	1	2	3	4	5	6
	Percent of Total Trees						
5 (1974)	69	10.3	7.8	7.9	3.2	1.3	0.6
10 (1979)	63	14.8	8.7	6.8	3.7	2.5	0.5
Change	-6	+4.5	+0.9	-1.2	+0.5	+1.2	0

There was a general upward movement of DMR within the allowable categories over the ten-year interval, particularly in the 0-6.4 inch d.b.h. trees and the lower DM ratings. These changes may or may not be real (Table 4). The stairstep line in Table 4 separates allowable crop trees from undesirable ones.

Those to the left are allowed; those to the right are not. We found that 91.3 percent of all trees measured in 1974 and 90.4 percent measured in 1979 fell within allowable ratings. The fact that these two calculations are so close ten years after treatment suggests that the internal changes in DM ratings are not yet of great importance. Given enough time between treatments, the ratings would change much more.

TABLE 4. Percent of trees found in each diameter class by DMR rating five and ten years after treatment. Trees to left of stairstep line are desired crop trees; those to right of line were also left following treatment, but are neither desired nor allowed under the rating rules.

	Dwarf Mistletoe Ratings													
	0		1		2		3		4		5		6	
d.b.h. Class	Years Since Treatment													
	5	10	5	10	5	10	5	10	5	10	5	10	5	10
	Percent of Diameter Class													
6.5+	60	62	13.8	15.0	10.1	9.2	9.9	7.7	3.7	4.1	1.7	1.7	0.4	0.5
4.5-6.4	79	58	4.8	15.7	5.9	9.9	5.9	7.4	3.7	5.0	0.5	3.3	0.5	0.9
2.5-4.4	77	65	5.5	13.5	5.5	7.7	6.8	0	2.7	0	2.7	11.5	0	2.0
0.0-2.4	88	84	6.8	10.7	1.1	1.8	2.3	3.5	0	0	0	0	2.3	0

The infected trees with ratings too high for crop trees (8.7%-1974 and 9.6%-1979, to the right of the stairstep line) were probably missed during treatment instead of becoming infected since treatment. The fact that there was little change in overall infection percentages from five to ten-years after treatment supports this conclusion. A certain percentage of missed trees is inevitable because of hidden infections from the one-year-old DM seed at time of treatment.

The slow but steady upward trend of reinfection means the job of full DM control is not complete. Careful records preferably kept in some sort of permanent retrieval system will help keep track of treated stands. Each time the stand is harvested, the DMR of infected trees will dictate cutting patterns.

DWARF MISTLETOE CONTROL PROBLEMS

Eastern Washington silviculture traditionally encourages uneven-age management. This is contrary to management of dwarf mistletoe infected stands. Spread of dwarf mistletoe is most rapid from overstories to rising understories. The prerequisite for control in a low level canopy is to remove all infected overstory. For a time the stand should be managed on an "even-age", or better, a single story basis. Typical east-side stands have a wider age spread than those managed on a clearcut basis. Age spread may be considerable even after the overstory is removed but the canopy level should be as even and as low as possible.

The biggest problem we found in the treated units was the infected overstory, much of it with infection ratings over the maximum allowed. Most eastern Washington stands have some degree of overstory, a good deal of it infected. Administrators had great difficulty finding adequate acreage suitable for DM control in the precommercial thinning size classes without infected overstory. We prefer trees this size for control because the DM plants are easier to see since they are much closer to the ground.

In the long run it would have been better to cut infected but merchantable trees and either attempt to sell them or leave them on the ground. I do not recommend that these trees be left for a merchantable harvest as their growth is too slow and they only serve to reinfect younger faster growing trees within the stand. As markets improve such trees could probably be sold. Every effort should be made to market them.

The best approach to effective control is to have an overstory removal timber sale first, then schedule precommercial thinning and DM control together. The DM reduction is thus integrated carefully with complete silvicultural treatment of the stand. An important consideration in selling a control program is that the complete removal of infected merchantable overstory volume is usually a one time affair only and the stands are being improved for several generations of trees to come.

Some plots had too many trees per acre which restricted diameter growth response. Removing more stems would place better growth response on fewer stems making a useable product in a shorter time.

Selecting qualifying acreages for cooperative financing of treatment was another problem. The Forest Service did not wish to pay for thinning on acreage that was DM free. It was costly to map small infected areas of a stand. It might be better to determine percentages of infected trees in the stand and base financing on that figure. We are seeking to build greater flexibility into dwarf mistletoe control qualifications to make the Local Managers' unit selection job easier.

RECREATIONAL USE CONSIDERATIONS

Occasionally, dwarf mistletoe control units encompass recreational use acres such as trails, scenic viewpoints, scenic roads or camping areas.

Special untreated corridors or "maintenance control" may be necessary for these areas, providing infection is not severe, when it is desirable to retain the original stand character. Overstory that is infected could be retained when no understory is present. Pruning such infected trees is feasible only under these conditions. The same marking and rating rules apply. Barriers either free of dwarf mistletoe infected trees or containing other immune species may be necessary along edges of these special corridors.

Planning special treatment for sensitive areas could be worked out with interested local citizens. The basic philosophy for these special use areas is to retain the original character of the stand, including the overstory.

SUMMARY

Dwarf mistletoe control works!!!!

CONTROL OF THE EASTERN DWARF MISTLETOE ON BLACK SPRUCE

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The eastern dwarf mistletoe Arceuthobium pusillum Peck is the most serious disease of black spruce, Picea mariana (Mill.) BSP, causing deformity and rapid mortality. Anderson estimated that dwarf mistletoe was present in 12-18% of the black spruce stands in Minnesota and suggested cutting infected trees or burning logging slash as control measures (1). Cutting infected stems was later shown to be ineffective and costly (2). Subsequent research has concentrated on developing the use of prescribed burning (4). However, few sites have been burned to control mistletoe. To illustrate some of the reasons for this, one of our most recent burns is described as follows.

Color infrared 35 mm 1:30,000 scale vertical aerial photography of the dwarf mistletoe infested stand was taken from a small aircraft in August, 1975. This system allows the land manager himself to do the photography, and obtain high quality, low cost imagery which can be used to detect dwarf mistletoe infection centers (3). Areas of infection were determined on the photographs, and cutting boundaries were extended 1-2 chains (20-40M) beyond infection to delimit the area to be harvested. During January, 1976, the 4.8 hectare area was strip cut and harvested using a shortwood system; that is, trees were topped, limbed, and bucked into 2.54m lengths where they fell, and then moved to a landing. This system is necessary to provide adequate slash for prescribed burning. During the harvest, all trees taller than 2.4m were felled.

Based on slash transects, 58% of the area was estimated to be covered with slash. The longest gap in the slash cover was 17.3 m, but the average gap length was 4.9m. Based on 17 0.02 ha plots there were 193 apparently healthy and 30 infected stems per hectare (Fig. 1).

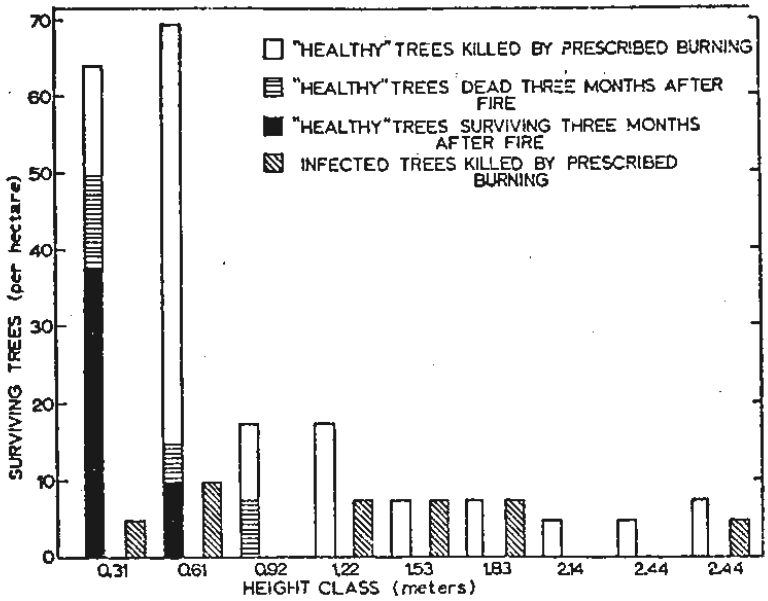

Figure 1. Survival of healthy and infested black spruce by size immediately following the fire and 3 months later.

In August 1976, an attempt was made to burn the site, but was aborted due to excessively hot, windy conditions. Weather was not suitable for burning until July 26, 1977, when the site was burned. A center firing ignition pattern was used to minimize control effort. This pattern has the added advantage of bending the flames over the vegetation, which increases the likelihood that the trees will be killed by the intense heat if they are not consumed by the fire. This is particularly important in pockets of infection, where slash is lacking and residual trees are more common. The difference between the number of trees surviving immediately after the fire and three months later demonstrates the importance of heat (Fig. 1). Three months after the fire, forty-seven trees, all less than 0.6 m in height per hectare, survived. Infected trees of this size are not frequently encountered, because they have such a small surface area to intercept dwarf mistletoe seeds (5). These trees pose little threat of reinfesting the new stand, and thus we believe dwarf mistletoe has been eradicated from this stand. This site was regenerated to black spruce by applying seed from a helicopter using 280 g/ha.

The cost of prescribed burning in this stand was $190.61 per hectare. This does not include the cost of the aborted attempt to burn the stand in 1976. Difficult access required the use of special vehicles for travel and also the presence of a large crew for fire control. These factors were responsible for much of the cost of the operation.

Although prescribed burning is an effective method for eradicating trees remaining after harvesting, and in some cases can improve the seedbed, it has little future as a control measure for A. pusillum. First, forest managers considered the cost too high. Even if the cost were acceptable, access and safe burning weather limit the use of burning. Prescribed burning is only effective in small centers of infection, as there are not enough trees in large centers to provide slash for a fire of sufficient intensity to kill residual trees. Of far more importance is the evolution of the harvesting method from a shortwood to a full-tree system, where the tree is filled with a mechanical harvester and the full tree skidded to the landing, where it is limbed and topped. The use of this system is increasing. Presently, 50-70% of the black spruce cut is harvested in this manner. This system does not provide adequate slash for prescribed burning, since most of the debris is deposited at the landing. In some cases, however, residual trees are destroyed during the harvest operation. On one site, of 1557 trees present on plots established prior to harvesting, 78 remained after harvesting (Table 1). All but one of these trees were less than 0.6m tall, and that one tree had been wounded but had not yet died. For reasons presented above, we think these small trees are not likely to be infected, and thus it is reasonable to assume that dwarf mistletoe has been eradicated from the site.

The full-tree skidding system is a very low cost alternative for the land manager, as the harvest operation itself provides the control.

It is extremely important, however, to remove residuals taller than 1.5 m during the harvest operation, as they have a high probability of being infected, and allowing dwarf mistletoe to reinfest the new stand. These trees can be removed by killing or by destroying them with a skidder or other equipment during the harvest operation. In years when winter temperatures are warm or deep snows accumulate early, vehicle movement in spruce stands is difficult because the ground is not frozen. Operators establish skid trails and stay on them to avoid becoming mired which reduces the vehicle traffic over much of the stand. Thus, fewer residual trees are destroyed. Vehicle traffic is also reduced in large centers of infection due to a lack of merchantible trees. Because of these factors, full tree skidding can only be expected to eradicate residuals when infection centers are small and the ground is frozen.

Table I. NUMBER OF LIVING SPRUCE BEFORE AND AFTER HARVESTING

PLOT NO.	BEFORE	AFTER
1	434	0
5	194	0
6	96	0
13	266	37
14	302	10
15	265	31

In warm winters, or in large pockets of infection, residuals will survive harvesting. We believe that these residuals pose a serious threat to the new generation of trees. One possible means for removing them would be the application of an herbicide. To examine this possibility, four compounds were evaluated in 1976 and 1977 for their ability to kill black spruce. The compounds, the rates tested, and the results of these tests are shown in Table 2. Roundup and Trysben 200 were ineffective at any rate tested. Tordon 101 at 60 l/ha and Sodium TCA at 30.2 Kg/ha provided effective control. To determine if residues of these compounds could affect regeneration of the site, soil samples were collected one year after application of the herbicide, and seeded with black spruce. Residues of Tordon 101 reduced germination, and caused chlorosis, necrosis and twisting of needles. Seedlings did not outgrow these effects within three months. Seedlings grown at the same time in soil treated with Sodium TCA did not appear different from those grown in untreated soil. On this basis, Sodium TCA was selected for operational testing.

TABLE 2. PERCENTAGES OF BLACK SPRUCE KILLED BY HERBICIDES APPLIED IN 1976

HERBICIDE	RATE (L/Ha)	PERCENT KILLED
ROUNDUP	2.6	0
	7.8	7
	13.0	9
TORDON	20.0	89
	40.0	78
	60.0	97
TRYSBEN 200	10.4	32
	20.9	60
	31.3	80
SODIUM TCA	KG/HA	
	16.8	67
	28.0	92
	39.2	98

In July, 1979, three sites totalling 40.5 hectares were treated with 62.3 liters of Sodium TCA (Hopkins Chemical Co.) in 62.3 liters of water per hectare applied from a helicopter. Although the results of this test will not be available until July, 1980, it appears that trees less than 2m tall are readily killed by this treatment. Many larger trees appear to have survived. This substantiates the need to remove residuals taller than 1.5m. Sodium TCA also kills competing brush (species of Ledum, Chamaedaphne, Salix and Alnus) and grass, which are major causes of regeneration failure. No effects on Sphagnum seedbed were noted.

The cost of this treatment was $240 per hectare. The chemical cost $23.09 per hectare, and the remainder was the cost of application, which was very high because of the small area treated. Based on the application cost of release sprayings, the total cost of this operation would decrease to an estimated $48 per hectare if large areas were treated. This cost is acceptable to forest managers.

Residue studies still in progress have shown no increase of Sodium TCA or closely related metabolites in unsprayed areas. In fact, low levels of these compounds were detected in the treated areas prior to spraying. Nevertheless, this compound is an herbicide, and will be subject to environmental scrutiny.

Summary

If Sodium TCA proves capable of killing residual black spruce, the recommended program for dwarf mistletoe control will involve detection of the parasite through the use of 35 mm aerial photography, harvesting

the stand 20-40m beyond obvious infection, and destroying residuals taller than 1.5m. This may be done either by killing or using logging equipment to run over these trees, but it must be done. Following the harvest operation, the stand must be surveyed to determine the number and size of residuals. Where many residuals are present, especially those of large size, these trees must be eradicated either by prescribed burning, where adequate slash is present and this alternative is acceptable, or by the aerial application of Sodium TCA. The site can then be regenerated by natural or aerial seeding.

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A NOTE ON BACTERIAL NOMENCLATURE

Oscar J. Dooling

Insect pathologists have long been intrigued with the possibility of using microorganisms, especially bacteria, to control pestiferous insects. One bacterial pathogen that has been widely accepted for controlling a wide group of insects is *Bacillus thuringiensis* var. *thuringiensis* Berliner. Entomologists like short names for all insecticides, biological or chemical, and refer to this bacterium as BT.

Heimpel and Angus (1958) designated *B. thuringiensis* var. *thuringiensis* as the type variety of the species. Steinhaus (1961) has pointed out that *B. sotto* Ishiwata apparently has priority over *B. thuringiensis* since *B. sotto* was used in the literature as early as 1908. The rules of nomenclature are quite clear on this point, and entomologists can no longer refer to this bacterium as BT, but must use the legitamate name of *B. sotto*, or BS.

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WESTERN GALL RUST AND ITS HYPEPARASITES IN WESTERN CANADA

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In recent years forest industries in the prairie provinces of Canada (Alberta, Saskatchewan, and Manitoba) have been expanding rapidly, and forest management is becoming increasingly intensive. As wood harvesting increases, reforestation activities have to be stepped up mainly to meet provincially imposed reforestation standards. Production of healthy nursery stock and survival and health of planted stock are of great concern for managing foresters. Disease problems of tree nurseries and young managed forests are significantly different from those of mature and overmature natural stands. Certain diseases that are important in older trees often do not affect young trees, but some others are much more important in young managed forests than in mature natural forests. Western gall rust is an example of the diseases that I believe will become increasingly important in highly managed situations.

This disease is caused by a rust fungus, *Endocronartium harknessii*, or if you do not like that name or the mycologist who named it, you can use other names such as *Cronartium harknessii* or *Peridermiun harknessii*. The disease is distributed more or less throughout the natural range of lodgepole pine and jack pine across North America, from the Pacific to Atlantic (Hiratsuka and Powell 1976). Because of this distribution, some people argue that the name "western gall rust" is not appropriate for this disease, and new names such as "pine-pine gall rust", "globose gall rust" and a few others have been proposed. I believe that once a common name of a disease is coined it should not be changed just because of new distribution data or other information; therefore, I prefer to keep calling this disease "western gall rust".

In recent years many severe infestations of this disease have been reported in plantations, tree farms, and nurseries. For example, in an Alberta tree farm raising ornamental trees, more than 60% of young lodgepole pine trees over 20 acres had galls, with an average of more than 20 galls per tree; all infected trees had to be destroyed (Powell and Hiratsuka 1973). Another example is a plantation in Manitoba that was planted with jack pine 7 years ago and considered as one of the most successful plantations; close examination revealed that about 15% of the surviving trees had western gall rust at the base of the stem. Furthermore, several seedlings showing some signs of yellowing were found to have galls below the soil line, proving that the infection came from

the nursery. A survey of the nursery where the seedlings came from revealed that more than 3% of the seedlings had visible western gall rust symptoms.

I would like to point out four main reasons why I believe this disease will become increasingly important in young managed hard pine forests in our region.

1. The disease exists abundantly across this continent, following lodgepole pine and jack pine distribution, and inoculum is available throughout the region.

2. Susceptible hosts, lodgepole pine and jack pine, are the two major reforestation species in the prairie provinces.

3. This rust does not need alternate hosts to complete its life cycle and can multiply and disseminate within the stands without going to alternate hosts.

4. Like many other rusts, this rust also prefers to attack vigorously growing host plants rather than weakened ones; therefore, when man encourages growth of trees, this disease tends to intensify. Thinning, spacing, and fertilizing may encourage rather than discourage the disease.

5. Once a gall is formed, it can be active for many years and can become a source of infection to other trees in the stand. The fact that this disease does not kill the tree quickly is also noteworthy. Although trees with main stem galls never become merchantable trees, they occupy the site for a long time, and often are counted as survivals in plantation surveys, thus resulting in false information.

To find out the proper management strategies for this disease it is important to understand the conditions and requirements for infection. It is known that heavy years of infection in a given area occur once every 5 to 6 years; these are called "wave years". (Peterson 1971, Powell and Hiratsuka 1973). It is speculated that suitable climatic conditions during the critical period of spore dissemination are the main factors governing the occurrence of the "wave years". Our observations suggest, however, that some biotic factors such as fungi, bacteria, and insects also might play important roles in reducing the production and dissemination of viable spores. With the added interest of possible biological control of the disease, we have been conducting investigations of fungi found on galls and especially of those that affect viability, production, and dissemination of spores.

I would like to report briefly on three such fungi that we have found on western gall rust. This work is being done mainly by Dr. Aki Tsuneda,

who is working with me as a recipient of a National Research Council of Canada Visiting Fellowship.

The first fungus I want to discuss is *Cladosporium gallicola*. This fungus has been observed often on western gall rust and was described by Dr. B.C. Sutton (Sutton 1973). We have observed that this is an agressive mycoparasite of western gall rust (Tsuneda and Hiratsuka 1979). Within two days after spraying conidial suspension on a rust gall, the whole sporulating surface becomes covered with this fungus. With scanning electron microscopy we can observe many signs of penetration of rust spores by *C. gallicola*. On water agar, *C. gallicola* positively seeks out the rust spores and parasitizes them. Although this is a destructive hyperparasite of the rust, it usually attacks only several spore layers on the surface of the gall and does not affect the rest of the spore layers. This may limit its potential as a biological control agent.

The next fungus is *Scytalidium uredinicola*. This fungus was described in 1976 (Kuhlman et al. 1976) as a mycoparasite of fusiform blister rust (*Cronartium quercuum fusiforme*) on slash and loblolly pine in the southeastern U.S. Recently, we found this fungus on western gall rust and also on stalactiform blister rust (*Cronartium coleosporioides*) (Hiratsuka et al. 1979, Tsuneda et al. 1980). We think that this fungus is playing a significant role in reducing the inoculum density of the rust in nature. Numerous arthroconidia are distributed among spores of the rust. Spores of *E. harknessii* contacted by *S. uredinicola* were deeply indented at the area of contact and collapsed without any sign of penetration by the parasite. This phenomenon suggests that *S. uredinicola* aquires nutrients from the host spores by impairing their spore cell permeability control. Previously, when galls were not producing spores for the season, we called the phenomenon "gall inactivation". We have been observing that these inactivated galls often become active again in 1 or 2 years. I speculate that mycoparasites such as *S. uredinicola* are partly responsible for such a phenomenon. When unopened galls were cut open, we often found young sori completely parasitized by *S. uredinicola*. This fungus penetrates to the bottom of the rust sori. Tests showed that spores from infected galls were killed, and no germination was observed. *S. uredinicola* seems to be very common in certain places, and up to 80% of the galls in a given area were known to be infected with it. This fungus may have a good potential as a biological control agent of western gall rust.

The third fungus is *Monocillium nordinii*. This fungus was described long ago in Alberta from wood of lodgepole pine (Bourchier 1961). We found this fungus as a mycoparasite on western gall rust and stalactiform blister rust (Tsuneda and Hiratsuka 1980). This fungus is inconspicuous on galls but was isolated several times from gall surface areas not producing viable spores, which seems to suggest that the fungus is responsible for surpressing the rust. This is an imperfect fungus that also shows strong mycoparasitic characteristics. Hyphae penetrate into the wart

layer of the rust spores and eventually disintegrate the warts. This fungus produces at least two strong antibiotics, including one undescribed antibiotic substance. Isolation and identification of the antibiotics have been conducted by us with Dr. W. Ayer of the University of Alberta and will be reported soon.

In conclusion, I would like to stress the importance of western gall rust in young managed hard pine forests in the prairie provinces of Canada, and I would like to suggest that biotic factors may be playing important roles in the fluctuation of inoculum potential of the rust.

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INSIGHTS INTO THE FUTURE OF FOREST PATHOLOGY RESEARCH

by

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The program chairman has asked me to spend a few minutes and relate to you some insights into the future of forest pathology as viewed from the position of a research administrator. Since I am rather new to the game of research administration, I suspect my insights will more accurately reflect the views of a seasoned research scientist that retains somes scars and sensitivity from skirmishes over steadily declining research budgets and inflation of the dollar.

In order to develop some insights as to what the future holds, we need to take an assessment of trends and events of the recent past that come to bear on the subject. These events and trends of the past, and indeed the future, are all linked at the local, regional, and national levels. While there has been considerable talk and concern expressed over the decline of forest pathology in western North America, little of this concern has reached the publication stage. Consequently, there is very little documentation in the literature to reference. Therefore, my main source of reference is my personal experiences and knowledge of forest pathology as an employee of the Forest Service. In addition, I have located a few recent articles that address basic research in agriculture in the United States which contain some information relevant to our subject.

It is common to hear present-day research administrators express concern for innovation, creativity, accountability, and cost effectiveness as they relate to productivity. This is a sign of our times. It reflects the pressures and demands to "stay in step" and be responsive to the needs of a rapidly changing society. The entire Nation is concerned about productivity and the importance of research and development (R&D) in maintaining it. Yet, we do not have a clearly defined national policy on science and technology.

Let us look at some trends of the recent past that have impacted the present situation. First, we need to review some traditional sources of funding for basic science research in the United States. In doing this, I will rely heavily upon the information provided by Philip Handler in his article, "Basic Research in the United States."

In post-World War II years, the major incentives for support of research in basic science were: 1) to assure our national security, 2) to develop peaceful uses for nuclear energy, 3) to provide a con-

tinuing energy supply, 4) to advance our standards of public health, 5) the quest to explore outer space, and 6) to expand and increase agricultural productivity. A large portion of this research was funded by the Department of Defense (DOD), the Atomic Energy Commission (AEC), and the newly created National Institutes of Health (NIH). Other Federal agencies supporting research functioned at a much lower scale. The National Science Foundation (NSF) was established in 1950 to provide a balanced national program of research and education for basic science. Since then, the National Oceanic and Atmospheric Administration (NOAA), Environmental Protection Agency (EPA), and Department of Energy (DOE) were authorized by Congress. Today, NSF has evolved as a major source of Federal funding for research in basic science. In the past 25 years, its appropriations have grown almost 40-fold to a proposed budget for FY 1980 of $1 billion. It is second only to NIH in funding of research at universities.

The AEC is no longer in existence and the role of DOD in financing research in basic science has been declining. The role of the Bureau of Standards, Geological Survey, and USDA have remained much the same for the past 35 years. National expenditures for R&D have been in a downward trend, until just recently, while such expenditures in other developed countries, i.e., Japan, West Germany, and Great Britain have been steadily rising. The President's budget for national expenditures for R&D for FY 1980 is $32.5 billion.

In terms of constant dollars (1972) Forest Service expenditures for R&D have increased 31 percent since 1969. The distribution of these increases have been somewhat disproportionate because of ear-marking and oversight designation of specific funds. Consequently, some functions have benefited while others have actually experienced reductions in funds.

In response to an action item in the Combined Program Review for the Intermountain Forest and Range Experiment Station (1979), Dr. A. L. Schipper, FIDR-WO, was assigned the task of analyzing the trends reflected in the FIDR budgets for 1969 through 1979. During this 11year period, the Forest Service Research (FSR) appropriations increased 2.7-fold from ca. $40 million to ca. $108 million in actual dollars. In constant dollars (1972), this represents a 40 percent increase with the difference being lost to inflation. FIDR has received relative constant increases during the period in terms of actual dollars. Such increases, however, have served largely to keep pace with inflation as evidenced by a constant purchasing power during the period as measured in 1972 dollars. During this same period, forest industry expenditures for R&D were among the lowest for major industries in the United States. This is somewhat surprising because recent reports show returns on investments in agricultural research at 35-40 percent which is well above the 10-15 percent realized on typical investments (Evenson, et al., 1979). Other interesting statistics about FIDR during this 11-year period that were generated by the analysis are:

1. The number of SY's remained relatively constant. Further analyses of this statistic shows that Forest Disease Research (FDR) had a decline of 13-15 percent (70 in 1969 to 60 in 1979) in number of pathologists while the number of entomologists in Forest Insect Research (FIR) rose from 98 to 116 for the same period. This attrition of pathologists has occurred through retirement, resignation, and transfer into research administration and the vacated positions were not filled.

2. Actual dollars/SY have increased at a steady rate for FIDR. This ratio has remained relatively constant in terms of 1972 dollars. The difference between funding for FDR and FIR have increased from $1.8 million (actual dollars) less for FDR in 1969 to $3.2 million less in 1978. However, because of the increase in FIR scientists, the dollars/SY for FIR is less than those for FDR.

3. The national average age of Forest Service scientists is 44.5 years; FIDR scientists average 46.25 years.

4. Examination of average level of education of scientists in the various functions of FSR shows that FIDR has a greater proportion of Ph.D.'s than other functions.

5. The average grade of FIDR scientists is 13.4 compared to the FSR average of 13.0. The average grade of TMR scientists is slightly above the FSR average also. All other functions were at or below the FSR average. The average salary for FIDR scientists is $30,500 compared to the FSR average of $28,750.

6. FIDR has the lowest average operating funds/SY at $45,500. The FSR average is $69,000. This difference is greater than the total annual operating budget for many FIDR work units in the West.

Handler (1979) states that perhaps 80 percent of all science has been learned since the birth of the National Science Foundation in 1950. It is certain that research progress in basic science will be more difficult and more expensive in the future than it has been in the past. In facing this future, FDR is confronted with a decline in numbers of scientists and extremely deficient operating budgets. Resolution of this dilemma will not be easy in view of nationally imposed personnel ceilings and appropriation levels.

The challenge to ensure the future of FDR is perhaps best stated as the need to obtain greater support from our user groups, particularly forest industry, for our products. I have heard several speakers and colleagues attending this conference refer to the need for pathologists to get together and talk to one another. I agreed that this is beneficial to pathologists with respect to exchange of technical knowledge. I submit, however, that if we are to have a future we must make a greater and more effective effort of selling our research and technology developments to our clientele, the forest land managers and the forest products industry.

Without their support, FDR will be hard pressed to justify its existence in a society so accustomed to a continually strong economic development and productivity, because this is how society defines progress. Without acceptable progress, erosion of the science will continue until FDR can no longer serve the needs of society and we pathologists will go the way of the dinosaurs. We need to tell our story and advertise our wares to the public and its elected officials, because this country runs on politics, not on biological facts.

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WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE
DWARF MISTLETOE COMMITTEE REPORT
HIGHLIGHTS OF 1979 DWARF MISTLETOE RESEARCH

John G. Laut, Chairman

I. Taxonomy, Hosts and Distribution

a. Hemlock dwarf mistletoe was found at Chilkoot Lake, north of Haines, Alaska, at a latitude of approximately 59°, 20'. This is the northernmost known locality for this mistletoe and is approximately the same as the northern known limit of A. americanum in Alberta. (T. Laurent, USFS, Juneau, and F. G. Hawksworth, USFS, Fort Collins).

b. A new species of Arceuthobium was found in the pinyon Pinus discolor in San Luis Potosi, Mexico. This dwarf mistletoe is unique in many ways; it has long (up to 20 cm) pendulous shoots on systemic brooms, systemic brooms are formed only by staminate plants, and it is exceedingly rare. With this discovery, the total number of dwarf mistletoes now known in Mexico is 20. (F. G. Hawksworth, USFS, Fort Collins and D. Wiens, Univ. of Utah).

c. Flavonoid compounds from 36 of the 38 known taxa of the genus Arceuthobium (dwarf mistletoes) were examined. The flavonoid chemistry of the genus is rather uniform, all taxa producing 3-O-glycosides of the flavonols quercetin and myricetin. No infraspecific chemical variation was encountered, and in those instances where subspecific taxa are recognized, their chemistry was uniform. At the subgeneric level, members of subgenus Arceuthobium synthesize primarily glucosides, whereas galactosides are more common in subgenus Vaginata. In two of the four Old World species of subgenus Arceuthobium (A. juniperiprocerae and A. oxycedri) only myricetin 3-O-glucoside was detected. There was no absolute flavonoid differences between subgenera, sections, or series. On the other hand, flavonoids are useful in several instances at the species level. In several cases, chemical data lend support to the recognition of species which in the past have been considered doubtfully distinct on the basis of morphology. (Published in Brittonia 31:212-216, 1979). (D. J. Crawford, Ohio State Univ., and F. G. Hawksworth, USFS, Fort Collins).

d. We have found perennial aerial shoots of Arceuthobium pusillum which have flabillate branching. This supports the contention that this species belongs in the subgenus Vaginata. A note on this finding has been accepted for publication in the Canadian Journal of Forest Research. Also reported in this note are observations of bird damage to aerial shoots and the incidence of secondary infections (F. Baker and D. W. French, Univ. of Minnesota).

e. I located Arceuthobium cyanocarpum 15 miles south of Sisters, Oregon at Little Three Creeks Lake. Extensive infection occurs on white bark pine and mountain hemlock. Stands include some subalpine fir and lodgepole pine. Area is about 300 acres ± and extends from the west shore of the lake of the last trees against McArthur Rim. A manuscript has been submitted to Plant Disease (Knutson, PNW).

f. Arceuthobium abietinum f. sp. concoloris was found parasitizing white fir (Abies concolor) in Marshall Gulch, Santa Catalina Mountains, Arizona. This is only the second report of this dwarf mistletoe in southern Arizona (R. L. Mathiasen, Univ. of Arizona).

g. Arceuthobium microcarpum has been found parasitizing corkbark fir (Abies lasiocarpa var. arizonica) in the White Mountains, Arizona. This rare host-parasite combination has been reported previously from the Pinaleno Mountains and the San Francisco Peaks, Arizona (R. L. Mathiasen, Univ. of Arizona).

h. Preliminary results of studies of A. douglasii in mixed conifer stands in the Apache-Sitgreaves National Forest, Arizona indicate that corkbark fir (Abies lasiocarpa var. arizonica) is not a secondary host of this dwarf mistletoe. Approximately 5 -- 25% of the corkbark firs in A. douglasii infested stands -- are infected. Most infected corkbark firs have had low dwarf mistletoe ratings and few trees are killed by the mistletoe (R. L. Mathiesen, Univ. of Arizona).

i. In Chelan State Park, Chelan County, Washington, we found dwarf mistletoe, Arceuthobium campylopodum, growing well and producing fruit on Norway spruce. This is the first report of this dwarf mistletoe growing on Norway spruce naturally. Spruce trees are in close proximity to the pine and are approximately 20 to 25 feet tall. They have several plants and apparently the Arceuthobium thrives well on the spruce (K. W. Russell, Washington Department of Natural Resources, Olympia).

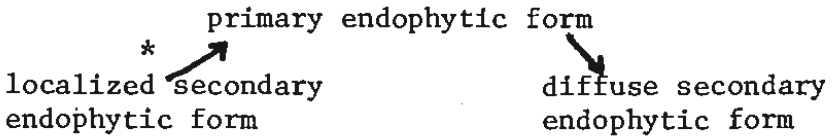
j. Some preliminary results have been obtained from field tests begun in winter of 1977-1978 to test the host-parasite relationships of the Arceuthobium campylopodum-occidentale complex. Both Digger pine (Pinus sabiniana) and Knobcone pine (P. attenuata) growing in the field have been successfully infected with several collections of both A. campylopodum and A. occidentale. Ponderosa pine (P. ponderosa) in the field, inoculated with the same collection of seeds, has shown no infection yet (R. F. Scharpf, PSW; W. Mark, Cal. Poly; F. G. Hawksworth, RM).

II. Physiology and Anatomy

a. The discovery of phloem elements in a dwarf mistletoe from Mexico (Calvin, Portland State Univ.).

b. I finished an essay on "How parasitic plants induce disease" published in Plant Disease, Vol. 4. Edited by Horsfall and Cowling (Knutson, PSW).

c. Morphological studies of A. douglasii on Douglas-fir: host/parasite tissue relationships were studied in infected primary shoots, young woody stems, and older branch tissues. The variability of A. douglasii encophytic form was found to reflect a sensitivity of the parasite to the same growth factor(s) which determine and coordinate primary or secondary growth in the host tree. The "primary endophytic form" consists of hyphal-like strands within the host cortex, primary phloem, buds and procambium. No sinkers are formed. The primary form converts to the "diffuse secondary endophytic form" when host tissues of the infected shoot convert to secondary growth. Multiseriate endophytic stands and sinkers then develop simultaneously throughout the young woody branch. In contrast, the "localized secondary endophytic form" develops from an infection of existing host secondary tissue, and typical, fusiform, localized infections result. The relationship between the three endophytic forms is thus:



* Portions of secondary endophyte can convert to primary, if buds are reached (Alosi, Berkeley).

d. Cytological features (A. douglasii, A. tsugense, A. occidentale, A. californicum): Although thoroughly integrated with host tissues, parasite cells can be identified by their distinctive chromocentric nuclei in contrast to the conifer reticulate-type of nuclei. Also Arceuthobium cells have abundant lipid stores. Starch is very rare in sinker cells associated with host phloem, but becomes more and more abundant towards and into the aerial shoots. Arceuthobium plastids and mitochondria are morphologically distinct from those in host cells at the electron microscope level. Sinker cells have abundant endoplasmic reticulum and wall/membrane specializations which may serve to enhance solute flux across the parasite's plasmamembrane. The walls of host and parasite are fused at the middle lamella common to both organisms and contain pit-like wall specializations. In infected pine material these pits are penetrated by half-plasmodesmata emanating from host cytoplasm. These

half-plasmodesmata end blindly at a thin, unperforated portion of the Arceuthobium cell wall. Cytopathological effects on the host cells appeared mild, although host cell ratios were affected. No apparent anomalies were observed in conjunction with host sieve cell structure and development (Alosi, Berkeley).

III. Life Cycle Studies

a. 1980 seeds of Arceuthobium pusillum collected from five locations were placed on branches of black spruce and eastern larch in each of the past three years. Branches were observed at two-week intervals for one year. Attack by fungi and insects, and seed movement, loss, and germination were recorded. These data are currently being analyzed (F. Baker and D. W. French, Univ. of Minnesota).

b. Seed dispersal of Arceuthobium pusillum was monitored at two locations in Minnesota in 1978. Seed dispersal began August 25 and continued through September 26 on the plots near Togo, Minnesota. The mean distance of dispersal here was 2.1 meters. In the Fond du Lac State Forest, seed dispersal began September 2, and continued through September 25. An unusually wet period reduced dispersal, until a storm with winds greater than 20 miles per hour caused most of the seed to be expelled. Seeds were dispersed an average of 1.4m from the source trees (F. Baker and D. W. French, Univ. of Minnesota).

c. The mode of pollination was studied in two dwarf mistletoes, Arceuthobium douglasii and A. strictum, from Utah and northern Mexico. Insect visitations to the staminate flowers of A. strictum were common. Visits were found to be rare, however, to the pistillate flowers and to either sex of A. douglasii. Pollen was found to be consistently liberated by the wind from the well-exposed anthers. Air-borne pollen concentration decreased rapidly with distance from the source. No inferences, however, could be made from the data as to dispersal distance. The paucity of insect visitations, the abundance of air-borne pollen, and the moderate to high seed set combined with the absence of apomixis (in A. douglasii) were considered evidence for an anemophilous pollination syndrome. The genus as a whole appears to be anemophilous based on the uniformity of the reproductive morphology. The floral characteristics of Arceuthobium were re-evaluated with respect to their suggested role in wind pollination. (Published in Ecological Monographs 49:73-87, 1979.) (G. Player, Univ. of Utah.)

IV. Host-Parasite Relations

a. The electron microscope evidence above indicates that nutrient acquisition does not involve direct flow of nutrients via interspecific plasmatic bridges. In the absence of plasmatic integrity, it must be assumed that host-originating photosynthate, normally mobile in host phloem cells, is leaked into the cell wall region common to both organisms -- where it then becomes available for absorption into the parasite sinker tissue. The cytological evidence suggests that dwarf mistletoe sinker cells are capable of enhanced loading of apoplastically-held material (e.g., that in the free space of the cell walls). A gradient provided by differential starch stores may contribute to the mobilization of water and solutes into aerial shoots (Alosi, Berkeley).

V. Effects on Hosts

a. Studies were conducted on the interrelationships between mountain pine beetle (MPB) and ponderosa pine dwarf mistletoe along the Colorado Front Range. Studies consider both brood production and susceptibility of mistletoe-infected vs. mistletoe-free trees in the same stands. It is a commonly held belief by foresters in the area that MPB does not develop in mistletoe-infected trees but these studies show that there is very little difference in brood production in heavily infected (Class 5 or 6) vs. mistletoe-free trees. In an intensive study of a 1,200-acre area near Fort Collins, MPB-caused mortality was ca. 20% in non-mistletoed trees and 30% in trees infected by dwarf mistletoe. This ratio of about 1:1.5 for mistletoe-free vs. infected trees was fairly consistent for each diameter class so the relationship is probably real (W. F. McCambridge and F. G. Hawksworth, USFS, Fort Collins; and J. G. Laut, Colorado State Forest Service).

b. An evaluation is under way to quantify damage caused by dwarf mistletoe in 40- to 100-year-old western hemlock stands in Western Oregon and Washington. Effects of infection on growth rates and wood quality are being measured. Early results suggest infection causes little impact on growth, but the wood quality may be significantly reduced (Hadfield, Region 6).

c. Data collection on the effects of A. douglasii on Douglas-fir (Pseudotsuga menziesii) in commercial Southwestern mixed conifer forests is under way in the Apache-Sitgreaves National Forest, Arizona. At the end of August approximately 80 temporary 0.2-acre plots had been completed, and over 5,000 Douglas-firs examined. The remainder of the 1979 field season will be spent collecting data in the Gila National Forest, New Mexico and possibly in the Coconino National Forest.

Data collection in 1980 will be concentrated in the Carson, Lincoln, and Santa Fe National Forests, New Mexico. The results of this study will provide needed information on growth and mortality losses in Douglas-fir as a result of parasitism by A. douglasii in the Southwest. Results will also provide information which will aid in the development of improved management guidelines for dwarf mistletoe-infested Douglas-fir stands in Region 3 (R. I. Mathiasen, Univ. of Arizona; F. G. Hawksworth, RM Station, Fort Collins, Colorado; Don Graham, FI&DM, Region 3, Albuquerque, New Mexico; Ed Wood, RI&DM, Region 3, Albuquerque, New Mexico).

VI. Ecology -- No reports received.

VII. Control -- Chemical

a. It is suggested that herbicides that are strongly and specifically mobile in the apoplast[1] (i.e., xylem and cell walls) be tested in regard to their ability to accumulate in dwarf mistletoe tissue. (An example is Monuron.) Another herbicide that warrants testing is dinoben. Dinoben is a selective herbicide which inhibits fatty acid and lipid synthesis in fat-storing plants[2]. It is possible that selective inhibition Arceuthobium sinker cell metabolism can be achieved with this herbicide despite the close physical relationship of endophytic cells with host vascular tissue. This is because sinker cells store lipids almost exclusively, while host cells store mainly starch (in warm months).[3]

(1) Re: Crafts and Crisp. 1971. Phloem Transport in Plants. Pg. 176.
(2) Muslih and Linscott. 1977. Plant Physiol. 60: 730-735.
(3) Herbicides may be most effective if stem-injected below the mistletoe.

(Alosi, Berkeley)

b. Four compounds were evaluated for their ability to kill dwarf mistletoe-infected black spruce. Sodium TCA was effective, inexpensive and did not harm black spruce seedlings planted the following spring. One hundred acres of dwarf mistletoe-infected black spruce were treated in July 1979. The results of this operational test will be available in July 1980 (F. Baker and D. W. French, Univ. of Minnesota).

c. Continuing a greenhouse screening program for mistletoe poisons -- especially among the herbicides (Knutson, PNW).

VIII. Control -- Biological

a. We published a report on *Wallrothiella* on *Arc. douglasii* in a recent issue of *Mycologia* (Knutson and Hutchins, PNW).

b. Studying the infection biology of *Cylindrocarpon gillii* of various host species, but with emphasis on Western hemlock (Hutchins and Knutson, PNW).

c. A field survey for hyperparasites on the genus *Korthalsella* (Loranthaceae) in Hawaii was made. *Teichospora obducens* and its *Phoma* state were associated with lesions on shoots of *K. complanata* at three locations on the Island of Hawaii. The fungus was pathogenic when applied to nodal regions of the mistletoe. Unidentified microlepodoptera larvae were associated with some lesions. A similar fungus, *Pleospora* sp., was found on *K. latissima* on the Island of Kauai; pathogenicity was not tested. *Colletotrichum gloeosporioides* was associated with shoot dieback of *K. complanata* and was pathogenic in field inoculations. Other fungi isolated from shoot dieback of *K. complanata* but not pathogenic in a field test, included *Pestalotia* spp., *Fusarium* and *Botryodiplodia* (J. T. Kliejunas and R. S. Smith Jr., USFS, Region 5; R.F. Scharpf, PSW).

d. A spittlebug (presumably *Clastoptera distincta*) on dwarf mistletoe (*Arceuthobium vaginatum*) was found for the first time in the Central Rocky Mountains (San Juan National Forest, Colorado). Its effects on mistletoe here seem to be negligible (F. G. Hawksworth, USFS, Fort Collins).

e. One of the first attempts to breed trees for disease resistance was begun in the early 1920's in Colorado for mistletoe-resistant ponderosa pine (C. Bates, *J. For.* 25: 130-144, 1927). Two plantations of trees resulting from controlled crosses were established near Colorado Springs in 1932. The plantations were re-examined in 1979. One plantation had no mistletoe-infected trees nearby so no data on resistance could be obtained. The other plantation was surrounded by heavily-infected residual ponderosa pines. Of the 552 trees planted, 389 (or 70%) were alive, and 222 (57%) were infected. Results were as follows:

		Conditions in 1979		
Seed & Type	Trees Planted in 1932	Trees Live %	Trees Infected %	Average Mistletoe Rating
"Resistant"	367	68	53	1.7
"Doubtful"	120	79	63	1.9
"Susceptible"	65	66	65	1.7

Thus, it is concluded that the putative resistance in these trees was not confirmed. The maximum distance that the parasite had spread into the plantation from the closest residual trees was 68 feet, for an average of ca. 1.4 feet per year (F. G. Hawksworth and C. B. Edminster, USFS, Fort Collins).

IX. Control -- Silvicultural

a. Dwarf mistletoe-infected hemlock were observed in both old growth and cutover mixed hemlock and spruce stands with regeneration of various ages in Southeast Alaska during two weeks in May 1979. Recent research on hemlock mistletoe by Terry Shaw of the USFS in Southeast Alaska, and observations made during this visit, suggest that earlier recommendations aimed at minimizing the adverse effect of this mistletoe in Southeast Alaska were based on pathogen spread and intensification in British Columbia (Vancouver Island), Washington, and Oregon, and may not be appropriate for Southeast Alaska. The parasite seems to be spreading to and intensifying in reproduction at much slower rates than for hemlock dwarf mistletoe in B.C. and in the Pacific Northwest (D. Drummond, USFS, Davis, California, and F. G. Hawksworth, USFS, Fort Collins).

b. To determine the effects of thinning in mistletoe-infected ponderosa pine and lodgepole pine stands, a series of permanent plots (mostly 0.4 acre) are being established. Stands with no mistletoe and those with different intensities of infection are being thinned to various growing stock levels. Also several are being left unthinned as checks. The plots will be used to evaluate the mistletoe and tree responses to thinning. To date, about 120 plots have been established: 95 in ponderosa pine (in Colorado and Arizona) and 25 in lodgepole pine (in Colorado). (F. G. Hawksworth, USFS, Fort Collins.)

c. Removed A. americanum infected overstory lodgepole pine from 262 acres on the Wind River Ranger District, Shoshone National Forest (D. Johnson, Region 2, Denver).

d. Two stands were selected to test prescribed burning as an operational means of killing dwarf mistletoe-infected residual black spruce. The first site was strip cut, and prescribed burning was successful, but cost $77 per acre. The second site was harvested with a shear, and the trees skidded full length from the stand. The remaining slash was not adequate to carry a fire, but burning was unnecessary, since all infected residuals were destroyed. In other stands where similar harvesting equipment and methods were used, infected

residuals were not killed. Snow depth, frost depth and operator skill are factors affecting the success of mechanized harvesting in accomplishing the elimination of infected trees (F. Baker and D. W. French, Univ. of Minnesota).

e. A revised Region 6 Forest Service Manual Supplement was issued describing dwarf mistletoe management policies (Hadfield, Region 6).

f. The R-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 this year. Pre-thinning plots have been established on several National Forests in California and data collected. Some thinning of plots has been completed and post-thinning data collected, but most thinning and data collection is scheduled for 1980 (R. F. Scharpf, PSW; R. S. Smith, P. Vogler, Region 5).

g. A manuscript is in press -- "Effect of release, dwarf mistletoe, and live crown ratio on growth of red firs". PSW Station Paper (R. F. Scharpf, PSW).

h. Infected Douglas-fir trees on the Okanogan National Forest have a higher mortality than do uninfected trees. Analysis now under way will determine whether this mortality is related to site index, stand density and stand age (Knutson, PNW).

i. Summer 1979 saw the re-measurement of 32 plots installed in 10-year-old pre-commercial thinning-dwarf mistletoe control units in Eastern Washington. In the past, there has been some controversy over whether dwarf mistletoe control at that age really works. My observations following re-measurement of the 32 plots scattered throughout the state in both Douglas-fir and ponderosa pine show that although there were a few failures, the bulk of the control action was very successful. Data survey summary is not yet complete, but should show the success and importance of good dwarf mistletoe control emphasis in Eastern Washington.

We demonstrated that when crop trees are left with low ratings (6 point), the mistletoe remains in check long enough for exceptional growth in most cases to be put on the trees in the interval between thinnings. Most of the trees increased five-year diameter increments from eight millimeters average prior to thinning to more than 12 millimeters average in the five years immediately following thinning, and 12 millimeters again in the second five-year period. Occasional trees grew up to 20 millimeters in five years.

This growth does not represent growth where mistletoe was removed from individual trees. Instead it represents good growth placed on trees free of mistletoe and free to grow.

All the mistletoe trees were removed. Management guides based on the second re-measurement would suggest that commercial harvest cutting cycles following pre-commercial thinning should be not much more than 15 years later to pick up new infections. The second thinning should clean the stands quite well and a third thinning 30 years following the original pre-commercial thinning should see some fine volume increment and high quality stands as compared to untreated stands (K. W. Russell, Washington Department of Natural Resources, Olympia).

X. Surveys

a. Plots were established in 1966 and 1976 in A. pusillum infected white spruce in Manitoba, Canada, and read annually since 1976. The greatest impact of dwarf mistletoe is on the reproduction, where 20% were killed in a two-year period. Larger trees may live 40 years or longer after being infected, and may add reasonable increment during this time. A manuscript has been submitted to the Canadian Journal of Forest Research (F. Baker and D. W. French, Univ. of Minnesota; and J. G. Laut, Colorado State Forest Service).

An average of 8% of the black spruce infected with A. pusillum died each year since 1975. Because of the manner in which these plots were established, no statements can be made about yield loss (F. Baker and D. W. French, Univ. of Minnesota).

b. Dwarf mistletoe loss assessment survey -- Bighorn and Shoshone National Forests in Wyoming. As part of a continuing effort to assess growth loss and mortality caused by lodgepole pine dwarf mistletoe, Arceuthobium americanum, in the Rocky Mountain Region, a road-plot survey was conducted during the 1978 field season. The road portion of the survey revealed that 36.3% of the 174 miles traversed were adjacent to mistletoe-infested stands and 22.7% of the plots established in conjunction with the survey contained infected trees for the Bighorn National Forest. The estimated annual cubic foot volume loss for 263,000 acres of lodgepole pine type for this forest was 725,800 cubic feet and 1.6 and 3.9 cubic feet per acre per year for pole-size and sawtimber-size stands respectively.

The road portion of the survey for the Shoshone National Forest revealed that 64% of the 78 miles traversed through lodgepole pine type were adjacent to mistletoe-infested stands and 59.4% of the plots established in conjunction with the survey contained mistletoe-infected trees. The estimated annual loss for 136,000 acres was 698,500 cubic feet per year. For pole-size and sawtimber-size stands, the estimated loss was 3.5 and 5.8 cubic feet per acre per year respectively.

A similar survey was conducted in Colorado during 1979 on all National Forests with lodgepole pine type. The results will be available late in 1979 (D. Johnson, D. Drummond and F. Hawksworth; Region 2, MAG, USFS and RM Station).

c. During 1978 pre-suppression surveys for lodgepole pine dwarf mistletoe, A. americanum, were conducted by the Bureau of Land Management over 7,000 acres and 397 stands in North-Central Colorado. Sixty-two representative stands were processed through the RMYLD Program to estimate total merchantable yields with and without dwarf mistletoe control treatments. Recommendations for scheduling the priority of treatment for these stands is presented. Report available from USFS, Region 2, Report No. R2-79-5 (D. Johnson, Region 2, Denver).

d. A survey procedure to aid in the evaluation of thinning projects in dwarf mistletoe infested lodgepole pine stands is available (D. W. Johnson and F. G. Hawksworth, 1978). An evaluation survey procedure for proposed thinning projects in lodgepole pine stands infested with dwarf mistletoe. USDA Forest Service, Forest Insect and Disease Survey Methods (Manual), 5 pp. plus appendix (D. Johnson, Region 2, Denver).

e. I reported last year on the completion of the field part of an impact survey in lodgepole pine on six east-side forests in Region 1. The data analysis is complete; infestation ranges from 28% on the Custer, to 52% on the Beaverhead. Annual loss ranges from 106,000 cubic feet on the Custer to 2,500,000 cubic feet on the Deerlodge. Details have been published in FI&DM Report No. 79-13. We are extending the impact survey to the rest of Montana; this year we completed the field work on the Lolo and Bitterroot National Forests (O. Dooling, Region 1, Missoula).

f. A survey of incidence of and growth loss caused by dwarf mistletoes was made on 13 of the 16 National Forests in Region 4 in 1978. The study consisted of a roadside and plot survey.

The survey results emphasize the magnitude of the dwarf mistletoe problem in lodgepole pine stands throughout the Region. An average of 60% of the lodgepole pine is infected resulting in a total volume loss of 28,500,000 cubic feet per year.

Most of the growth loss occurs in mature and overmature stands. On some Forests, a substantial amount of the parasite is in pole-sized timber, but at the present there is little growth loss.

Information on the presence of dwarf mistletoes in ponderosa pine and Douglas-fir stands was also collected. Some Forests have up to 65% of the ponderosa pine and 80% of the Douglas-fir infected. Research is needed to quantify the volume loss caused by the incidence of these mistletoes. (Jim Hoffman, Region 4)

XI. Miscellaneous

a. The Second International Symposium on Parasitic Weeds was held in Raleigh, North Carolina in July 1979. The papers presented were primarily on parasites of agricultural crops (Striga, Orobanche, Cuscata, etc.), but there were five on mistletoes. As a result of the meeting, an International Parasitic Seed Plant Research Group was formed. The next symposium will be held in about 1983, at a location to be determined later (F. G. Hawksworth, USFS, Fort Collins).

b. A Forest Insect and Disease Management Leaflet on the six species of Phoradendron that attack conifers has been submitted for publication by the USFS in Washington (F. G. Hawksworth, USFS, Fort Collins; and R. F. Scharpf, USFS, Berkeley).

XII. Addendum

A manuscript has been conditionally accepted by Can. J. Forest Research describing a model of spread and intensification of dwarf mistletoe infection in young western hemlock stands. Authored by W.J. Bloomberg, R.B. Smith, and A. Van Der Wereld, the model is intended to generate silvicultural control guidelines for stands up to 30 years after logging.

Testing of the model is expected to be completed within the next few months using verification plots representing a range of stand ages, densities, and infection levels. (Bloomberg CFS, Victoria).

DISEASE CONTROL COMMITTEE

HIGHLIGHTS OF 1979 CONTROL INVESTIGATIONS

Kenelm Russell, Chairman

Details of annual meeting (Salem) at end of disease control trials section.

I. SEEDLING DISEASES

A. Nursery Root Diseases

Host: Various conifers
Causal Organisms: Pythiaceous and Moniliaceous fungi
Control: Chemical, Biological, Silvicultural
Development Stage: Field Trial

1) 3 chemicals and their combinations as pre and post emergence soil drenches
2) date of sowing
3) sugar pine
4) continuing

(R. Bega and A. McCain, PSW-USFS and U.C., Berkeley, CA)

B. Grey Mold

Host: Pseudotsuga, Tsuga
Causal Organisms: *Botrytis cinerea*
Control: Chemical
Development Stage: Operational

Good operational control using Bravo-Chlorothalonil. (J. Hadfield, USFS-R6, Portland, OR)

C. Fusarium Root Rot

Host: *Pseudotsuga*, *Pinus*, *Abies*
Causal Organisms: *Fusarium oxysporum*
Control: Chemical
Development Stage: Operational

Excellent operational control using 350-420 lbs/acre methylbromide-chloropicrin (MC-33). (J. Hadfield, USFS-R6, Portland, OR)

D. Fusarium Crown Rot

Host: Pines and Fir
Causal Organisms: *Fusarium oxysporum*
Control: Chemical, Biological, Silvicultural
Development Stage: Field Trial

Benomyl, captan and Fusarium monliforme are being applied on a regular basis. Success of the trial depends upon disease pressure; last year there was little disease. Also have a planting date trial. Early planting may be the answer. (A. McCain, U.C., Berkeley, CA)

E. Fusarium Root Rots
Host: Nursery conifers
Causal Organisms: Fusarium spp.
Control: Methylbromide, choropicrin, Benlate
Development Stage: Operational

(J. Hoffman, USFS, Region 1, Boise, ID)

F. Damping-off
Host: Conifers - E. spruce, lodgepole, ponderosa pine
Causal Organisms: Pythium spp., Fusarium spp.
Control: Chemical
Development Stage: Operational

Fumigation of the beds at Mt. Sopris Nursery with 400 lb/acre of Dowfume MC-33 has cut losses from damping-off fungi by over 100% and aids weed control. (L. Gillman, USFS, Region 2, Denver, CO)

G. Container Foliage Diseases
Host: Douglas-fir, western hemlock, Sitka spruce, noble fir, ponderosa pine
Causal Organisms: Primarily Botrytis cinerea, Fusarium and others
Control: Chemical
Development Stage: Operational Trial for registration

Test done under an IR-4 grant. Four chemicals tests: Dithane-M45, Banrot, captan and Bravo, all used with X-77 spreader-sticker. Captan gave very good control of Botrytis. Some phytotoxicity with others. Captan trt also had greener trees. Very good data suitable for registration of several prescriptions. (K. Russell, WDNR, Olympia, WA)

H. Botrytis cinerea
Host: conifers - lodgepole pine
Causal Organisms: Botrytis cinerea
Control: Chemical
Development Stage: Operational, Greenhouse

Because of benomyl tolerance, a combination of different fungicides are being used. (R. James and L. Gillman, USFS, Region 2, Denver, CO)

II. FOLIAGE DISEASES

A. Elytroderma Needle Blight
Host: Ponderosa and lodgepole pine
Causal Organisms: Elytroderma deformans
Control: Silvicultural, remove infected trees to reduce inoculum
Development Stage: Operational

(J. Hoffman, USFS, Region 1, Boise, ID)

B. Swiss Needle Cast
Host: Douglas-fir (primarily Christmas trees)
Causal Organisms: Phaeocryptopus gaumani
Control: Chemical
Development Stage: Operational Trial

Benlate alone, Dithane alone and B + D tank mix are being sprayed on badly infected Christmas trees with a large tractor drawn orchard type mist blower. Early results show the sprays have better quality needles than non-sprayed. Applications made in May, June, July and September. (K. Russell, WDNR, Olympia, WA)

C. Anthracnose
Host: Sycamore
Causal Organisms: Gnomonia platani
Control: Chemical - copper salts
Development Stage: Operational

(J. Hoffman, USFS, Region 1, Boise, ID)

III. STEM DISEASES AND WILTS

A. Dutch Elm Disease
Host: Elms
Causal Organisms: Ceratocystis ulmi
Control: Removal of infected trees
Development Stage: Operational

(J. Hoffman, USFS, Region 1, Boise, ID)

B. Phomopsis Blight
Host: Eastern Red cedar
Causal Organisms: Phomopsis juniperovora
Control: Chemical
Development Stage: Operational

Standard procedures using Bordeaux mixture to control this persistent disease. (R. James and L. Gillman, USFS, Region 2, Denver, CO)

C. Tip Die Back
Host: Austrian Pine
Causal Organisms: Rhizoctonia
Control: Chemical
Development Stage: Greenhouse

PCNB to control Rhizoctonia on Austrian pine. (R. James and G. Boutz, USFS, Region 2 and Kansas State Extension Forestry, Denver, CO)

IV. ROOT ROTS

A. Laminated Root Rot
Host: Douglas-fir et al
Causal Organisms: Phellinus weirii
Control: Chemical
Development Stage: Pre-Field Trial

Monolourin a fatty acid inhibits Pw in culture. We are testing: a) whether it moves down through wood and b) whether it maintains toxicity to P. weirii. Test uses natural root and stump wood in a lab study prior to field testing on stumps. (C. Li, D. Knutson, W. Thies, USFS, Forestry Sciences Lab, Corvallis, OR)

B. Laminated Root Rot
Host: Douglas-fir
Causal Organisms: Phellinus weirii
Control: Biological
Development Stage: Pot Trial

A Bacillus sp. was isolated from soil that produces a heat stable, broad-spectrum antibiotic inhibiting Phellinus weirii, Fomes annosus, Armillaria mellea and Phytophthora cinnamomi. Pot trials have been started using this Bacillus against P. weirii. (A. Hutchins, Forestry Sciences Lab, Corvallis, OR)

C. Laminated Root Rot
Host: Douglas-fir, western red cedar, W. hemlock, Sitka spruce, white pine and red alder
Causal Organisms: Phellinus weirii
Control: Silvicultural
Development Stage: Field Trial Setback (9 years)

A disastrous fire wiped out our valuable species tolerance trial near Concrete, WA. Planted trees were 7-9 years old and approaching head high in some species. We are going to replant. (K. Russell, WDNR, Olympia, WA)

D. Laminated Root Rot
Host: Douglas-fir
Causal Organisms: Phellinus weirii
Control: Silvicultural
Development Stage: Field Trial - 7 years

Red alder was planted alternately with Douglas-fir on 4 x 4' spacing. A companion plot of pure fir, same spacing, adjoins the combination plot. In 1979 approximately 7-9% of DF have died from P.W. Almost all mortality is on the pure fir plot. (K. Russell, WDNR, Olympia, WA)

E. Laminated Root Rot
Host: Douglas-fir
Causal Organisms: Phellinus weirii
Control: Chemical
Development Stage: Pot Trial

Phellinus weirii infected roots have been buried in lime treated soil. Douglas-fir seedlings have been planted in this soil to test if: (1) change in pH of soil protects seedlings from infection by P. weirii, (2) if P. weirii infected roots survive the change in pH of soil. (A. Hutchins, USFS, Forestry Sciences Lab, Corvallis, OR)

F. Laminated Root Rot
Host: Douglas-fir
Causal Organisms: Phellinus weirii
Control: Silvicultural
Development Stage: Field Trial

We are installing a control demonstration area incorporating commercial thinning guidelines, crop rotation, salvage and bulldozing. Preliminary mapping now, treatments 1981? (E. Hansen, OSU, Corvallis, OR)

G. Root Rots
Host: Western conifers
Causal Organisms: Phellinus weirii and Fomes annosus
Control: Chemical, Biological
Development Stage: Field Trial

(C. Li, Forestry Sciences Laboratory, Corvallis, OR)

H. Annosus Root Rot
Host: Douglas-fir
Causal Organisms: Fomes annosus
Control: Biological
Development Stage: In Vitro

Streptomyces griseoloalba, isolated from roots, inhibited Phellinus weirii, Fomes annosus and Phytophthora cinnammomi in culture media. This organism is being tested in field trials against Fomes annosus. (Rose, C. Li, A. Hutchins, Forestry Sciences Lab, Corvallis, OR)

I. Armillaria Root Rot
Host: P. pine, Douglas-fir
Causal Organisms: Armillaria mellea
Control: Silvicultural
Development Stage: Field Trial

1) Plots in Sisters, OR have been in for 13 years now (Dave Johnson's study). We will read data this year.

2) Plots on Blue River Rd. (Willamette NF) and White River Rd. (Mt. Baker-Snoqualmie NF) have been in for one year now and will be read this year. (G. Filip, USFS, Region 6, Portland, OR)

V. RUSTS

A. Western Gall Rust
Host: Ponderosa and lodgepole pine
Causal Organisms: Peridermium harkmesii
Control: Branch pruning
Development Stage: Operational

(J. Hoffman, USFS, Region 1, Boise, ID)

B. Commandra Rust
Host: Ponderosa and lodgepole pine
Causal Organisms: Cronartium commandra (Idaho)
Control: Cut seedlings and older spike tops to reduce inoculum for rust
Development Stage: Operational

(J. Hoffman, USFS, Region 1, Boise, ID)

Meeting Notes

Seven or eight of us met over lunch and recommend the following:

1. Concentrate on securing registrations (24 C's) or equivalent for new fungicides. We lose if we don't keep up!

2. Try new fungicides; one is Ciba-Geigy 49948 (subdue) a systemic.

3. Captan is a good fungicide for Botrytis control in container greenhouses.

4. A report is available through the USFS Washington office on operational disease control recommendations nationwide. Direct inquiries to Dan Brown, c/o USFS WA.

5. A minor change in format will be used next year for disease control listings. The investigator will be moved into a special heading.

Last year there were 24 entries for this report. In 1979 there were 25. Keep it up. Your contributions help us know who is doing what.

ROOT DISEASE COMMITTEE

Greg Filip, Chairman

The following reports were presented at the 1979 meeting:

Biology

1) Effects of edaphic factors on Ceratocystis wageneri (F. Cobb, U. C. Berkeley)

2) Verticicladiella wagenerii on Pinyon pine in Mesa Verde National Park--spread rates, viability after host death, insect relationships (B. James, FIDM Lakewood)

3) Armillaria mellea at Glenwood, Washington--spread rates and impact on reforestation (K. Russell, WDNR Olympia)

Control

1) Stump treatments for Fomes annosus in precommercially thinned stands (G. Wallis, PFRC Victoria)

2) Susceptibility of conifers to Phellinus weirii in Oregon and Washington--field trials (W. Thies, PNW Corvallis)

3) Selection and breeding of Douglas-fir for resistance to P. weirii (C. Driver, U. of Washington, Seattle)

Surveys

1) F. annosus and A. mellea distribution in Colorado (B. James, FIDM Lakewood)

2) Testing of survey methods to detect area infected by root disease (G. Filip, FIDM Portland).

Reports submitted but not presented:

1) Mycorrhizae inoculation trials, biological and chemical control trials, and date of sowing trials at various California nurseries (B. Bega, PSW Berkeley)

2) Studies on C. wageneri: (a) infection, colonization, and symptom development in ponderosa pine trees, b) overland spread and vectors, and c) variability and host range (F. Cobb, UC Berkeley.

3) Relationship of underground quantities of Armillaria inoculum to potential for disease spread (R. Fuller, FIDM Lakewood).

4) Comparison of various isolates of Verticicladiella. Use of resistant rootstocks of ornamental lawson cypress to control Phythophthora lateralis (R. Hunt, PFRC Victoria).

5) Monitoring F. annosus inoculum potential in Montana; dynamics of P. weirii centers in northern Idaho; survey of A. mellea using optical bar CIR imagery in Montana; modeling of stand parameters associated with root disease centers; and soil-root disease associations in northern Idaho (R. Williams, FIDM Missoula).

6) Testing of a newly-developed survey method to detect area incidence of root disease; correlations between stem diameter growth and cross-sectional area of P. weirii-infected roots, and relationship between root contacts and soil depth, tree size, stand density, slope, root type and size (W. Bloomberg, PFRC Victoria).

7) Evaluation of black stain root disease in Eldorado County and a five-year project involving F. annosus root disease (J. Byler and W. Freeman, FIDM San Francisco).

Greg Filip was elected (unopposed) chairman for the 1980 meeting. Meeting format is to remain the same. Chairman will assist Gordy Wallis in organizing a root-disease workshop in Corvallis in February 1980.

PROBABILITY OF TREE FAILURE

Tables for probability of failure of trees are being developed for forest and urban sites. Table values indicate the probability that a tree of a given species and diameter will not live to achieve the next larger one-inch diameter class without suffering failure of the type specified. All of the trees are drawn from populations where some target of value is at risk; the trees represent only unexpected single failures. Such tables provide a guide for the evaluator especially when no gross indications of potential failure are evident. Used together with the tables of expected loss, the guides to probability of failure permit more reliable evaluation of hazard particularly for large, healthy-looking trees. The tables do not apply to catastrophe failures which, in any case, result in very low accident rates.

(Lee A. Paine, Pacific Southwest Forest and Range Experiment Station)

NEW AND ACTIVE PROJECTS

A. Forest Disease Surveys - General

71-A-1 *Dermea pseudotsugae* survey (M. Srago).

71-A-2 Aerial photography and root disease surveys (F.W. Cobb, J. Byler, J. Caylor).

71-A-3 Impact evaluation of white pine blister rust (J. Byler, N. MacGregor).

71-A-4 Appraisal of damage caused by forest pests in B.C. (G.A VanSickle).

71-A-t Detection and reporting, forest insect and disease survey (D.A. Ross). (Formerly 68-A-1).

71-A-10 Development of a continuous nursery disease survey (W.J. Bloomberg).

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

71-A-9 Forest insect and disease survey in the Prairie Provinces, Yukon and Northwest Territories (W.G.H. Ives, Y. Hiratsuka, and H.R. Wong).

73-A-3 Pest damage inventory (J. Byler and D. Hart).

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 *Verticladiella* in Douglas-fir in Oregon (E.N. Hanson).

77-A-1 Evaluation of the extent and cause of tree mortality on the San Bernadino National Forest (Byler, Cobb, and Ed Wood).

78-A-1 Evaluation of jack pine mortality on the Nebraska National Forest (R. James).

79-A-1 Computer integration and analyses of tree-problem-survey data: frequencies, locations, associations, volume losses (A.D. Partridge).

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

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

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

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

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

78-B-1 Evaluation of salt damage to conifers along roadsides in Colorado (R. James).

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

C. Cone, Seed, and Seedling Diseases

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

73-C-1 Composting for organic matter additives at the nursery (K.W. Russell).

73-C-2 Pathology of forest seedlings in storage (J.C. Jopkins).

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

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

76-C-3 Potential of several species of *Phytophthora* for damage to coniferous forests and forest nurseries (E. Hansen, L. Roth).

76-C-4 Effects of pathogen control on performance of container-grown Douglas-fir seedlings (Thies, Owston).

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

78-C-1 Fungicidal tolerance of Botrytis cinerea (L. Gillman and R. James).

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

78-C-3 Chemical and biological control of sugar pine root diseases at U.S.F.S., Placerville Nursery using seven fungicides and one suppressive soil (R.V. Bega).

79-C-1 Evaluation of fumigation with Dowfume ® MC-33 at Mt. Sopris Nursery (Gillman, Hildebrand).

79-C-2 Diseases of Cottonwood cuttings at the Kansas State Forestry Nursery (James and Boutz).

79-C-3 Diseases of containerized conifers at the Kansas State Forestry Nursery (James and Boutz).

79-C-4 Identification of fungi on Northern Region conifer seed, their detrimental effects, and methods to reduce detrimental effects (J. Woo, INT; R.E. Williams, R-1; J. Guthrie, U. of Idaho).

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).

72-D-2 Armillaria mellea root rot: importance and biology (D.J. Morrison).

72-D-3 Identification, distribution and intensity of root rots in western Montana and northern Idaho (R.E. Williams). (Formerly 67-D-5).

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

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

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

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

73-D-5 Fomes annosus evaluation (M. Srago).

74-D-1 Distribution and epidemiology of Verticicladiella wagenerii on pinon pine in Colorado (L.B. Helberg).

74-D-2 The role of ectotrophic mycelium in the initiation of Phellinius (Poria) weirii infections (E.M. Hansen).

74-D-3 Survival infectivity of P. weirii in Douglas-fir stumps (E.M. Hansen).

74-D-4 Changes in severity of P. weirii resulting from forest management (E.M. Hansen).

74-D-5 Cytology and sexuality of P. weirii (E.M. Hansen).

74-D-6 Silvicultural prescriptions for managment of stands affected by root diseases (C.D. Leaphart, R.E. Williams).

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

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

76-D-1 An evaluation of Verticicladiella in Oregon (E. Hansen).

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

76-D-5 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-2 Light and temperature induced sporophore formation of Phellinus weirii (C.Y. Li).

77-D-3 Effects of lipids on colonization of wood disks of Fomes annosus (C.Y. Li).

77-D-4 Soil bacterial populations antagonistic to Phellinus weirii in 40-year-old alder and pure conifer stands of coastal Oregon (C.Y. Li).

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

77-D-6 Identification of site factors associated with Poria root disease and rating stands for probability of damage in northern Idaho (R.E. Williams).

77-D-7 Identification of chemical and physical characteristics of minaloosa soils which are highly correlated with high incidence of Poria root disease in northern Idaho (R.E. Williams).

77-D-8 Dynamics of Poria root disease centers in northern Idaho (R.E. Williams).

77-D-9 Airborne spore density of Fomes annosus in selected sites in west rn Montana (R.E. Williams).

77-D-10 Inoculation of containerized Engelmann spruce and lodgepole pine seedlings with ectomycorrhizal inoculum from two potential natural sources and an artificial (laboratory-produced) source for comparison of effectiveness (D. Hildebrand, L.S. Gillman and T.D. Landis).

77-D-11 Evaluation of Pisolithus tinctorius inoculum produced by Abbott Laboratories for ectomycorrhizal development in Region Two nurseries (L.S. Gillman, C.E. Cordell).

77-D-12 Develop a format with which to integrate disease and insect information with biotic, climatic, and edaphic site factors so that potential for damage may be identified and used in the land management planning process (R.E. Williams, M. McGregor, O.J. Dooling).

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

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-2 Expanded survey for root disease centers on three National Forests on about four million acres (R. Williams).

78-D-3 Dynamics of root disease centers and environmental factors correlated with centers (R. Williams).

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-8 Chemical control of Armillaria near Glenwood, Washington (K.W. Russell).

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

78-D-10 Biology of Phaeolus schweinitzii (S. Dubreil and A.K. Partridge).

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

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

79-D-3 Verticicladiella wagnerii on Pinyon pine at Mesa Verde National Park - disease spread characteristics and vector relationships (James and Lister).

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

79-D-5 Spread of Armilariella mellea disease centers in managed pine stands (Gillman and James).

79-D-6 Survey of root disease centers on the Nezperce National Forest, Idaho (R.E. Williams).

79-D-7 Survey of root disease centers on the Lolo National Forest, Montana (R.E. Williams).

79-D-8 Evaluation of color infrared optical bar imagry to survey for Armillaria mellea root disease centers on the Lewis & Clark National Forest, Montana (R.E. Williams).

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

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

79-D-12 Evaluation of root disease in 50 year 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 F 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-19 Root-disease fungi and bark beetle interaction in ponderosa pine in southcentral New Mexico (W.H. Livingston, A.C. Mangini and H.G. Kinzer).

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

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

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 (A. Harvey).

79-D-26 Pathogenicity of Verticicladiella spp. and interaction of Verticicladiella spp. and associated insects (A.D. Partridge).

79-D-27 Comparison and evaluation of root disease survey methods (A.D. Partridge).

79-D-28 The sequences and interactions of organisms causing mottled-root-disease in northern Idaho (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, cooperation with PNW Station).

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

E. Foliage Diseases

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

71-E-1 Elytroderma deformans - Mortality and growth impact on Jeffrey pines (R.F. Scharpf and R.V. Bega).

76-E-2 Evaluation of the growth impact of Rhabdocline pseudotsugae on sapling Douglas-firs in western Oregon (D. Goheen).

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

77-E-2 Inheritance of resistance to Dothistroma pini in Austrian pine (Joint with RWU 1501) (G.W. Peterson and D.F. Van Haverbeke).

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-1 Life tables for lodgepole pine and ponderosa pine dwarf mistletoe (F.G. Hawksworth and T.E. Hinds).

62-F-2 Ecology of lodgepole and ponderosa pine dwarf mistletoes (F.G. Hawksworth).

62-F-4 Taxonomy, hosts, and distribution of Arceuthobium (F.G. Hawksworth and D. Wiens).

62-F-5 Silvicultural control of ponderosa pine dwarf mistletoe in the Southwest (F.G. Hawksworth).

63-F-1 Spread and intensification of dwarf mistletoe in ponderosa and Jeffrey pines in California (R.F. Scharpf, and J.R. Parmeter).

65-F-1 The effect of dwarf mistletoe on growth of western hemlock (K.W. Russell).

68-F-1 Silvicultural control of dwarf mistletoe in young lodgepole pine stands (G.A. Van Sickle).

68-F-2 Silvicultural control of lodgepole pine dwarf mistletoe (Dave Johnson, F.G. Hawksworth, T.E. Hinds).

68-F-4 Spread and intensification of dwarf mistletoe in young unistoried stands of western larch, Douglas-fir and lodgepole pine with controlled stocking (E.F. Wicker and C.D. Leaphart).

71-F-1 Growth impact, associated mortality, and spread and intensification of dwarf mistletoe in stands of Douglas-fir, lodgepole pine, and western larch (O.J. Dooling and E.F. Wicker).

71-F-2 Dwarf mistletoe control in rural and suburban residential developments (J.G. Laut and F.G. Hawksworth).

72-F-1 Simulation of the effects of dwarf mistletoe in ponderosa pine and lodgepole pine stands (F.G. Hawksworth, T.E. Hinds, and C.B. Edminster).

76-F-4 Inoculation studies to determine the host ranges of Arceuthobium campylopodum and A. occidentale in California (W. Mark, R. Scharpf, F.G. Hawksworth).

76-F-5 Biology and epidemiology of a Peridermium associated with lodgepole pine dwarf mistletoe (F.G. Hawksworth).

77-F-1 Evaluation of herbicides as chemical control agents of dwarf mistletoe (D.M. Knutson).

77-F-6 Implementation of the simulated yield program SWYLD2 in dwarf mistletoe-infested ponderosa pine stands (J. Walters).

78-F-1 Expanded field plot study (into SW Oregon) of Douglas-fir dwarf mistletoe development in thinned precommercial stands (D. Knutson). (modification of 73-F-2).

78-F-2 Control of dwarf mistletoe-caused losses in young true fir stands by thinning (R.S. Smith and R.F. Scharpf).

78-F-3 Population dynamics of dwarf mistletoe on true firs in California (R.F. Scharpf, and J.R. Parmeter).

78-F-4 The effect of dwarf mistletoe on mortality and volume loss in released true fir stands (R.F. Scharpf).

78-F-5 Reduction of dwarf mistletoe-caused mortality of Jeffrey pines by broom pruning (R.S. Smith and R.F. Scharpf).

79-F-1 Lodgepole pine dwarf mistletoe surveys on the Gunnison National Forest (Johnson).

79-F-2 Dwarf mistletoe loss assessment survey - Colorado (Johnson, Hawksworth, and Drummond).

79-F-3 Dwarf mistletoe loss assessment in Douglas-fir, lodgepole pine and western larch on Montana and north Idaho National Forests (O.J. Dooling).

79-F-4 Dwarf mistletoe infection in young-growth western hemlock beneath infected old-growth residuals in southeast Alaska (Terry Shaw, cooperation with FIDM, R-10).

79-F-5 Genetics of resistance of western hemlock to dwarf mistletoe (B. van der Kamp).

79-F-6 Relationship between spread of dwarf mistletoe and stand development in western hemlock (B. van der Kamp).

79-F-7 Growth loss in managed, even-age, dwarf mistletoe infested stands of ponderosa pine in the PNW (E.E. Nelson).

79-F-8 Impact of dwarf mistletoe in the Intermountain Region (J. Hoffman).

79-F-9 Evaluation of losses and managment implications of Douglas-fir dwarf mistletoe in the Southwest (R.L. Mathiasen and R.L. Gilbertson).

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).

72-G-3 Studies of the host range and control of canker stain Ceratocystis fimbriata f. platani of plane tree (A.H. McCain).

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-3 Decay hazard in advanced regeneration of tolerant conifers in Oregon and Washington (P.E. Aho) (Formerly 49-G-6).

73-G-4 The role of microorganisms associated with bark beetles attacking conifers (H.S. Whitney). (Formerly 72-G-5).

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

76-G-1 Incidence, extent and rate of decay associated with grand fir tops killed by the Douglas-fir tussock moth (Aho, Wickman).

77-G-1 Fomes fraxinophilus stem rot of green ash: incidence and damage (J.W. Riffle and R. James).

79-G-1 Evaluation of decay in released stands of advanced grand and white fir regeneration in eastern Oregon and Washington (G. Filip and P.E. Aho).

79-G-2 Stand thinning and change in resistance of ponderosa pine to inoculation of blue stain fungi associated with the round headed pine beetle (W.H. Livingston).

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, M. Srago and G. Fiddler).

79-G-5 Decays and cavity nesting birds in the Pacific Northwest (A.D. Partridge).

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).

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 inter-species hybrids, toward accumulation of a pool of rust-resistance genes from other white pines of the world (R.T. Bingham).

66-H-1 Comparitive 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).

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).

69-H-1 Thinning and pruning western white pine to control the blister rust disease (R.E. Williams and N. Martin).

71-H-3 Forest tree rusts of western North America (Y. Hiratsuka). (Formerly 67-H-8 and 67-H-9).

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).

78-H-1 Survey of the incidence and damage caused by conifer broom rusts in Colorado (D. Johnson and L. Gillman).

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

79-H-2 Evaluations of canker diseases on energy plantation hardwoods in Kansas (James and Boutz).

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).

79-H-5 Distribution and impacts of stalactiform rust in the Inland Northwest (A.D. Partridge).

I. Wilt and Blight Disease

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

74-I-1 Control of Dutch elm disease using vector pheromones (Coop. with USFS, NEFES and CSFS, C.B. Helburg, D.E. Leatherman and 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-1 Dutch elm disease control demonstration project in Colorado (D. Johnson and J.G. Laut).

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

J. Defects and Decays of forest products

58-J-1 Deterioration of beetle-killed Engelmann spruce in Colorado (T.E. Hinds).

68-J-1 Deterioration of stored pulp chips in outdoor piles (R.S. Smith).

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).

71-J-2 An analysis of aspen chip deterioration during outside storage (R.S. Smith and Mrs. C.B. Johansen).

71-J-3 Bioassay of pentachlorophenol within cell walls of Cellon-treated wood (W.W. Wilcox).

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).

72-J-4 Effect of pathological conditions of properties and utilization of California woods (W.W. Wilcox).

73-J-1 Interaction of fungi and chemicals - pentachlorophenol (A.J. Cserjesi). (Formerly 66-J-2).

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

K. Miscellaneous Studies

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

67-K-2 Factors involved in mechanical failure of hazardous trees in 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 Mycospaerella on Salicaceae in western interior of Canada (H. Zalasky).

71-K-5 Winter injury in poplar - a histological study (H. Zalasky).

71-K-6 Prevention of winter injury to conifers and other hardwoods (H. Zalasky).

72-K-1 The pathology of Ohia decline in Hawaii (C.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 (K.W. Russell) Objective: to develop a guide on how to do it right for architects, planners, contractors, and homeowners.

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).

76-K-1 Developing guidelines for silvicultural control in immature western hemlock stands PC-40-275 (W. Bloomberg, R. Smith and A. Thomson).

76-K-2 Etiology and importance of a new twig disorder of aspen (C. Livingston and T. Hinds).

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

78-K-1 Effect of thinning on the incidence and impact of cytospora canker, fir engraver beetle, and Fomes annosus in white fir stands on the Eastside Sierra Nevada (E. Wood, G. Ferrell, R.F. Scharpf, and J.R. Parmeter).

78-K-2 Reduction in stem volume of grand firs defoliated by western spruce budworm outbreaks on the Payette National Forest, Idaho (G. Ferrell and R.F. Scharpf).

79-K-1 Use of the Shigometer for assessment of tree vigor and growth in 25 to 100 year old Sitka spruce and western hemlock (Terry Shaw).

79-K-2 Mortality of Douglas-fir: biotic systems and impacts (A.D. Partridge).

79-K-3 Managment alternatives in forests with Douglas-fir mortality centers (A.D. Partridge).

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

NEW PUBLICATIONS

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addendum

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BUSINESS MEETING MINUTES

The business meeting of the 27th Conference was called to order by Chairman Tom Laurent at 10:30 AM, September 28, 1979.

The minutes and the treasurer's report as printed in the Proceedings of the 26th Conference were approved.

Old Business and Committee Reports

Dwarf Mistletoe Committee. Met for lunch on September 26, 1979. The committee report by Chairman John Laut is included in the Proceedings.

Root Disease Committee. Met for lunch September 25, 1979. The committee report by Chairman Greg Filip is included in the Proceedings. The Chairman was reelected for the 1980 conference and offered to assist Gordy Wallis in organizing a root-disease workshop in Corvallis during the week of February 11, 1980.

Disease Control Committee. Met for lunch on September 28, 1979. The committee report on highlights of 1979 control investigations by Chairman Ken Russellis included in the Proceedings.

Interim Program Chairman. John Schwandt's report follows the Treasurer's report.

New Business

Meeting Place. John Laut discussed the pros and cons of holding the 28th Conference at Pingree Park, Colorado State University Mountain Campus, 55 miles west of Fort Collins. A show of hands revealed a majority in favor of Pingree Park.

Bart van der Kamp invited the 29th Conference to western Canada. John Schwandt extended an invitation to hold the Conference in Idaho. Bob Scharpf moved and the motion was carried that the 29th Conference be held in Canada in 1981.

Election of Officers. Dick Parmeter, chairman of the Election Committee, and members, using a modified 7 point dwarf mistletoe rating system, recommended the candidates for the coming year. By the usual quick democratic process of accepting the Committee's recommendations, Bob Gilbertson was elected Chairman and Oscar Dooling, Secretary-Treasurer.

Honorary Life Members. Lewis F. Roth was "graduated" to Honorary Life Member.

National Symposium on Forest Entomology and Pathology. Frank Hawksworth announced that this National Symposium will be held in Uruapan, Mexico on February 18 and 19th, 1980. He plans to attend and will give a report on the Symposium at next year's WIFDWC meeting.

The meeting adjourned at 11:30 AM.

WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE

TREASURER'S REPORT

Balance on hand following twenty-sixth meeting..... $ 326.95

Interest paid September 8, 1979..... $ 2.99

Total..... $ 329.94

Receipts - Twenty-seventh WIFDWC Meeting

Registration (96 persons)..... $ 1,690.00

Expenses - Twenty-seventh WIFDWC Meeting

Conference room rent.....	$	300.00
Coffee.....	$	234.60
Field trip bus rental.....	$	178.86
Banquet.....	$	778.50
Hospitality room & field trip supplies.....	$	140.50
Miscellaneous and tips.....	$	25.10
Total.....	$	1,657.56

Balance from Twenty-seventh WIFDWC Meeting (1,690.00-1,657.56) - $ 32.44

Balance October 18, 1979..... $ 362.38

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

Our thanks to Ken Russell for maintaining the continuous WIFDWC account at the Washington State Employees Credit Union.

INTERIM PROGRAM CHAIRMAN'S REPORT

John W. Schwandt

The following topics were suggested for the 1980 WIFDWC meeting at Pingree Park in Colorado:

1. A workshop demonstration of economic analysis methods for justifying forest disease control efforts.

2. A panel/workshop concerning roadside dwarf mistletoe surveys. What have we learned (if anything) from them?

3. A workshop to demonstrate survey systems and impact measurements for root diseases.

4. A nomenclature workshop to compile a list of acceptable common and scientific names for fungi.

5. A workshop/demonstration of how to recognize needle diseases, their impact and control.

6. A workshop/demonstration of the Shigometer and CODIT theory; its problems and potential in the West.

7. A panel discussion of disease problems associated with environmental factors (e.g., moisture stress, sunscald, winter injury, etc.)

8. A panel discussion of site characteristics associated with disease problems.

9. A panel discussion of the role of forest products in determining research priorities.

10. A panel/workshop concerning methodology and mechanisms for getting disease management prescriptions to the public and to the land manager.

11. A panel discussion of reforestation problems.

12. A panel discussion of pest control as a part of vegetation management in recreation areas.

Other topics mentioned:

Decay in true-fir forest types.

Root disease - bark beetle interactions.

The natural role of disease in the ecosystem.

Root diseases in plantations.

Stalactiform rust distribution and impact.

Program Format and Proceedings Suggestions

Concentrate on a couple of specific tree species (as was done with hemlock and white pine this year).

Have fewer papers read and more discussions on specific topics.

Have a few small group discussions held simultaneously.

Have more workshops ("How to") and less panel discussions.

Rearrange seating to encourage more discussion.

Have participants bring 100 copies of "new", "active", and "terminated" projects.

Include a list of past meetings and officers in the proceedings.

Make an award for the best T-shirt slogan.

Do away with initial introductions.