PROCEEDINGS OF THE 15th ANNUAL WESTERN INTERNATIONAL FOREST DISEASE WORK CONFERENCE

Santa Fe, New Mexico
September 1967

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FOREWORD

The ancient city of Santa Fe, New Mexico, with its splendor of charm and atmosphere provided the historic environ for our Fifteenth Western International Forest Disease Work Conference held September 11-15, 1967. The Desert Inn Motel served as headquarters for registration, lodging, and personality adjustments. The meetings were held in the auditorium of the State Land Office Building. Attendance was excellent, although unfortunately, members and guests from Mexico were unable to attend.

Chairman Alex Molnar formally opened the meeting with an acknowledgement of officers; welcome to members, guests, and visitors; and a resumé of the program. A welcoming address emphasizing forest disease problems in the Rocky Mountains was presented by Lloyd Hayes. Another welcoming address by Roy Bond on behalf of Region 3 related the importance of forest disease problems relative to land management in New Mexico and Arizona. The mayor of Santa Fe was unable to attend the opening of the Conference, but the fine cooperation, services, and information provided by the Santa Fe Chamber of Commerce effected a very cordial welcome. In keeping with Conference traditions, excellence and authority were inherent in the four panel presentations. This can only reflect favorably on the splendid efforts of the program chairman and his committee. The five special papers were both interesting and informative.

As usual, the field trip was an enjoyable aspect of the meeting. The visit at Los Alamos Scientific Laboratory was exciting and inspiring. The dwarf mistletoe control studies at Bandelier National Monument were informative and stimulating, and the aesthetics of the museum and cliff dwellings were enjoyed and frequently photographed by all attending. The banquet terminated the field trip. Following the dinner of prime roast beef, Dr. Albert Schroeder, Archeologist of the U. S. Park Service, presented an exquisite resumé of the past culture of some of the historic Indian tribes of the Southwestern United States.

The membership accepted Dr. C. D. Leaphart's invitation to reconvene in Coeur d'Alene, Idaho or vicinity, in 1968. Having achieved a successful climax, the Conference adjourned the afternoon of September 15, 1967. It will long be remembered for its excellence of quality.

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PAUL D. KEENER
1908-1966

Dr. Paul D. Keener died unexpectedly on August 6, 1966, in Tucson, Arizona of a heart attack. He is survived by his widow and by two sisters, Miss Jane D. Keener of Washington Grove, Maryland, and Miss Ruth D. Keener of New York City.

Professor Keener was born November 4, 1908, in Lawrenceville, New Jersey, where he attended local grade and high schools prior to enrolling at the Pennsylvania State College (now University). He was graduated with honors in 1932, having a senatorial scholarship during his junior and senior years as a botany major. He continued as a graduate stipend scholar and received his Master of Science from the same institution in 1934. Following service first as a field agent in the barberry eradication program of the U.S. Department of Agriculture from 1935 to 1939, and later with the Boyce Thompson Institute for Plant Research from 1939 to 1941, he enrolled in the doctorate program in mycology at the University of Pennsylvania. He received his Doctor of Philosophy degree in 1945, having held successively a scholarship from the George Leib Harrison Fund.

After joining the staff of the Department of Plant Pathology at the University of Arizona in 1945 as an Assistant Professor and Assistant Plant Pathologist, he advanced through the ranks, attaining his professorship in 1959. Although trained primarily as a mycologist and forest pathologist, he adapted readily to solving both basic and applied problems of significance to Arizona's agricultural industry. As forest pathologist, he made many contributions to our knowledge of the needle cast and rust fungi of Arizona's coniferous species. Under his curatorial direction the mycological herbarium of the Agricultural Experiment Station developed into one of the best reference collections in the Southwest.

Paul Keener devoted his life to scholarly pursuits and service to others. His research was characterized by meticulous attention to detail. A similar degree of thoroughness was apparent in his teaching, and the high standards he set for himself were expected of his students and colleagues.

He was active in the local affairs of Sigma Xi and Phi Kappa Phi, and was instrumental in organizing the Arizona Chapter of Gamma Sigma Delta and served as its first president in 1958.

In the death of Paul D. Keener we have lost a true friend and loyal colleague. He will be deeply missed by us all.

Paul C. Lightle

JOHN ERTEL BIER

Forest pathologists throughout the world were deeply saddened by the untimely death on January 24, 1967 of Dr. J. E. Bier. This loss was felt in full measure by members of the Western International Forest Disease Work Conference as Dr. Bier was not only an active participant and former Secretary and Chairman of the Conference, but was also a primary instigator of action which led to its formation.

Dr. Bier, Jack to those of us who were privileged to regard him as a personal friend as well as professional colleague, received his B.S.F., M.A., and Ph.D. from the University of Toronto in 1932, 1936, and 1938 respectively. He joined the then Division of Botany and Plant Pathology, Canadian Department of Agriculture, in 1935 and up to 1940 directed most of his attention to canker diseases of hardwoods and provided noteworthy contributions to a better understanding and control of Hypoxylon and Septoria of aspens in the prairie region. In 1940 he established the Forest Pathology Laboratory in Victoria, B. C., the first Regional Forest Pathology Laboratory in Canada, and up to 1947 developed the program at this centre to a high level of contribution. Beyond directing the program over a wide range of activities, he made noteworthy personal contributions in the fields of nursery diseases, root rot of Douglas-fir, and cull surveys. In 1947 he transferred to Ontario and opened another Regional Laboratory for the then Forest Biology Division of the Department of Agriculture, developing program and staff over a broad field of activity, including pathology, mycology, and physiology. Between 1951 and 1956 he served as Associate Chief of the Forest Biology Division in Ottawa, and provided leadership and guidance to the Forest Pathology program throughout Canada. In 1957 he accepted the chair in Forest Pathology with the Department of Biology and Botany, University of British Columbia. He held dual professorships in the Department of Botany and the Faculty of Forestry until 1965, at which time his discipline came to rest within the Faculty of Forestry.

Throughout his professional career, but particularly during the past ten years, Jack was instrumental in encouraging a number of competent undergraduates to develop careers in Forest Pathology and in guiding graduate and post-graduate students in their research programs. During this period he carried out a program of personally conducted research, latterly stressing tree physiology and microbiology in relation to forest disease control. He approached this field with the vigour and enthusiasm which marked his entire career and did much to illustrate the complex interactions between host, pathogens, and the naturally occurring microflora.

Dr. Bier was an active member of numerous regional, national, and international organizations, including the Canadian Institute of Forestry, Canadian Phytopathological Society, Michigan Academy of Sciences, Association of B.C. Registered Foresters, International Congress of Botany, and W.I.F.D.W.C. He was particularly active in the International Union of Forest Research Organizations, serving as chairman of two working groups and as a member of three others over a period of more than twenty years. His wide range of participation illustrated his broad training and interest as well as his stature in the scientific community.

Dr. Bier is survived by his wife and son, residing in Vancouver.

R. E. Foster

W.I.F.D.W.C.

OPENING REMARKS

Chairman Alex C. Molnar

Ladies and gentlemen, colleagues and honoured guests, I take pleasure in calling to order the 15th Annual Meeting of the Western International Forest Disease Work Conference.

Most of you are aware that a highly valued colleague, friend, and charter member of this Conference, Dr. Jack Bier, passed away in January. Jack continued to expound the principles of forest pathology to the end--and he continued his research with enthusiasm. He was at once a sage old teacher and a keen student. We shall miss him, but he left us a fine legacy of ideas--we could not ask for more. I ask you to join me for a moment's silence in his memory.

This fifteenth meeting of our Conference is dedicated to honour the memory of Dr. Paul Keener, a staunch supporter of our group for many years, whose untimely passing saddened us last year. He often urged us to meet in his beloved Southwest and I am sure he would be pleased he got us here without a Chamber of Commerce sales pitch. We are sorry that Betty Keener could not be with us today but you may be assured that her thoughts are with us. Paul's old student and long time friend, Dr. Paul Lightle, will make the dedication speech following the Welcoming Addresses.

On behalf of the Old Hands, I welcome into our midst new members, some of whom perhaps only recently embarked on a career in forest pathology. I highly recommend that they use this organization as an important touchstone throughout their careers. We do not have a complicated initiation ceremony; all we ask is that you pitch right in and participate vigorously in all phases of our Proceedings.

For some time I have been looking forward to the pleasure of welcoming our colleagues from Mexico and Guatemala. Apparently, I am to be denied this pleasure. I am sure you join with me when I say that we are all truly sorry they were not able to join us. Perhaps some future chairman will have the pleasure of welcoming them.

As your Chairman, my job has been to get the wheels rolling; having done so early in the year, the main burden of hammering out the meeting fell on your Local Arrangements Chairman, Paul Lightle; Program Chairman, Larry Weir; and your Secretary-Treasurer, Ed Wicker; whose blood, sweat, and tears will go on for some time. I wish to extend my grateful thanks to these gentlemen and their hardworking committees. I believe your program, which takes advantage of the locale to familiarize you with forest disease problems of the Southwest and Mexico, will also provide stimulus for thoughtful and vigorous discussion on three important topics: Silvicultural Control of Forest Disease, the Team Approach to the Solution of Forest Disease Problems and Host Response to Injury by Pathogens. We are pleased with our choice of assembled panelists who will provide the framework for discussion. It will be up to you to flesh out the framework.

I would like now to introduce Lloyd Hayes, Chief of the Division of Timber Management and Forest Protection Research of the Rocky Mountain Forest and Range Experiment Station, who will deliver the Welcoming Address.

WELCOME ADDRESS

G. Lloyd Hayes

Director Raymond Price of the Rocky Mountain Forest and Range Experiment Station regrets his inability to meet with you. He has asked that I convey his greetings and welcome you to the Station's territory.

You are meeting in one of America's oldest and most delightful cities here in the Land of Enchantment. I'm sure the next speaker will tell you more about that.

New Mexico and Arizona together make up a well-defined forest region. Most of the major highways through these two states traverse the lowlands where travelers do not see many forests. But forests there are. Forests cover 39 million acres of the two states, which is 26 percent of the land area. It is true that many of these forests do not produce commercial wood products under present conditions. But the nearly 10 million acres of commercial forest land support 60 billion board feet of sawtimber alone. These commercial forests have an estimated potential productivity under a reasonably attainable level of management of 1.5 billion board feet of sawtimber annually, plus a good many million cubic feet of sub-saw log sized timber products.

According to our best calculations, diseases kill 134 million board feet of timber annually in the two states. Cull, degrade, and growth reduction caused by diseases probably exceed mortality. Most damaging of Southwest forest diseases are those caused by the mistletoes. Yes, I know that mistletoes, and especially dwarf mistletoes, are found throughout the western United States. But not in the numbers and taxonomic varieties that are found here in the Southwest. This must be close to a place of genetic origin or of major differentiation. Every species of woody plant seems to host at least one species of mistletoe. And like the dog whose fleas had fleas, even the mistletoes have mistletoes. I believe it true that all commercial conifers harbor dwarf mistletoes. These are responsible for the greatest proportion of our timber losses to diseases.

For every acre of commercial forest in the Southwest we have three acres classified as noncommercial. That does not mean they are unimportant acres. As you start your field trip tomorrow, please notice the beautiful residential areas in northwest Santa Fe which are built amongst the pinyons and junipers.

This woodland environment makes a very attractive setting for many beautiful (and expensive) homes. Notice also at Bandelier National Monument the importance of the woodland type.

Here in the Southwest, as in many other parts of the nation, values of forests for purposes other than timber production are snowballing. To what extent do these values justify an intensification of protection from diseases and other destructive agents? We are becoming involved more and more in the protection of trees in national parks and monuments, trees in suburban areas like northwest Santa Fe, trees in heavily used camp and picnic grounds, and trees in roadside, stream- and lakeside environments. In these places, tree values may be higher than we realize. They may justify an intensity of protection greater than we realize.

I understand that some of the members of the Rocky Mountain Station's disease research staff have had a hand in the arrangements for this meeting and for the tour that is to follow. If you find that they have made good arrangements, please tell the fellows; Paul Lightle, Jerry Riffle, Frank Hawksworth, and Stu Andrews. If you find that they have let you down in one or more respects, please tell me about it. I have a hand in their annual performance ratings. Maybe I can hold that as a club over their heads to see that they do right by you.

The second Welcome Address, given by Roy Bond is not available.

PANEL I. -- FOREST DISEASES OF SOUTHWESTERN UNITED STATES AND MEXICO

Stuart R. Andrews, Moderator

MAJOR DISEASE PROBLEMS AND STATUS OF DISEASE RESEARCH IN THE SOUTHWESTERN UNITED STATES

Paul C. Lightle

Forest disease problems in southwestern United States are many and varied. This afternoon we hope to acquaint you with some of our major problems and what we are doing about them. We regret that time will permit us to cover only the major ones on which we are putting considerable effort. These problems fall into the following general categories: 1. Heart Rots; 2. Foliage diseases; 3. Rusts; 4. Canker diseases; 5. Mistletoes, particularly the dwarf mistletoes; and 6. Nematodes and mycorrhizae. I will cover each of these in a general way and then the panel members will consider each in detail, giving you a close look at their research and research accomplishments.

Practically all major western coniferous forest tree species are represented here in the Southwest. If we consider southern Colorado as part of this geographical area, we can even include lodgepole pine as one of our timber species. I don't believe we have any larch, but we do have substantial volumes of Douglas-fir, true fir, spruce, and aspen. However, our main commercial species is ponderosa pine. Ponderosa accounts for approximately 80 percent of the timber cut in Forest Service Region 3.

With one exception, western red rot, caused by *Polyporus anceps*, is the major cause of rot cull in the ponderosa pine forests of the Southwest. I am sure that most of you know that Stu Andrews has studied this heart rot for many years. He estimates that 15 to 25 percent of the gross sawtimber volume of virgin stands is destroyed by this fungus. We are continuing to amass data that substantiate this estimate. He and I have completed a manuscript, which we hope will soon see daylight, that indicates the virgin stands of ponderosa on the Navajo Reservation have about 15 percent of the gross volume already lost to red rot. Stuie is continuing his research on red rot, but, for the time being, I'm out of it.

The one area I mentioned where red rot is not the primary heart rot of ponderosa pine is the Sacramento Mountains in southern New Mexico. In this area red heart, caused by *Fomes pini*, is more serious than red rot. Just how much more serious we don't know, nor do we know the reason. It is interesting to note that *F. pini* usually requires a lower temperature for maximum growth and development than does *Polyporus anceps*, and one would expect, on the basis of temperature, that *P. anceps* would do better than *F. pini* in southern New Mexico.

Until now, we have had very little data on the organisms that cause cull in our stands of mixed conifers. Last July I started a study, in cooperation with Timber Management of Region 3, that should shed some light on this. The Region is destructively sampling some of their inventory plots. These represent about a 0.01 percent sample of the acres of commercial forest in Arizona and New Mexico. The Region is taking all the measurements and collecting samples from the rot columns they find. I am culturing the rot and isolating the organisms. We developed our methods on the Lincoln National Forest in southern New Mexico this year, and the next few years should provide a lot of data on the damage caused by the various organisms.

Although red rot is the primary cause of defect, the major cause of mortality and growth loss is dwarf mistletoe. Andrews and Daniels estimate that this parasite is present on at least 2.5 million acres of ponderosa pine and an additional 275,000 acres of Douglas fir are infected in Arizona and New Mexico. They further estimate that annual mortality losses alone total 55 to 75 million board feet in the ponderosa pine of this Region with an additional 20 to 27 million board feet in Douglas fir. All coniferous species are attacked by either mistletoe or dwarf mistletoe. Our Arizona cypress, juniper, and even white fir are susceptible to Phorodendron. Pinyon, ponderosa, lodgepole, Douglas fir, true fir, spruce, and all minor species of hard and soft pines are ravaged by Arceuthobium. Thus, we probably have more mistletoe species and forms on more tree species than any other area in the United States.

The magnitude of the dwarf mistletoe problem in this Region was recognized soon after the turn of this century and research of sorts began as early as 1910. It has continued ever since, first by C. F. Korstian and then by W. H. Long, Ira Hatfield, Lake Gill, and now by Frank Hawksworth. At the present time basic research on the dwarf mistletoes is Frank's responsibility. However, due to the importance of the parasite in Arizona and New Mexico, and because long term experiments on control have been established here since 1933, investigations on silvicultural control are conducted here. Close liaison is maintained between the two projects and practically no overlapping in effort exists. Frank will tell you about his work later this afternoon.

Interim results from the control plots established by Hatfield in 1933, augmented by other findings from experiments in the Fort Valley Experimental Forest, suggested certain practical aspects of control that have served as guides in several administrative operations. The first was a control project on the South Rim of Grand Canyon which began in 1950. This was what I call a noncommercial operation because the idea was to save as many trees as possible by pruning. Pruning, I'm sure you realize, is an expensive dwarf mistletoe control method, and is impractical where timber values alone are involved.

Soon after completion of initial control at the Grand Canyon in 1952 a similar large-scale operation began on 13,000 gross acres on the Mescalero Apache Indian Reservation. This was what I term a commercial operation because all infected overstory trees of merchantable size were harvested. Infected understory and submerchantable trees were felled and left in place because, at that time, there was no market for pulpwood or other small diameter wood. Very few trees were pruned. Pruning was done only on trees with infections more than 18 inches from the bole and then only when the infected branches could be reached with an axe by a man standing on the ground.

The Grand Canyon control area was expanded in 1955. The original control area was retreated in 1955, and both portions were retreated in 1961 and 1967. The Mescalero control area has been retreated once, during the period 1962-1965.

In 1965, following publication of Quick's results on chemical control of dwarf mistletoe, the National Park Service's Southwest Region became interested in testing for chemical control. In cooperation with them we established a series of tests at Bandelier National Monument. These tests have been re-examined once--a year ago--and you will see them tomorrow.

Although it may seem odd, foliage diseases of conifers have been "bugging" us here in the Southwest for a long time, and we seem to have more than our share of them. During the late 1930's Rhabdocline pseudotsugae was prevalent in certain areas of the Region and Ellis and Gill reported on it. This disease, here, as elsewhere, is cyclic and periodic local flare-ups cause National Forest Administration considerable concern. During the same period in the 1930's, Ellis reported that the needle cast of ponderosa pine, caused by Elytroderma deformans, was endemic here. However, there has been no indication that it is increasing to the point of causing serious losses in increment or mortality as it has done in the Pacific Northwest. It has, however, been mildly epidemic at times in certain National Park areas on pinyon pine.

A serious needle cast problem has existed on the Prescott and Coconino National Forests for more than 15 years. In 1957 a grant-in-aid was made to Dr. Paul D. Keener for a taxonomic study of the principal fungi associated with this problem. Dr. Keener continued his study until death in 1966. His notes and drawings indicate that at least three fungi, besides Elytroderma deformans, are commonly associated with the needle cast, namely, (1) Hypodermella medusa, (2) H. concolor, and (3) H. cerina. Of the three, H. cerina seems to be the cause of the most substantial proportion of the needle cast. Elsewhere in the Region, the needle casts have been apparent nearly everywhere, but are transient in nature and seldom persist for more than a year or two in any particular location.

Other foliage diseases occur on white fir and at least one rust periodically defoliates aspen.

John Staley has been investigating the foliage diseases of conifers in the Southwest for several years, primarily from a taxonomic viewpoint but also from the pathological point of view. He will tell you of his work a little later this afternoon.

We are just starting a research program in Albuquerque on the needle cast diseases. The first effort will be on damage appraisal but along with that we will study pathogenicity, life histories, and developmental morphology, hoping eventually to reach the point where we can apply control measures. There are a number of problems to be solved before this work can really "get off the ground." If any of you have suggestions on how to harvest enough spores for inoculation work, or, of methods of inoculation, I will be pleased to hear your ideas either during the discussion period or at any other time.

Canker diseases in the Southwest have, until recently, received very little attention. The most serious canker disease of conifers is caused by Atropellis piniphila which attacks ponderosa pine in southern Arizona and New Mexico. This is particularly interesting because Atropellis has has not been reported in the area between North Dakota and southern New Mexico and Arizona. Other canker-causing organisms have not been identified. By far the most serious losses from canker diseases occur in the hardwood stands with aspen as the chief host. In Colorado aspen stands cover 23 percent of the commercial National Forest lands, but they are only minor components of the forests in New Mexico and Arizona. However, with the recent increases in recreational interests these aspen stands have become very important. Cankers of cottonwood and willow are also important in Arizona and New Mexico because these species frequently provide the only shade and protection from a blazing sun.

Tommy Hinds has been studying aspen cankers for several years and he will tell you about his work at the appropriate time a little later. The Albuquerque laboratory had planned to start investigating willow and cottonwood diseases this year from the standpoint of bark moisture, as propounded by Jack Bier, but we are just spread too thin to work on the problem at this time.

Although there are a number of rusts that are serious pathogens of southwestern conifers, exceedingly little study effort has been expended on them. Probably the most serious rust in New Mexico, Arizona, and parts of Colorado is the so-called "limb rust" caused by Peridermium filamentosum. Peterson has recently distinguished races of this fungus and explained some of the apparent discrepancies that have appeared in the literature. Yellow witches broom of spruce and fir is also serious in certain areas in the region. While western gall rust occurs throughout the area, it is serious only on lodgepole pine. It is found so infrequently on ponderosa pine in Arizona and New Mexico that we know where almost every gall is located. One of them is close to where we will be tomorrow at Bandelier National Monument.

Roger Peterson has done a great deal to straighten out the confused rust picture here and elsewhere, and he will recount his work for you before the afternoon is over.

While the dry weather and fires in the Pacific Northwest, Canada, and the Inland Empire make headlines in the daily news, no one cares that the Southwest has frequent droughts. It's expected of us, and except for local news items, never reaches the daily papers. We do have frequent droughts, some of them extending over rather lengthy periods of time. The last extended dry spell was during the mid-1950's. Little mortality or other damage was noted during the period when moisture was deficient, but, on the return to normal rain, trees died by the thousands. Ponderosa, pinyon, and juniper were the primary species subject to mortality. Ponderosa and pinyon were killed outright. Juniper, however, frequently was only partly killed. Entomologists and pathologists were called in by Forest Administration and the National Park Service to investigate the mortality. Although a large proportion of the trees were attacked by bark beetles, a substantial part had no insect attack at all or were so lightly attacked that insect responsibility was questionable. Stu Andrews and Wally Eslyn were the investigating pathologists, and they reported that they could find no pathological organisms involved. They noted, however, an apparent dirth of mycorrhizae and some evidence of pathogenic nematode activity. For these reasons, we started a study of nematodes and mycorrhizae in the forest soils of the Southwest. So far, the work has been confined to the areas adjacent to Albuquerque, but eventually it will include the entire Region. Jerry Riffle will report on the nematode and mycorrhizae work that he's been involved in as the last member of this panel to speak.

Pinyon and juniper, particularly the latter, are considered weed species by the cattlemen, sheepmen, and range specialists in this area. However, literally thousands of people have purchased pinyon-juniper land for home sites, frequently at high per-acre costs. These people are quite vociferous and really "holler" when a tree dies. We hope that our nematode and mycorrhizae studies will eventually allow us to make recommendations to the range people on how to capitalize on the drought to rid the range of unwanted juniper and pinyon and also to tell the home owners what to do to save their ornamental trees.

These, then, are the major forest disease problems of the Southwest: (1) Heart rots of all conifers, (2) Dwarf mistletoes of all coniferous species, (3) Foliage diseases of conifers, (4) Rusts of conifers, (5) Cankers of both hardwoods and conifers, and (6) Nematodes and mycorrhizae and their effects on marginal ponderosa pine and its woodland associates. in relation to drought. I am sure that each speaker will point out, in connection with his work, the important areas that urgently require attention, but which must await a time in the future when even more urgent problem areas have been worked on and satisfactory answers obtained.

FOLIAGE DISEASES OF CONIFERS IN THE SOUTHWESTERN UNITED STATES AND MEXICO

John M. Staley

To discuss the subject as titled, I will be forced to extrapolate largely from information obtained on diseases as they occur on the east slope of Colorado's Front Range. There is a dramatic drop in the breadth of my experience with distance from that central location. The basis for my remarks on diseases in Arizona rests on a 3-day collecting trip near Flagstaff in Paul Lightle's company, on the examination of parts of Dr. Keener's collections and on Stu Andrews' studies of foliage disease in Arizona. The basis of my remarks on Mexican foliage diseases is the examination of specimens collected by Frank Hawksworth and on his field observations.

Nevertheless, I don't believe that generalizing from Colorado to Arizona will lead to significant inaccuracy. In Colorado there are most of the disease organisms found in the Southwest. We don't have _Hypoderma mexicanum_. But we have areas that have been damaged each year for at least 5 years by _Hypodermella cerina_, _Lophodermium ponderosae_ and _H._

concolor. Similarly for H. medusa, Elytroderma deformans and 4 other needle cast fungi we have observed continued damage to numerous observation trees. I believe this provides a broad enough familiarity with the different organisms to allow accurate interpretation of field observations and herbarium data collected in the Southwest.

It is because of their ability to cause noticeable damage to forest stands year after year that needle casts have attracted the most attention among foliage fungi in Colorado and the Southwest.

Identification of needle casts has been a matter of debate. There are several reasons for this. The descriptions given of the various fungi in Dr. Darker's monograph appear to be quite complete and adequate. In practice however these descriptions are confusing to many pathologists. The way out of this confusion is clear. It is to examine the type or critical specimens and using this as a starting point relate the variations in type morphology to variations encountered in the field.

With several exceptions, I have found Dr. Darker's species concepts to hold up well. This is somewhat surprising when some of the species such as H. arcuata and H. cerina were established on the basis of only 3 collections. The species differ in certain, sometimes minor, morphological features. What is interesting is the correlated difference in symptom development and ecology.

Some pairs of species appear closely related such as H. arcuata and H. montivaga and H. concolor and H. cerina. These species pairs could represent host specific forms, I don't deny the possibility, yet I have more faith in the evidence that suggests they deserve species rank.

Let us consider H. cerina and H. concolor. The case is simply this, H. cerina has long narrow spores and H. concolor shorter wider spores. The type collection of H. cerina is on lodgepole pine. This seems to argue against a host effect on spore shape. In Colorado H. cerina is found in a rather unique area abnormally warmer and moist for Colorado, where H. cerina on ponderosa pine can be found side by side with H. concolor on lodgepole. There appears to be a distinct separation in spore form on the two hosts and no H. cerina can be found on the lodgepole (as we must assume it could be from evidence in the type). H. concolor is widely distributed on lodgepole in Colorado, especially in the northern portion. Although ponderosa pine frequently grows there, intermingled with infected lodgepole, no H. cerina infections are found. The evidence is not strong enough to justify combining the species. I believe it points, however weakly, toward the support of Darker's concept and I prefer the clarity of reference his nomenclature provides.

The case of H. arcuata and H. montivaga as a species pair is less definite. We have no report of reciprocal host attack here. This is not surprising since I know of no solid morphological difference between the species. There is a host susceptibility difference that suggests a barrier to reciprocal infection. Also, H. montivaga has consistently caused necrosis of current season needle tissue by late August whereas the first symptom of H. arcuata infection is apparent in May. Again we are faced with consistent differences even if only host differences in this second instance (and with Darker's nomenclature). I submit that no one can as a practical matter separate these two species on the basis of Darker's claimed differences in morphology. Yet they are clearly separated on symptom development. Is this merely due to a host effect? Until we have more evidence on what I believe is a matter of little moment, permit me to take the easy way out by again happily accepting Darker's nomenclature.

I choose to discuss these two pairs of fungi first because I believe that Darker's remaining species are much more readily and validly distinguished from one another.

There is, however, one further problem in identifying needle casts according to Darker. This is due to a confusion of lumping and omission in the Hypodermella ampla group. I believe that H. medusa is widely accepted as a repository for almost anything black on ponderosa pine that cannot be assigned to either Elytroderma or Lophodermium. Actually H. medusa can be separated into 3 and perhaps 4 species. In addition, a new organism Lophodermium ponderosae has probably masqueraded as H. medusa.

From the group of medusa-like organisms on ponderosa pine can be separated a long filiform spored fungus with a prominent imperfect state. Lophodermium ponderosae, and 4 other distinct forms. One of these is the typical form of H. medusa. It is associated with a clavate-pyriform imperfect state. Collections indicate that it ranges from lower California to Montana. Another form with narrower spores associated with a rod-shaped imperfect state appears to range from northern California to Montana. I have not found this form except in my personal collections and never except in mixed infection with the typical form of H. medusa. It is, nevertheless, distinct from the typical form. I choose to regard it as a morphological variant.

In the central Rockies, we have two other fungi that Darker placed in H. medusa. One attacks ponderosa pine but has no imperfect state associated with it. Also the insertion of the ascocarp is distinct from that of typical medusa.

The second organism attacks lodgepole pine. It also has no associated imperfect state. It frequently attacks whole thickets of young trees in contrast to the one on ponderosa that usually affects only isolated trees. The ascospore and ascocarp characteristics are quite different from the fungus on ponderosa.

The ponderosa organism, I have found from Arizona to northern Colorado. The lodgepole organism, I have found largely in Colorado and also twice in Montana.

With this brief discussion of the organisms found in Colorado and the Southwest, I would like to make some generalizations.

The three organisms that appear capable of sustained widespread damage to mature forest trees are Hypodermella concolor (on lodgepole pine) and on ponderosa pine Lophodermium ponderosae and Hypodermella cerina. The other organisms either attack isolated trees or are restricted to moist stream bottoms or some other favoring environment. It is of course possible that a buildup of one of these minor organisms might occur and persist but at present the three most damaging are those I just mentioned. The infrequent or sporadic organisms include the ponderosa form of H. medusa and E. deformans.

As I mentioned earlier, my collecting in the Southwest was limited to a period of three days guided by Paul Lightle. We saw more mature and identifiable needle cast fungi in those three days than I have seen in any comparably short period elsewhere.

There was no evidence of ecological differences in the Arizona stands that would modify the relative roles of the organisms we found there in comparison to Colorado conditions.

Our procedure was very simple. We stopped frequently both inside and outside of recognized needle cast infestations. Inside these areas of damage we stopped at sites of typical damage and one twig was clipped from a tree field identified as H. cerina. Three or four other twigs were then clipped from different trees to encompass the range of variation in symptom expression. Eighty-four trees were sampled in this conservative manner to sample specifically for variation in symptom pattern (and presumably in cause). Lab examination showed that 80 percent of the twigs were attacked primarily by H. cerina. We know that L. ponderosae, our Rocky Mountain form of H. medusa, E. deformans, and H. planum were also present but only in insignificant amounts.

In my mind, the evidence that H. cerina is the cause of the damage in Arizona rests not so much on the lab examination as it does that this very conservative sampling confirmed what was quite obvious in the field. That is, that the symptoms of the injury were symptoms of H. cerina--very distinct from those of other ponderosa needle casts. H. cerina invades a band of needle tissue several inches back from the tip. When the distal portion dies it is bright reddish brown. L. ponderosae, H. medusa, and E. deformans can all be distinguished from this by the color of the diseased needles, their position relative to the current foliage, and by the frequent presence of the fruit body. Mixed infections did occur but there was no evidence that their frequency was greater than 1-2 percent as is quite normal in Colorado. I have since examined Dr. Keener's collections and found that H. cerina was present each year in the affected areas from 1955-62. There was no evidence of a continuum in spore shape between the cerina and concolor types. Since lodgepole pine is absent from the affected areas, I found it necessary to go to Colorado collections as the nearest source of material for comparison. The only evidence I can see as a cause of confusion between the two species is the immaturity of some cerina collections. The collections in the Fort Collins herbarium are fully available to anyone who feels it desirable to further study the validity of the two species.

Seven of twenty of my field inoculations (vs 0 of 20 controls) indicated the pathogenicity of H. cerina, if you choose to accept naturally diseased foliage as suitable inoculum. I am trying to show that H. cerina will directly penetrate healthy foliage, but at present the field inoculations are the only proof of pathogenicity I have.

I have not said much about needle cast in Mexico. From Frank's collections there we know that Hypoderma mexicanum is able to attack several other species of pine than P. leiophylla, especially P. cooperi. It appears capable of widespread damage. E. deformans is also present in Mexico as are H. medusa and H. arcuata.

I might conclude by throwing out an enticement to needle cast collectors. There exists in the Cataline Mountains east of Tucson an unnamed Bifusella that has the gross characteristics of H. cerina and H. concolor. There is no other concolorous Bifusella. The fungus is very scantily represented in Dr. Keener's collection. Perhaps additional search will turn up a more abundant collection. It's out there awaiting its "discoverer."

CONIFER RUSTS OF THE SOUTHWEST AND MEXICO

Roger S. Peterson[1]

Spruce broom rust, fir broom rust, and western gall rust of pines occur in the Southwest and the latter two extend well into Mexico, but damage that they cause in these areas is local or slight. Another typically northern disease, comandra blister rust, causes spike-tops in ponderosa pine in scattered parts of the Southwest, but no known epidemics have originated in the last four or five decades in this area. Of more interest in the subject areas are limb rust of pine, caused by *Peridermium filamentosum*, and the Mexican cone and stem rust of pine caused by *Cronartium conigenum*.

The ~~cone rust is~~ distributed throughout the mountains of Mexico except in Baja California. It is also in southernmost Arizona and throughout highland pine forests of Central America. On oak, the telial host, the distribution is even wider. Although at least 16 species of hard pines are attacked in Mexico, including important Arizona species such as ponderosa pine, the rust can be found in the United States only on the unimportant Chihuahua pine and oaks. Trunk and branch galls caused by *C. conigenum* are lobate, often stalked, and are composed mainly of primary tissue similar to pith. Some or all of the seeds are destroyed in infected cones, which also provide favorable breeding grounds for cone moths. Although the rust is abundant on cones, the economic impact may be slight inasmuch as seed production is not now the limiting factor in pine reproduction in Mexico.

Limb rust attacks only pines of the Ponderosae group, including ponderosa, Apache, Montezuma, and (in California and Baja California) Jeffrey pines. Races of *Peridermium filamentosum* that apparently infect directly from pine to pine are known in Baja California and northern Arizona, and probably are in northern New Mexico. The heteroecious race that infects *Castilleja* is throughout the Southwest. The race or races in Mexico are unknown: a few available specimens suggest great differences from forms studied in the United States. No large outbreaks of limb rust are known to have occurred in recent years, but infected trees from earlier epidemics survive on all national forests of the Southwest. Such trees serve as the most important home base for endemic *Dendroctonus* populations, if a large sample studied in Bryce Canyon National Park is representative.

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[1]These data are from the author's former employer, the Intermountain Forest and Range Experiment Station. Cone rust data were published more fully in Spanish by Peterson and Salinas (Inst. Nac. Invest. For., Mex., Bol. Tec. 19).

Life cycles of members of the Peridermium filamentosum complex are under study at the Native Rust Project in Logan. The Intermountain Station also plans to renew its cooperative studies with the Mexican government on P. filamentosum and C. conigenum in Mexico.

ASPEN CANKERS

Thomas E. Hinds

Quaking aspen (Populus tremuloides Michx.) is one of the most widely distributed and disease-prone native forest trees in North America. Commercial stands of aspen in Colorado alone cover over 2.5 million acres, or about 23 percent of the commercial forest land in the state. The majority of these stands are found west of the Continental Divide from the northern to the southern border of the state. Commercial stands in Arizona and New Mexico cover nearly half a million acres. In this Southwest region, aspen is plagued by a variety of canker diseases and the aggregate damage they cause is probably exceeded only by losses from heart rot.

The principal uses of aspen are for match-splint veneer, core stock for doors, excelsior, lumber, and dimension stock. Actual output of aspen saw logs varies from 1 to 8 million board feet annually in Colorado. Although many aspen stands do not appear sufficiently productive to justify timber management investments, they are valuable for other uses. The increasing demand for recreational areas has brought about many new camping sites in our aspen stands. The diseases of aspen which have been tolerated in wild stands will soon have to be dealt with in these new high-value recreational and scenic areas.

A limited survey on the distribution of aspen cankers in Colorado in 1960 revealed that cankers were generally distributed throughout the major stands and were more prevalent than previously suspected (6). The cause and pathological significance of two common cankers are already well known. Hypoxylon canker caused by Hypoxylon pruinatum (Klofzsch) cke. was first found in Colorado in 1956 (2). It is distributed throughout most of the aspen type but does not cause as much damage as in the Lake States. The true status of this serious disease remains questionable in Colorado. Cytospora canker caused by Cytospora chrysosperma Pers. ex. Fr., a weak parasite of aspen throughout its range, apparently has always been endemic in Colorado. This disease occurs in the form of cankers which may be fairly regular or very irregular in outline, and more frequently extensive without forming a definite canker on moribund bark. The fungus is often associated with other cankers and tree wounds.

Cenangium canker, caused by Cenangium singulare (Rehm.) Davidson and Cash, is a fairly recent Colorado discovery (1) and is considered the most damaging to aspen stands. It is capable of girdling and killing trees within 2 to 4 years although some infections become arrested before they completely girdle the trunk (5). This canker often becomes established in older stands that have been subjected to mechanical injury from road building, logging,or campground development. Once established in the stand, infections tend to build up rapidly, even on unwounded trees. Cankers are characterized by their elliptical shape which may reach a length of 10-15 feet before girdling a tree. Infected bark turns black over the cankered area and remains tightly attached to the wood for several years, even after the tree dies and falls to the ground. Apothecia develop abundantly on the old dead bark or underneath the loosened white surface layer.

Black canker and a Nectria-like canker have been recognized for many years (10, 8). They were distinguished from each other on a morphological basis even though their cause and significance were unknown. We are presently working on the etiology of these two cankers. Black canker distorts the tree trunk, enlarges with each season's growth, coalesces, and often attains a length of 10 to 20 feet in a period of 50 to 70 years without girdling the tree. Flaring dead bark around the perimeter of the canker with a blackening of the exposed canker face is typical. On the other hand, the Nectria-like cankers are more oval or elliptical "target-shaped" cankers with successive rounded annual ridges of callus tissue. Dead bark covering the callus folds often remains attached to the wood of small cankers. Investigations of these two cankers reveals that Ceratocystis fimbriata (Ell. & Halst.) Davidson is associated with both. The morphological differences are believed to be due to canker age and host response. C. fimbriata is easily isolated and perithecia of the fungus are usually found along the active portions of the cankers.

Ceratocystis cankers frequently start at fresh trunk wounds. At first a new wound appears to be healing but after several years the typical Nectria-like canker forms. Removal of the callus tissue formed around a young canker reveals the necrotic bark and extent of necrosis of the last year's growth. Stain in the wood beneath a young canker often extends several feet in the trunk and halfway into the bole. Old cankers stemming from fire and other old basal wounds usually have the appearance of the black canker type.

The presence of a dead branch stub in the center of a canker is also common. Zalasky (11) has shown that infection in unwounded stems can take place through leaf infections and that for direct penetration, C. fimbriata must be in intimate contact with living cells. This accounts for its failure to penetrate dead epidermis and cork cells in stems which have developed a periderm or a wound periderm.

Manion and French (9) recently published their results of inoculations made on Minnesota aspen with C. fimbriata. Because of the small canker-like development after 1 to 4 growing seasons, they concluded that the full potential of C. fimbriata for causing perennial cankers on aspen is still unknown. Our work on the pathogenicity of C. fimbriata consists of over 200 inoculations which are now four years old. While canker development from these inoculations has been slow, there is no doubt in my mind that the fungus is capable of entering through trunk wounds and forming cankers. We will make the final observations on these inoculations next summer.

Other new species of Ceratocystis have been isolated from typical cankers and are being tested for their canker producing capabilities. Early observations on inoculations with C. tremuloaurea Davidson and Hinds (3) indicate that it may be incapable of establishing a canker whereas C. populina Hinds and Davidson (7) may be an aggressive canker forming organism. C. crassivaginata Griffin (4), isolated from aspen in Ontario and Colorado, is also being tested.

Literature Cited

1. Davidson, R. W., and Edith K. Cash. 1956. A Cenangium associated with sooty-bark canker of aspen. Phytopathology 46: 34-36.

2. Davidson, R. W., and T. E. Hinds. 1956. Hypoxylon canker of aspen in Colorado. Plant Disease Reptr. 40: 157-158.

3. Davidson, R.W., T.E. Hinds, and E.R. Toole. 1964. Two new species of Ceratocystis from hardwoods. Mycologia 56: 793-798.

4. Griffin, H. D. In press. The genus Ceratocystis in Ontario. Can. J. Bot.

5. Hinds, T. E. 1962. Inoculations with the sooty-bark canker fungus on aspen. Plant Disease Reptr. 46: 57-58.

6. ____________. 1964. Distribution of aspen cankers in Colorado. Plant Disease Reptr. 48: 610-614.

7. Hinds, T. E., and R. W. Davidson. 1967. A new species of Ceratocystis on aspen. Mycologia. (in press)

8. Long, W. H. 1918. An undescribed canker of poplars and willows caused by Cytospora chrysosperma. J. Agr. Res. 13: 331-343.

9. Manion, P. D., and D. W. French. 1967. Nectria galligena and Ceratocystis fimbriata cankers of aspen in Minnesota. Forest Sci. 13: 23-28.

10. Meinecke, E. P. 1929. Quaking aspen. A study in applied forest pathology. U.S. Dept. Agr. Tech. Bull. 155. 33 p.

11. Zalasky, H. 1965. Process of Ceratocystis fimbriata infection in aspen. Can. J. Bot. 43: 1157-1162.

MISTLETOES OF THE SOUTHWEST AND MEXICO

Frank G. Hawksworth

Tommy Hinds has discussed aspen cankers mainly as they occur in Colorado--so things should average out if I skip over the Southwest and talk about the mistletoes of the forests of Mexico.

For the past few years, Dr. Del Wiens, of the University of Utah, and I have been preparing a taxonomic revision of the genus Arceuthobium. As part of these investigations, we have been privileged to make several trips to Mexico. These trips have been made in cooperation with pathologists at the Instituto Nacional de Investigaciones Forestales at Mexico City. Duplicates of collections are sent to this institute and also to the National Herbarium at the Instituto de Biologia at Mexico City.

Little research on the mistletoes is being conducted in Mexico although Rudolfo Salinas, of the Instituto Nacional de Investigaciones Forestales is doing some work on the dwarf mistletoes that occur near Mexico City. Riba (1963) recently published some general notes on the mistletoe family in Mexico. Roldan (1924) first called attention to the damage caused by dwarf mistletoes in Mexico and Sosa (1939) discussed the seriousness of A. vaginatum in one area in the state of Tlaxcala. Valdivia (1964) reported on the hosts and abundance of A. globosum in the State of Michoacan. De la Puente (1966) recorded observations on natural resistance of some species of pines in central Mexico.

To give an unbiased opinion, I'd say that mistletoes seem to be the number 1 forest disease problem in Mexico. But we should bear in mind that other factors besides diseases are considered more damaging to forest production in Mexico. For example, Garduno and Sierra (1962) in the proceedings of the Seattle World Forestry Congress list the following causes of forest destruction in Mexico in this order:

1. Fire
2. Clearing of forest land for agricultural purposes
3. Uncontrolled grazing
4. Insects and diseases
5. Irrational utilization

The most damaging Mexican mistletoes on conifers are Arceuthobium, but in addition there is:

1. Psittacanthus on pines and Abies--very common in central Mexico.

2. Phoradendron velutinum on pines--infrequent.

3. Phoradendron bolleanum subsp. pauciflorum--common in Baja California on white fir.

4. Phoradendron juniperinum subsp. libocedri--common in Baja California on Incense Cedar.

Arceuthobium is highly developed in Mexico; and at least 14 dwarf mistletoes occur there, eight of which are endemic. The dwarf mistletoe epicenter of the universe seems to be in the Sierra Madre Occidental in the state of Durango where four species that are known from nowhere else are to be found (Hawksworth and Wiens 1965).

Eight dwarf mistletoes occur here in the Southwest, including five that also occur in Mexico--A. vaginatum subsp. cryptopodum, A. douglasii, A. gillii, and A. campylopodum formae blumeri and divaricatum. Also the coastal ponderosa pine species--(A. campylopodum f. campylopodum) occurs in Baja California on Jeffrey and Coulter pines (Hawksworth, Lightle, and Scharpf--in press). It is possible that the sugar pine mistletoe may also show up in Baja California because it has recently been found in San Diego County, California, only about 25 miles north of the International Boundary.

Most of the Mexican dwarf mistletoes (12 species) attack pines but one occurs on Pseudotsuga menziesii (A. douglasii) and one on Abies spp.

(*A. abietis-religiosae*). At least 24 pines are known to be attacked in Mexico (Table I) and further study will undoubtedly extend the host list considerably. One tree, *Pinus engelmannii* is attacked by 6 dwarf mistletoes.

Table 1. Pines attacked by dwarf mistletoes in Mexico (Hawksworth and Wiens 1965), Hawksworth, Lightle, and Scharpf in press, Valdivia 1964).

Dwarf Mistletoe	Pine Hosts
1. *A. verticilliflorum*	*cooperi*, *engelmannii*
2. *A. gillii* subsp. *gillii*	*leiophylla*, *chihuahuana*, *lumholtzii*, *ponderosa*, *arizonica*
3. *A. gillii* subsp. *nigrum*	*leiophylla*, *chihuahuana* *lumholtzii*, *teocote*, *montezumae*
4. *A. globosum*	*lawsonii*, *tenuifolia*, *pseudotstrobus*, *montezumae*, *hartwegii*, *rudis*, *cooperi*, *michoacana*, *arizonica*, *durangensis*, *engelmannii*, *pringlei*, *douglasiana*
5. *A. vaginatum* subsp. *vaginatum*	*montezumae*, *hartwegii*, *rudis*, *cooperi*, *michoacana*, *ponderosa*, *arizonica*, *engelmannii*, *culminicola*
6. *A. vaginatum* subsp. *cryptopodum*	*ponderosa*, *arizonica*, *arizonica* var. *stormiae*, *engelmannii*
7. *A. vaginatum* subsp. *durangense*	*montezumae*, *durangensis*
8. *A. rubrum*	*teocote*, *cooperi*, *durangensis*, *engelmannii*
9. *A. strictum*	*chihuahuana*, *teocote*, *engelmannii*
10. *A. campylopodum* f. *campylopodum*	*jeffreyi*, *coulteri*
11. *A. campylopodum* f. *blumeri*	*strobiformis*
12. *A. campylopodum* f. *divaricatum*	*quadrifolia*

Some Mexican dwarf mistletoes are unique, and even bizarre. For example, one species (*A. globosum*) does not form witches' brooms. It has a broad host range that includes at least 15 species of pines. Another species (*A. verticilliflorum*) has deciduous staminate shoots but perennial pistillate shoots. Also this species has fruits up to nearly 1.5 cm long (the *seeds* of this species are larger than the *fruits* of any of our species, thus hard hats are in order for work in these forests during the firing period). Another species (A. strictum) is unique in that the staminate shoots are unbranched. Some of the "giant" dwarf mistletoes (A. globosum and A. vaginatum subsp. vaginatum and durangense) have shoots up to two feet high and nearly two inches in diameter.

Although the dwarf mistletoes seem to be the primary forest disease problem in Mexico, the amount of damage caused by them appears to be much less than in our Southwestern pine forests. The main form of loss is in growth reduction although we did observe mistletoe-caused mortality in many parts of the Sierra Madre Occidental in the States of Chihuahua and Durango. In general, damage seems to decrease (as growing conditions for for pines improve) as one proceeds southward toward Mexico City. Perhaps the lesser damage in Mexican pine forests is due to the generally better moisture conditions (as compared to our southwestern pine forests). Also in many Mexican pine stands, fires are more frequent than in the Southwest and this has tended to inhibit the development of dense understory stands that characterize many of our southwestern pine forests.

In spite of these differences, the Mexican pine forests, particularly the northern ones, are generally comparable to those of the Southwest and much of what we learn about control of dwarf mistletoe here will also be applicable in Mexico.

Literature Cited

Garduno Garcia, Roberto and Antonio Sierra Pineda. 1962. Causa de la Destrucción Forestal en Mexico. Fifth World For. Cong. 2: 901-904.

Hawksworth, F. G., and D. Wiens. 1965. Arceuthobium in Mexico. Brittonia 17: 213-238.

Hawksworth, F. G., P. C. Lightle, and R. F. Scharpf. 1965. Arceuthobium in Baja California, Mexico (accepted for publication in Southwestern Naturalist).

de la Puente, Jose M. 1966. Forest Disease and Insect Resistance in Latin America. In Breeding Pest Resistant Trees, H. D. Gerhold et.al., Eds., Pergamon Press, pp. 31-34.

Riba, Ramon. 1963. Notas sobre la familia Loranthaceae y el parasitismo segundario. Biol. Soc. Bot. Mex. 28: 1-10.

Roldan, A. 1924. Los árboles indigenos que ataca el muérdago en el Valle de Mexico. Mexico Forestal 2: 61-63.

Sosa, A. H. 1939. Informe de inspección sobre los montes de la finca de San Diego Pinar, Municipio de Citlatepetl, Distrito de Juarez, Estado de Tlaxcala. Bol. Dept. Forest. y Caza y Pesca 4(15): 123-156.

Valdivia Sanchez, J. de J. 1964. [Forest Pathology 1. Dwarf mistletoe (Arceuthobium sp.) in N. E. Michoacan]. Bol. Com. For. Michoacan 15, 67 p. [For. Abstr. 26, #3854, 1965].

INVESTIGATIONS ON NEMATODES AND MYCORRHIZAE OF MARGINAL PONDEROSA PINE AND ASSOCIATED SPECIES IN CENTRAL NEW MEXICO

Jerry W. Riffle

Substantial premature mortality of marginal (low elevation) ponderosa pine and its woodland associates, pinyon and juniper, has followed frequent periods of prolonged drought in New Mexico and Arizona. Investigations by pathologists during the periods of severe damage failed to disclose any pathogens on the roots or aerial parts of the trees that might cause mortality. Exploratory research indicated, however, that root staining and decay fungi, a deficiency of mycorrhizae, and plant-parasitic nematodes might have weakened the trees. Investigations on nematodes and mycorrhizae of marginal ponderosa pine and associated species began in 1963.

Nematological Studies

From 1963 through 1966, the taxonomy, geographical distribution, frequency, and density of nematodes in non-drought and drought-affected ponderosa pine stands were investigated. Twenty-four species representing 22 genera of free-living and predaceous nematodes, and 30 plant-parasitic species representing 14 genera were found associated with ponderosa pine, pinyon, and three species of juniper commonly found in central New Mexico (*Juniperus monosperma*, *J. deppeana*, and *J. scopulorum*). Four undescribed plant-parasitic species were found and two of them have been described (9). Another undescribed nematode has been shown to feed on and reduce, under laboratory conditions, the linear growth and viability of *Suillus granulatus*, a common southwestern mycorrhizal fungus (10). This nematode, as well as *Xiphinema americanum* and *Tylenchus exiguus*, was found in seven widely separated marginal ponderosa pine stands that were sampled during the investigation. Overall, 23 species were found in drought stands compared to 26 in non-drought stands.

In addition to its widespread distribution, *Tylenchus exiguus* was the most frequently encountered nematode. Regardless of tree species, this nematode was found in at least 96% of 804 samples examined. Species of *Xiphinema* and *Paratylenchus* were found most frequently around roots of juniper. Species of *Aphelenchoides* and *Criconemoides* occurred with about equal frequency on both juniper and pine. Species of *Tylenchorhynchus*, *Hemicycliophora*, *Trichodorus*, and *Longidorus* were found only in a very small percent of the samples examined. *Tylenchorhynchus cylindricus* and *Hemicycliophora* spp. were found on both pine species, whereas *Trichodorus* sp. and *Longidorus elongatus* were found only on ponderosa pine.

Large and consistent differences in the density of most plant-parasitic nematodes in non-drought and drought-affected stands were not apparent. Perhaps one reason for the small differences is that there has not been any serious drought in even the drought-affected areas since the 1950's when most tree mortality occurred. If there were any large differences in the density of nematodes on specific tree species in drought and non-drought stands during or immediately after the drought, they have diminished and are no longer apparent.

The effect of plant-parasitic nematodes on the growth of southwestern tree species is unknown. However, feeding of nematodes on tree roots over a long period of time, especially on marginal trees growing at the lower limit of their normal altitudinal range, may weaken the trees and make them more vulnerable to adverse environmental conditions, diseases, and insects. Studies will soon be undertaken to determine which nematodes are parasitic on ponderosa pine and its woodland associates. Species of most of the nematode genera found in central New Mexico have been reported parasitic on various pine species or other woody plants. For example, Tylenchus emarginatus has been reported to feed and reproduce on the roots of 4 spruce and 3 pine species (14), and Criconemoides xenoplax, Hemicycliophora vidua, Helicotylenchus dihystera, Tylenchorhynchus claytoni, and Trichodorus christiei parasitize and reproduce on at least 5 pine species (13). In addition, Hoplolaimus tylenchiformis caused severe damage to lateral and mycorrhizal short roots of slash and loblolly pine (12).

A Meloidogyne species has been found parasitizing mycorrhizal rootlets of ponderosa pine in southwestern New Mexico (11). The infected rootlets of the declining trees displayed considerable hypertrophy, and internally typical giant cells were formed.

A study is now underway to determine seasonal population dynamics of various plant-parasitic nematode species commonly found in marginal ponderosa pine stands. Very little is known about life cycles and populations of nematodes in natural forest stands. Of particular interest is the influence of the environment on nematode populations, especially the effects of high soil temperature and low soil moisture over extended periods of time. High populations of plant-parasitic nematodes have been found from June through August when soil temperature and soil moisture are favorable for active root growth and nematode reproduction. When information is available on nematode populations for non-drought years, it may be possible to distinguish any marked changes that occur in nematode populations during or immediately after future droughts and thereby obtain some indication of the importance of nematodes in the decline of vigor of southwestern tree species.

Mycorrhizal Studies

Mycorrhizae are known to benefit the growth and survival of trees by improving the absorption of nutrients from the soil and by protecting the roots against root pathogens. Since there also is some evidence that mycorrhizae may increase the drought tolerance of pines (1), investigations were undertaken to determine which fungal species form mycorrhizae on marginal ponderosa pine and its associated species.

Cultures of suspect mycorrhizal fungi were obtained from sporophores or mycorrhizal rootlets. Sporophores were collected from marginal ponderosa pine stands or from mixed conifer stands and identified to species. Isolations were made by planting small pieces of tissue directly on Hagem agar or by suspending hymenial tissue over an agar surface and germinating the deposited spores. Over 30 identified fungi representing 17 genera of basidiomycetes have been isolated from sporophores.

In addition, isolations have been made directly from mycorrhizae to culture the fungal symbionts, but the identification of these symbionts remains a major problem. Our approach to this problem is to develop an extensive collection of cultures from identified sporophores and compare the cultural characteristics of the fungi grown in standard environments. Pantidou (8) has reported that some mycorrhizal fungi show sufficiently distinct combinations of characters in pure culture to facilitate eventual construction of species keys for given cultural conditions (15). Detailed descriptions of the colonies and hyphae, reactions to various chemicals, and growth rates on nutrient media at various temperatures are necessary to make this identification technique a success. I have isolated Suillus granulatus from mycorrhizal rootlets, but the identity of the majority of fungi is not known. I would welcome and appreciate suggestions from any of you on other approaches to use to identify these fungi.

One of the most indisputable means for confirming a mycorrhizal association is by using Melin's (6) technique, with various adaptations by other investigators (2, 5, and 7), of synthesizing mycorrhizae between fungi and tree roots in pure culture. I have used this technique and synthesized mycorrhizae on ponderosa pine with Suillus granulatus, but the results have been negative with other fungi tested. Two factors that have been troublesome in these experiments are insufficient light quantity and high substrate temperature. I hope these problems have been resolved with the recent purchase of a growth chamber which will allow control of temperature and light.

An interesting aspect of the mycorrhizal investigations has been the effect of mycophagous nematodes on the growth and viability of mycorrhizal fungi. These nematodes have been isolated from mycorrhizal rootlets collected from widely separated low elevation stands in central New Mexico.

Laboratory studies have been undertaken to determine if these nematodes could reduce the growth and viability of mycorrhizal fungi common to central New Mexico as well as similar fungi found in other areas of the United States.

Forty fungi, representing 6 orders, 17 families, and 31 genera of basidiomycetes, and one imperfect fungus, were selected for the investigation. Eleven of these fungi, namely Boletus edulis, Suillus granulatus, Leccinum scabrum, Xerocomus chrysenteron, Lactarius uvidus, Clitocybe crassa, C. multiceps, Flammulina velutipes, Pholiota squarrosa, Cantharellus cibarius, and Russula aurantialutea, were obtained from mixed conifer stands in central New Mexico. The remaining 29 fungi were obtained from forest research colleagues of the U. S. Forest Service.

A suspension of 200 nematodes (an undescribed species of Aphelenchoides) was added to 5 cultures of each fungus, and 5 other cultures of each fungus were not inoculated and served as controls. These fungi were grown on potato dextrose agar. The experiment was continued for 40 days after the nematodes were added, and then a small piece of mycelium from each culture was transferred to PDA to determine if the fungi had been killed by the nematodes.

Reduction in linear growth of most fungi was first apparent after the nematodes had fed 8 to 12 days, and reduction was quite distinct after 16 to 20 days. After 40 days of feeding, the nematodes had little or no effect on the linear growth of 19 fungi, their growth being 75 to 100% of their respective controls. Growth of 13 other fungi was reduced to 51 to 73% of their controls, and growth of the remaining 8 was reduced to 50% or less of their controls. Naematoloma fasciculare is an example of a fungus whose linear growth was little affected by the nematodes. Cenococcum graniforme is an example of a fungus in the 51 to 73% category, its mean growth was reduced to 60% of its controls. A fungus whose linear growth was severely affected by nematode feeding was Clitocybe crassa, its mean linear growth was reduced to 25% of its respective controls.

Nematodes concentrated at the periphery of the fungus colonies and fed on surface as well as substrate hyphae. Upon locating a hyphal cell, the nematode penetrated the wall with its spear after about 5 to 10 thrusts, and fed on the cellular contents for 5 to 60 seconds. During feeding, cytoplasm was observed flowing toward the spear orifice. The median bulb valve moved rapidly at the end of each feeding, and bubbles usually appeared in the hyphal cell at the feeding site. These parasitized cells were probably killed.

The viability of 16 fungi was not affected by nematode feeding, but the viability of 13 other fungi was partially affected. The viability of 3 fungi of the latter group, namely Inocybe lacera, Calvatia subcretacea, and Lepista personata, was reduced to 20 to 40%. Eleven fungi failed to revive when transferred to a fresh medium. These 11 fungi were Amanita muscaria, Suillus borealis, Suillus ponderosus, Leccinum scabrum, Hygrophorus piceae, Corticium bicolor, Truncocolumella citrina, Ithyphallus ravenelli, Pisolithus tinctorius, Cenococcum graniforme, and Crinipellis campanella. Most of these fungi grew very slowly on PDA. Two of the fungi, Amanita muscaria and Leccinum scabrum, are frequently found in mixed conifer stands in central New Mexico. Cenococcum graniforme, a fungus that exhibits outstanding resistance to drying and predominates or even becomes the sole mycorrhizal associate of trees growing in droughty climates, also failed to grow. Another fungus that failed to grow, Corticium bicolor, was the one used by Zak to demonstrate, in addition to the work of Marx (3) with Leucopaxillus cerealis var. piceina, that roots with ectotrophic mycorrhizae afford protection against pathogenic fungi.

Nematode populations increased readily on 21 of the test fungi, their numbers increasing to over 100 times the initial population in 40 days. Eggs were observed in cultures of most fungi from 4 to 8 days after the nematodes were added until the experiment was terminated. On 4 fungi, namely Calvatia subcretacea, Russula emetica, Lepista personata, and Clitopilus prunulus, the populations increased 1300, 1700, 1900, and 2300 fold over the initial population. Cultures of some fungi that that were seriously reduced in linear growth supported low nematode populations. These low populations indicated that the nematodes reduced the fungal growth in a short time, and then many of the nematodes gradually starved and were not recovered by the recovery method used. The nematodes were not able to maintain themselves in other cultures that contained an agar medium but lacked the fungus. In fact, nematode populations in these dishes declined almost to zero during the experiment. Nematode eggs were not observed on the agar surface in these dishes. The nematodes remained active for 20 to 28 days, but then their activity gradually declined until they appeared moribund on the agar surface when the experiment was concluded. Nematodes also failed to maintain themselves on Pleurotus corticatus, Paxillus panuoides, and Pholiota squarrosa. Perhaps these fungi and other fungi that were little affected in linear growth and fungus viability produced substances that were toxic to the nematodes. Marx (4) has found that Leucopaxillus cerealis var. piceina secretes an antibiotic, diatretyne nitrile, that, besides being fungitoxic to zoospores of Phytophthora cinnamoni at low concentrations, also had adverse effects on spiral nematodes. Another species of Leucopaxillus, L. albissima, was used in this study and its linear growth and viability were not reduced by the nematodes.

The results of this investigation have revealed that the Aphelenchoides species reduced the growth and viability of some mycorrhizal fungi whereas other fungi were not affected at all. Of the 11 New Mexico fungi tested, the linear growth of 2 was reduced to 38% or less of their respective controls, growth of 4 was reduced to 50 to 75%, and growth of 5 was slightly affected or not affected at all. Viability of 7 of these fungi was not affected, viability of 3 fungi was partially affected, and 1 fungus was killed by nematode feeding. Since this nematode has been found in widely separated low elevation ponderosa pine stands in central New Mexico, it is hypothesized that it may have a role in the premature mortality of ponderosa pine by suppressing the development of its mycorrhizae which in turn may result in reduced tree vigor and exposure of vulnerable tissue to attack by various root pathogens.

Studies are now necessary, however, to determine the significance of mycophagous nematodes on the development of mycorrhizae of southwestern tree species. With an understanding of the effects of these nematodes on the development of mycorrhizae and on the growth and viability of root-pathogenic fungi, the significance of these organisms in the decline of tree vigor and premature mortality of southwestern tree species may be clarified.

Literature Cited

1. Cromer, D.A.N. 1935. The significance of the mycorrhiza of Pinus radiata. Australian For. Bur. Bull. 16. 19 p.

2. Hacskaylo, E. 1953. Pure culture synthesis of pine mycorrhizae in terralite. Mycologia 45: 971-975.

3. Marx, D. H. 1966. The role of ectotrophic mycorrhizal fungi in the resistance of pine roots to infection by Phytophthora cinnamoni Rands. Ph. D. thesis. North Carolina State University.

4. ___________. 1966. Personal communication.

5. Marx, D. H., and B. Zak. 1965. Effect of pH on mycorrhizal formation of slash pine in aseptic culture. Forest Sci. 11: 66-75.

6. Melin, E. 1921. Uber die Mykorrhizen pilze von Pinus sylvestris (L.) and Picea abies (L.) Karst. Svensk. Bot. Tidskr. 15: 192-203.

7. Norkans, Birgitta. 1949. Some mycorrhiza-forming Tricholoma species. Svensk. Bot. Tidskr. 43: 485-490.

8. Pantidou, Maria E. 1961. Cultural studies of Boletaceae. Gyrodon merulioides and four species of Boletinus. Canad. J. Bot. 39: 1149-1162.

9. Raski, D. J., and J. W. Riffle. 1967. Two new species and further notes on Criconemoides Taylor, 1936 (Criconematidae: Nematoda). Proc. Helminthol. Soc. Washington, D. C. 34: 212-219.

10. Riffle, J. W. 1967. Effect of an Aphelenchoides species on the growth of a mycorrhizal and a pseudomycorrhizal fungus. Phytopathology 57: 541-544.

11. Riffle, J. W., and D. D. Lucht. 1966. Root-knot nematode on ponderosa pine in New Mexico. Plant Disease Reptr. 50: 126.

12. Ruehle, J. L. 1962. Histopathological studies of pine roots infected with lance and pine cystoid nematodes. Phytopathology 52: 68-71.

13. __________. 1966. Nematodes parasitic on forest trees I. Reproduction of ectoparasites on pines. Nematologica 12: 443-447.

14. Sutherland, J. R. 1967. Parasitism of Tylenchus emarginatus on conifer seedling roots and some observations on the biology of the nematode. Nematologica 13: 191-196.

15. Trappe, J. M. 1962. Fungus associates of ectotrophic mycorrhizae. Bot. Rev. 28: 538-606.

PANEL II. -- DISEASE CONTROL: SILVICULTURE AND FOREST MANAGEMENT

or

DISEASE CONTROL BY ENVIRONMENTAL MANIPULATION

Benton Howard, Panel Moderator

INTRODUCTION BY MODERATOR

I can't resist the changing of titles; hence, the subtitle given above.

This illustrious panel will present to you the very basis of forest pathology; the guts of the situation; disease control through silviculture and forest management, or environmental manipulation.

Everything we do, everything that has been given before in this session, and everything yet to come in some way or another relates to the environment.

Now I have four panelists, all of whom are erudite, skillful, knowledgeable, witty, and best of all, articulate. My problem, of course, was how to handle such a "fearsome foursome." Well, just let them run wild.

They will start from the stone age of forest pathology and one by one lead us to outer space. From voodoo to to the computor! Let's start the count down and away we go!

Don Leaphart. You all know him so why spend time in reviewing his illustrious career. He will build us a launching platform blended of fairy tales, medieval magic, folklore, and fact. Don, please take over.

HISTORY OF FOREST DISEASE CONTROL THROUGH SILVICULTURE AND FOREST MANAGEMENT

Charles D. Leaphart

Modern practices in control of tree diseases probably had their origin in the 18th Century. Then people emphasized individual trees, as did J. C. Riedel in his Garden Lexicon in 1751, where he recommended a remedy for cankers by cutting out diseased parts and coating with grafting wax. However, perhaps influenced by their medical contemporaries, they also suggested bleeding by boring holes and splitting roots, presumably to rid the tree of causative spirits. Not until a century later were the relationships between diseases and their causes understood, thus providing the basis by which controls could be practically and effectively formulated and applied through silvicultural measures.

Robert Hartig, considered by most foresters and pathologists to be the father of modern Forest Pathology, worked out many of these relationships and consequently provided the impetus for expanded study of Forest Pathology the world over. Hartig actually credited M. Willkomm with stimulating interest in diseases of forest trees through his investigations reported in 1866, primarily on the larch canker which he misidentified. I doubt that Willkomm should be criticized for this oversight because 100 years later the mycologists still haggle over the nomenclature of the genus.

Interestingly, Hartig's philosophy was strikingly similar to some modern pathologists'. As editor of Sommerville's (1894) translation of Hartig's (1882) text, Marshall Ward commented in the preface of the translation about the foresters' criticism of Hartig that too little information was provided relative to details of combative or therapeutic treatment of special diseases. Because I think the comments are partly applicable to the present day, I quote part of Ward's response to criticism of Hartig's text. "To this (i.e., the criticism) the obvious reply is that it is not necessarily the duty of the scientific pathologist to devise the particular mode of attack to be employed in special cases--these plans of remedial treatment involve the outlay of money, labour, etc., which vary in different countries and in different cases, and enough has been done by the investigator who indicates the factors involved. Special works must be consulted regarding the <u>details</u> of treatment, though it seems to me the author, while clearly recognizing this, goes even out of his way to give practical hints as to treatment, and has in many cases put the principal factors concerned in the treatment so clearly that every thinking practical man can do the rest himself. No better illustration of the thoroughly practical nature of his writings could be selected than his recommendations for the treatment of dry-rot."

Most notable among the early workers of the late 19th Century and contemporaries of Hartig were Rostrup in Denmark, Tubeuf in Germany, and Ward in England. Many of our present concepts in control of diseases through silvicultural manipulation had their origin with these men. For example, from their writings came the following recommendations which I believe you will recognize in modified form in today's practices.

1. Eradication of <u>A. mellea</u> and <u>F. annosus</u> from plantations by trenching and destroying affected portions of trees. In older forests, the same procedure could be used to limit destruction.

2. Recognized spores as a danger source, but considered that they (quoting from Ward, 1909) "Need be by no means so much so where knowledge is intelligently applied in removing young fructifications." Thus, the concept of forest sanitation was coming into being as Hartig some years earlier had recognized in comparing the low incidence of foliage diseases in stands where forest litter had been cleaned to the high incidence in others where the litter was allowed to accumulate.

3. Avoidance of certain sites for certain species as for example, larch with respect to the larch canker and Scotch pine with <u>L. pinastri</u>. The value of mixtures of species where larch canker prevailed and of alternating species or using more resistant species where the needle cast was severe was also pointed out to the foresters of that day.

4. Mistletoes, including dwarf mistletoes, were treated by pruning infected branches so as to remove the infection source. Pruning, its timing, and follow-up coating treatment were considered as general preventive measures for other diseases, e.g., heartwood decays, in addition to providing a clean bole, illumination for the undergrowth, and a product for utilization.

Coincident with these silviculturally oriented recommendations, others that have certain similarities to present-day management practices were:

1. From the recognition of the alternate hosts and life cycle of Peridermium pini came the recommendation that seedbeds and nurseries be kept clean of alternate hosts. Mentioned at this time also were C. ribicola and its alternating host cycle. Consequently, the groundwork was at least laid for the recommendations that later followed for the massive ribes eradication programs to control blister rust.

2. Damping-off fungi were handled in the pre-chemical days by altering the environmental factors favorable to spread, alternating seedbeds, and sanitation. However, knowledge of the fungi and related recommendations on sanitation at that time probably had little influence in reducing the hazards from these fungi.

3. Avoidance of wounding and treatment of wounds.

4. Recognition of seed source, seed size, and planting stock size for regeneration. Hartig (1894) qualifyingly stated that transmission of resistant or susceptible characters was not possible from parent to progenies. It has remained to the relatively modern workers in forest pathology to clarify and utilize resistance traits for forest management purposes.

5. Recognition of man's involvement in spread of diseases, even though quarantine laws were to be established later. The first generally effective national enactment did not come until 1912 when the United States passed its National Quarantine Act. However, the Act was too late for the chestnut blight and blister rust and not comprehensive enough to exclude the Dutch elm disease.

When one reviews the more modern American treatments on forest pathology (Baxter 1943, 1952; Boyce 1938, 1948, 1961; and Hubert 1931) to which should be added foreign works, not all were personally reviewed (Nager 1924; Orlos 1951; Peace 1962; Roll-Hansen 1958; Schwerdtfeger 1957), he finds that the principles of disease control have not varied greatly from the original concepts. One could philosophize that the early workers had a wide open field in which they could let their imagination run. Nevertheless, this imagination was well organized and based on astute observation in a day when knowledge was limited and modern research equipment, techniques, and facilities were non-existent.

Whether the silviculturist and forest manager presently react as they did in Hartig's day to a lack of control measures for specific problems is a moot question. From the origin of forest pathology to present, both the forest pathologist and silviculturist have had to work with a complex environment interaction within an exceedingly restrictive economic framework. Now as then, our attention needs focusing on intercommunication about our knowledge of specific problems and integration of control measures into the objectives of management. Our current treatises, e.g., Boyce 1961, attest to considerable improvement of the principles of disease control; and our state of knowledge has been considerably broadened, for which many people, some here, deserve considerable mention. The latter will be summarized by Larry Weir.

To me at least, discouraging as it is to find no better recommendations for control of some diseases than came with the origin of our profession, it is equally disappointing to find that our management people have been unable to make more advantageous use of new information as it came along than they have to date. For example, how long was it before management utilized the concepts developed by J. R. Weir (no relation to Larry) in 1916 for dwarf mistletoe control? Equally at fault, the manager and pathologist might be condemned for not implementing a program that Carl Hartley proposed in 1927 for developing resistant progenies as a means of combating blister rust. Our current rust resistance program 40 years later is considerably late and but little more polished. But while history provides us with critical hindsight, it alternatively gives us, with judicious appraisal, leads which our "crystal-ballers," Harold Offord and Chuck Wellner, can imaginatively apply to the future. One thing is certain to me, i.e., history demonstrates that we silviculturists, managers, and pathologists must turn the wheels together if progress is to be mutually enjoyed in control of diseases on wild or managed forested lands.

References and Books for Leisure Reading

1. Baxter, D. V. 1952. Pathology in Forest Practice. 2d ed. New York, John Wiley and Sons, Inc. (1st ed., 1943).

2. Boyce, J. S. 1961. Forest Pathology. 3d ed. New York, McGraw-Hill Book Co., Inc. (1st ed., 1938).

3. Hartig, R. 1874. Wichtige Krankheiten der Waldbäume. Berlin: Springer, Verlag.

4. __________. 1900. Lehrbuch der Pflanzenkrankheiten. 3d ed. Berlin: Springer-Verlag. (1st ed., 1882).

5. __________. (Transl. from the German by W. Somerville). 1894. Textbook of the Diseases of Trees. London: George Newnes, Ltd.

6. Hartley, Carl. 1927. Forest genetics with particular reference to disease resistance. J. For. 25: 667-686.

7. Hubert, E. E. 1931. An Outline of Forest Pathology. New York, John Wiley and Sons, Inc.

8. Neger, F. W. 1924. Die Krankheiten unserer Waldbäume und Wichtigsten Gartengeholze. 2d ed. Stuttgart: Ferdinand Enke. (1st ed., 1919).

9. Orlos, H. 1951. Przewodnik do Oznaczania Chor'ob Drzew Izgnilizny Drewna (A guide to the determination of tree diseases and wood rots). Warsaw: State Agricultural and Forestry Publisher.

10. Peace, T. R. 1962. Pathology of Trees and Shrubs with Special Reference to Britain. London: Oxford Univ. Press.

11. Reidel, J. Christ. 1751. Kurz Abgefasstes Gartenlexikon Nebst einen Garten-C Lendar. Nordhausen.

12. Roll-Hansen, F. 1958. Forstpatalogi. Norges Landbrukshögskole.

13. Rostrup, E. 1889. Afbildning og Beskrivelse de Farligste Snyltesvampe i Denmarks Skove. Copenhagen: Philipsens Boghandel.

14. Schwerdtfeger, F. 1957. Die Waldkrankheitsen. Ein Lehrbuch der Forstpatalogie und des Forstschutzes. 2d ed. Hamburg and Berlin: P. Parey.

15. Tubeuf, K. von (Transl. from the German by W. G. Smith): 1897. Diseases of Plants Induced by Cryptogamic Parasites. London: Longmans, Green and Co., Ltd.

16. Ward, H. M. 1909. Timber and Some of its Diseases. 3d ed. London: Macmillan and Co., Ltd. (1st ed., 1889).

17. Weir, J. R. 1916. Mistletoe injury to conifers in the Northwest. U.S.D.A. Bull. 360: 1-39.

18. Whetzel, H. H. 1918. An Outline of the History of Phytopathology. Philadelphia and London: W. B. Saunders Co.

19. Willkomm, M. 1866. Die Mikroscopischen Frende des Waldes. Dresden.

Moderator -- Larry Weir is one of those delightfully witty Canadians. He is my neighbor to the North, so frequently I have an opportunity to visit with him. Those of you who only see him occasionally are in for a real treat. Among his many talents: a distinguished scientist, a puckish sense of humor, and a great deal of common sense. He will now ready the rocket for its space venture.

My friend, colleague, and delightful personality --

THE PRESENT STATUS OF SILVICULTURE AND FOREST MANAGEMENT AS IT RELATES TO DISEASE CONTROL

L. C. Weir

The present status of silviculture and forest management as they relate to disease control is, from the standpoint of an increase in our knowledge, quite advanced. The literature abounds with reports of trials and recommendations as to what can be done with silviculture and management to combat forest tree diseases. As to what is accepted by forest managers and silviculturists, the issue, except in a few instances, is somewhat in doubt.

Much of what has been done has been summarized by Hartley (1950), Hepting (1959), and Kuntz (1964), with abbreviated additions supplied in the Reports of the Working Groups in Forest Insects and Diseases since its inauguration some 6 years ago. One might best proceed by dividing up diseases for which silviculture has been recommended as a tool in disease control into 6 groups, namely dwarf mistletoes, stem rots, root and butt rots, cankers, rusts, and a miscellaneous group. Thus they can be dealt with one at a time.

The dwarf mistletoes have received a great deal of attention from researchers with regard to silvicultural control. The reports of Childs (1963), Flora (1966), Gill (1957), Gill and Andrews (1942), Gill and Hawksworth (1964), Hawksworth and Andrews (1961), Hawksworth and Johnson (1961), Kimmey (1957), and Parker (1942), all attest to the value of controlling mistletoe infections through silviculture. There is even evidence to indicate that forest management is interested enough to do something. A number of field trips taken in the past by this group have been based on work done in the study of mistletoe control. Even in British Columbia where it appears that the terms silviculture and disease control are mutually antagonistic to a large extent, sales of forest land for logging purposes have been made with the specific objective of cleaning up mistletoe-infected areas. Studies on the spread and degrees of thinning have also been instigated, so some effort is being made. As a sort of culmination to illustrate the degree of interest that is being shown by management, the Canadian Rocky Mountain National Parks have initiated a program designed to eliminate dwarf mistletoe, and this from a group of guiding hands who in the past have not been noted for their readiness to accept the recommendations of mere foresters and even "merer" forest pathologists. Consequently, I am inclined toward the belief that when the Parks administration acquiesces, then the strongest bastion of resistance has fallen, the wall has been breached, and all is a downhill pull for future recommendations. The reports presented by Flora typify to me the type of study, based on some extrapolation of fundamental research, that is necessary because it puts control in terms of dollars and cents which are the mainstays of forest managers. In some ways, although not entirely of their own making, forest managers are reminiscent of Missouri mules that you have to stun with a 2 x 4 first to get their attention. That attention is best obtained when it can be illustrated in terms like "how do you spell cat?" that the pocketbook will suffer if they don't do something yesterday. Gradually, with the mistletoes, the idea is getting across.

The story is somewhat different when one looks at stem rots. Andrews (1954) pointed out the advisability and the advantages to be gained by pruning ponderosa pine under the age of 60 to reduce loss from western red rot. Sleeth and Bidwell (1936), Hepting (1942), and others have made recommendations about reducing decay in the eastern hardwoods. Baxter (1954), Maloy (1963), Meinecke (1916), and Weir (1923), have observed and reported on what silviculture could do in western forests, and yet I suspect that not many of these recommendations have been listened to and acted upon by forest managers. That last statement is open to alteration from more up-to-date information supplied by you if you would be so kind. The problems of butt rot, with a view toward preventative measures largely silvicultural in nature, have been analyzed and presented by Negruckij (1960), Peace (1938), Rohmeder (1937), and Sinclair (1964), and many others. While some of these relate in part to logging and fire damage that results in butt rot, some specifically, like those of Driver (1965) recommending changes in time of cutting to lessen loss from <u>Fomes annosus</u>, relate silviculture to disease control. A similar situation exists with regard to work done on canker diseases such as that done by Grant and Childs (1940), Hepting

(1940), and Roth and Hepting (1954). Recommendations were made that would lower losses if followed but I suspect that action taken has been limited if, in fact, it exists at all.

With the rusts, particularly white pine blister rust, emphasis has been laid on the problem of Ribes eradication. Here the reports of Davis and Moss (1940), Martin (1939), Moss and Wellner (1953), Offord et al (1952), and Quick (1954), and a host of others have given forest management something to work on and for the most part a great deal of effort has been expended. Unfortunately, because of inherent difficulties in eradication and rising costs, the program is being terminated in Idaho, Montana, and eastern Washington (Bongberg 1967) and is being replaced by the emphasis to be placed on management of commercially valuable trees such as larch and fir that normally form part of the forest cover in the white pine forest type. However, eradication of Ribes is being continued pending analysis of effect in Oregon, California, the Lake States, and the Northeastern States. In Canada the Ribes eradication program is limited to Ontario where work is continuing under the auspices of the provincial government in conjunction with the federal body. Pruning has been reported to be effective in white pine by Weber (1964) and in slash pine by Harms (1961). It appears probable to me that this form of action has more promise when applied to plantations than to natural stands. In some cases like the sweetfern rust of hard pines, control is probably limited to the thinning out of diseased trees (Anderson 1963). In connection with blister rust and other rusts, one must mention the immense amount of work that is being done in genetics and the breeding of resistant species, because here also the degree of emphasis will be increased. Adequate coverage of what has been and what is being done has been summarized by Clapper (1952) in relation to diseases in general and you are all familiar with the limitations of breeding as expressed by Boyce (1957). The studies of individuals like Bingham must be mentioned because they are well worthy of note.

Root rots present a particular problem to disease control through silviculture at the moment. Poria weirii and Armillaria are cases in point. Sanitation fellings and/or clear-cuts do little to aid the situation because of the continued presence of viable inoculum. Here is also the added hazard (Wallis 1954) of blow-down of residuals that were infected and apparently without symptoms. Without wanting to tread into territory to be covered by the next speakers, I should like to point out that with the root-rots, much work has to be done very shortly.

With the miscellaneous group some work has also been done. The use of fire to control the brown-spot disease of longleaf pine is perhaps the most outstanding example of success while the sanitation and pruning treatments for control of oak wilt and Dutch elm disease have been less evidently successful in total.

In effect, the prevailing feeling one gets in perusing the literature is that a great deal of work has been done to illustrate to the silviculturist and the forest manager the value of coordinated effort in obtaining successful control of diseases while simultaneously achieving the aims of a purely forestry point of view. As Macon (1967) points out, one might decry the lack of response from the forest manager but still must admit that there are frequently bases for the lack. Those on whose shoulders rests the responsibility for practicing forestry in the field have in truth been doing much of what has been recommended although their motives for so doing are not those we emphasize. For example, when marking crews are sent out to prepare an area for a thinning operation, they automatically remove trees that fail to conform to the standards set up for crop trees. Consequently much of what is to be removed also fits because of disease conditions to the pathologist's ideas. I think we might have a legitimate complaint that they are not taking what is essentially good forestry far enough in that their marking rarely extends beyond the boundaries of the area to be thinned and, as a result, inoculum in almost every conceivable form is present to wreak havoc in the residual crop trees.

I have given a few of the many examples available to illustrate the application of silviculture and forest management as it presently is being employed in disease control. There is much that I have missed and for that I tender my apologies. That I am not completely alone is illustrated by the fact that I made application to International Forest Tree Disease Register for all literature from all countries and all hosts related to control of disease through silviculture. I did this with some fear and trepidation expecting to be inundated. I received in a remarkably short time the imposing list of 19 titles. My own list is roughly four times that size and I felt it was inadequate. However, the next speakers to whom I now give the floor need only worry about their imaginations and holding them in check for I feel, on the basis of what I have read, that their talks should be entitled "Opportunities Unlimited."

Literature Reviewed

1. Adams, W. R., et al. 1939. The relation of stand composition to crop security. J. For. 37(1): 49-54.

2. Anderson, G. W. 1963. Sweetfern rust on hard pines. U.S.D.A. For. Pest Leaflet 79.

3. Andrews, S. R. 1954. Effect of pruning on western red rot in young ponderosa pine in the Southwest. J. For. 52(1): 33-38.

4. Baxter, D. V. 1954. Differential treatment in American westcoastal forests based on the distribution of certain fungi. Proc. VIII Int. Congr. Bot., Sect. 13: 117-119. (Paris).

5. Bongberg, J. W. 1967. United States: Forest insect and disease survey and control. In: Rept. of 5th Meeting FAO North American Forestry Commission. Working Group on Forest Insects and Diseases. Pineville, Louisiana. April 1967.

6. Boyce, J. S. 1957. Limitations of forest disease control by breeding and selection. Proc. Soc. Amer. For., 1956-57.

7. Buckland, D.C., and G.W. Wallis. 1956. The control of yellow laminated root rot of Douglas fir. Can. J. Bot. 32(1): 14-19.

8. Buckland, D. C., A.C. Molnar, and G.W. Wallis. 1954. Yellow laminated root rot of Douglas fir. Can. J. Bot. 32(1): 69-81.

9. Campbell, W. A. 1965. Reducing losses from disease. In A Guide to Loblolly and Slash Pine Plantation Management in Southeastern U.S.A., Georgia Forest Research Council, Report No. 14.

10. Campbell, W. A., and P. Spaulding. 1942. Stand improvement of northern hardwoods in relation to diseases in the northeast. Allegheny For. Expt. Sta. Occ. Pap. 5.

11. Childs, T. W. 1947. Management of second growth forests of the Douglas fir region. U.S.F.S. PNW For. and Rge. Expt. Station Prog. Rept.: 94-100.

12. Childs, T. W. 1963. Dwarfmistletoe control opportunities in ponderosa pine reproduction. PNW For. and Rge. Expt. Sta. Paper.

13. Clapper, R. B. 1952. Breeding and establishing new trees resistant to disease. Econ. Bot. 6(3): 271-293.

14. Davis, K. P., and V. D. Moss. 1940. Blister rust control in the management of western white pine. Nor. Rocky Mtn. For. and Rge. Expt. Sta. Pap. 3.

15. Driver, C. H. 1965. The occurrence and control of Annosus root rot in southern pine plantations. Southland Exp. Forest Tech. Note 23.

16. Flora, D. F. 1966. Economic guides for ponderosa pine dwarfmistletoe control in young stands of the Pacific Northwest. U.S.F.S. PNW For. and Rge. Expt. Sta. Res. Pap. 24.

17. __________. 1966. A method of forecasting returns from ponderosa pine dwarfmistletoe control. U.S.F.S. PNW For. and Rge. Expt. Sta. Pap. 32.

18. Fowler, M. E. 1958. Oak wilt. U.S.D.A. Forest Pest Leaflet 29.

19. Fritch, K. C., M. K. Schwartz, and H. S. McNabb, Jr. 1957. Observations on the effect of logging on the spread of oak wilt in the Brayton Memorial Forest, Iowa. Iowa Acad. Sci. 64: 110-112.

20. Gadd, C. H. 1940. Ring-barking of trees and root disease. Tea Quart. 13(3): 117-123.

21. Gillespie, W. H., and R. P. True. 1963. Impact of the deep-girdle treatment and associated effects upon the production of fungus mats by naturally infected oak wilt trees. Plant Dis. Reptr. 47(8): 748-752.

22. Gill, L. S. 1957. Dwarfmistletoe of lodgepole pine. U.S.D.A. Forest Pest Leaflet 18.

23. Gill, L. S., and S. R. Andrews. 1942. Behaviour of dwarfmistletoes of western conifers in relation to control. Phytopath (Abs.) 32(1): 21-22.

24. Gill, L. S., and F. G. Hawksworth. 1964. Dwarfmistletoe of lodgepole pine. U.S.D.A. Forest Pest Leaflet 18 (revised).

25. Grant, T. J., and T. W. Childs. 1940. Nectria canker of northeastern hardwoods in relation to stand improvement. J. For. 38: 797-802.

26. Hartley, C. 1950. The division of forest pathology. Plant Dis. Reptr. (Suppl.) 195: 445-462.

27. Harms, W. R. 1961. Growth of fusiform cankers on young slash pine. SE For. and Rge. Expt. Sta. Res. Note 159.

28. Hawksworth, F. G., and S. T. Andrews. 1961. Guides for pruning dwarfmistletoe-infected ponderosa pine branches. RM For. and Rge. Expt. Sta. Res. Note 64.

29. Hawksworth, F. G., and N. E. Johnson. 1961. Guides for pruning dwarfmistletoe-infected lodgepole pine branches. U.S.F.S. RM For. and Rge. Expt. Sta. Res. Note 65.

30. Hepting, G. H. 1940. Early removal of surplus oak sprouts desirable to reduce butt rot. U.S.F.S. Appalachian For. Expt. Sta. Tech. Note 40.

31. __________. 1940. Eastern forest tree diseases in relation to stand improvement. Emergency Conservation Work For. Pub. 2 (revised).

32. __________. 1942. Reducing losses from tree diseases in eastern forests and farm woodlots. U.S.D.A. Farmers Bull. 1887.

33. __________. 1959. Forest pathology in forest management. Proc. IX Int. Congr. Bot. Vol. 2: 161-162. (Montreal).

34. Hepting, G. H., and J. K. McClarren. 1946. Preventing rot in young oak stands. U.S.D.A. Agr. Res. Admin. R.A.S. 68 p.

35. Jacquiot, C. 1956. (Lignivorous fungi: their function in nature and their technical importance). Nature (Paris) 3258: 395-400.

36. Jemison, G. M., and G. H. Hepting. 1949. Timber stand improvement in the southern Appalachian region. U.S.D.A. Misc. Pub. 693.

37. Jensen, V. S. 1940. Results of thinning and its effects on residual yellow birch and associated hardwood species. U.S.F.S. NE For. and Rge. Expt. Sta. Tech. Note 33.

38. Kimmey, J. W. 1957. Dwarfmistletoes of California and their control. U.S.F.S. Cal. For. and Rge. Expt. Sta. Tech. Note 19.

39. Klyushnik, P. I. 1958. (Means of increasing the resistance to fungal diseases in the steppe zone). Bull. Soc. Nat. Moscow, Ser. Biol. 63(1): 65-69. (Rev. Appl. Mycol. 37: 602).

40. Krangauz, R. A. 1961. (Dessication of deciduous trees from disease and control measures). Sel'khozgiz (Moscow): 206-222. (Biol. Abs. 44: 619).

41. Kuntz, J. E. Forest disease control: indirect measures. FAO/IUFRO Symposium, Inter. Dangerous For. Diseases and Insects Vol. 2.

42. Macon, J. W. 1967. On connections between research and forest management. J. For. 65(1): 24-28.

43. Maloy, O. C. 1963. Sporulation and sporophore survival in Echinodontium tinctorum. Plant Dis. Reptr. 47(7): 627-631.

44. Martin, J. F. 1939. Eradication of the cultivated black currant in the white pine regions. U.S.D.A. Leaflet 175.

45. Meinecke, E. P. 1916. Forest pathology in forest regulation. U.S.D.A. Bull. 275.

46. Morris, C. L. 1955. Control of mat formation by the oak wilt fungus by early felling of diseased trees. Plant Dis. Reptr. 39(3): 258-260.

47. Moss, V. D., and C. A. Wellner. 1953. Aiding blister rust control by silvicultural measures in the western white pine type. U.S.D.A. Forest Service Circ. 919.

48. McGillivray, K. D. 1946. New methods and an old menace. Bulldozers can help to control Armillaria. Agr. Gaz. N.S. Wales 57: 362-363.

49. McNabb, H. S., Jr. 1955. Oak wilt research--a lesson in cooperation. Ames Forester 42: 17-19.

50. Negruckij, S. F. 1960. (The intensity of sanitation fellings to control Fomes annosus). Les/n Z. Arbangel'sk 3(1): 162-164. [For. Abs. 22(4): 4727].

51. Offord, H. R. et al. 1952. Improvement in the control of Ribes by chemical and mechanical methods. U.S.D.A. Circ. 906.

52. Ohman, J. H., and K. J. Kessler, Jr. 1964. White trunk rot of hardwoods. U.S.D.A. For. Pest Leaflet 88.

53. Parker, H. A. 1942. Control of mistletoe. Can. Dept. Mines and Resources For. Branch Silviculture Leaflet 15.

54. Peace, T. R. 1938. Butt rot of conifers in Great Britain. Quart. J. For. 32(2): 81-104.

55. Prutenskaya, M. 1962. (The control of heartwood mold of Juglans regia). Sel'skoe Khoz. Kirgizii 1: 33-35. [Biol. Abs. 43: 1638 (20830)].

56. Quick, C. R. 1954. Ecology of the Sierra Nevada gooseberry in relation to blister rust control. U.S.D.A. Circ. 937.

57. Rayner, R. W. 1959. Root rot of coffee and ring-barking of shade trees. Kenya Coffee 24(285): 361-365.

58. Rohmeder, E. 1937. [Stem rot (root rot and wound rot) of standing spruce]. Mitt Landes Forstverw. Bayerns 23(7): 166.

59. Roth, E. R., and G. H. Hepting. 1954. Eradication and thinning tests for Nectria and Strumella canker control in Maryland. J. For. 52(4): 253-256.

60. Roth, E. R., and B. Sleeth. 1939. Butt rot in unburned sprout oak stands. U.S.D.A. For. Serv. Tech. Bull. 584.

61. Sinclair, W. A. 1964. Root and butt rot of conifers caused by F. annosus with special reference to inoculum dispersal and control of the disease in New York. Cornell Agr. Expt. Sta. Memoirs 391.

62. Sleeth, B., and B. Bidwell. 1936. Recommendations for keeping the decay hazard at a minimum in silvicultural cuttings in Connecticut sprout hardwood stands. Prog. Rept. Circ. 3. U.S.D.A.

63. Spaulding, P., and J. R. Hansbrough. 1964. Decay of balsam fir in New England and New York. U.S.D.A. Tech. Bull. 872.

64. Varadaraja-Ivengar, A. V. 1961. An effective method of controlling spike disease in sandal. Ind. Forester 87(9): 563-571.

65. Walker, J. M. 1945. Recommendations for control of canker stain of plane trees. U.S.D.A. Bur. Plant Ind. Pub.

66. Wallis, G. W. 1954. Poria weirii root rot II.: some factors relating to the control of the disease of Douglas fir. Interim Rept., Can. Dept. Agr. For. Biol. Div. Victoria. (unpublished).

67. ________. 1954. Commercial thinning in Douglas fir in relation to control of Poria weirii root rot. Can. Dept. Agr. Bi-Monthly Prog. Rept. 10(3): 4.

68. Wallis, G. W., and D. C. Buckland. 1955. The effect of trenching on the spread of yellow laminated root rot of Douglas fir. For. Chron. 31(4): 356-359.

69. Wardlaw, C. W. 1950. Armillaria root and trunk rot of oil palms in the Belgian Congo. Trop. Agr. Trin. 27(4-6): 95-97.

70. Weber, R. 1964. Early pruning reduces blister rust mortality in white pine plantations. U.S.F.S. Research Note LS-38.

71. Weir, J. R. 1923. The effect of broadcast burning of sales areas on the growth of cull-producing fungi. J. For. 21(2): 183-184.

Moderator--Now count down time and all systems go -- AOK. Where we will end up I don't know, but I do know this launching won't abort! Not with Hall Offord and Chuck Wellner as the astronauts.

Harold Offord--urbane, suave, debonair man about town, unreliable--says so himself, connoisseur of good food and liquor, and noted pathologist. A vivid imagination and most skillful in communicating his concepts to us lesser mortals.

Hall take us to the bright and shining future.

THE FUTURE OF FOREST DISEASE CONTROL IN FOREST MANAGEMENT: THE SPLIT-LEVEL FOREST PATHOLOGIST

H. R. Offord

Leaphart and Weir have brilliantly summarized what has been done and what is being done in silviculture and forest pathology. Later we will hear a stimulating and informative report by Wellner giving the silviculturist's viewpoint on today's problems and tomorrow's plans for forest management. These three papers meet the high standards of WIFDWC for logic, scientific narration, and scholarly references to past and current accomplishments. I propose to avoid a pragmatic pessimism about current problems and to take a fanciful look at what lies ahead on the heavenly course of those twin stars of the forestry universe--pathology and silviculture. They have been, are, and will be "doppelgangers" in both fact and fancy. Thus, much of what I have to say about pathology will be an extrapolation of the objectives and new techniques of the silviculturist.

Disease problems that plague foresters have their origins in (1) native pathogens that we generally understand but which in a man-changed environment may take on new and undesirable behavior patterns. And--(2) exotic or foreign pathogens that are introduced into new hosts and into new environments.

Both classes of pathogens will have to be studied in relation to a dynamic biological environment and in the light of shifting priorities made by increased populations needing the products and services of forest stands. These are complex jobs in applied ecology that will have to be tackled in his share of the job by the split-level forest pathologist. Fortunately new tools will simplify these Herculean tasks.

Forest pathologists will share with forest managers in the faster, more reliable and more productive ways of performing work. Truly if the mountain won't come to Mohammed we can now move the mountain. No question that we will be doing a better physical job of site preparation, planning, pruning, thinning, and harvesting. And by the same token much of the prescribed forest protection will be done more rapidly and effectively. With these physical improvements will come biologically better seed and improved lines of planting stock for pest resistance and hardiness to extremes of moisture and temperature. When energy becomes less costly, as it surely will, we can afford to water, fertilize, modify light and temperature, and eliminate forest trash, thus minimizing many diseases associated with low vigor of crop plant.

The split-level pathologist will have to know more about the ecology of native pathogens while he keeps a sharp watch on exotic organisms. With supersonic jets spanning the continents in a few hours it is unlikely that we can do more than slow down the rate at which forest pathogens are being distributed about the world. The pathologist will have to come up with many improvements in survey, identification, and control if we are to keep up with the needs of forest managers in the fast moving, competitive, and demanding world of the 21st century. First a prediction about the survey situation.

SURVEY

New methods of sampling and rapid techniques for scanning individual trees or groups of trees are on hand or on order. For example the simulation techniques of computer analysis permits the rapid evaluation of alternate sampling procedures without the costly method of establishing plots or running strips in the field. Sequential sampling for example can, under certain specified conditions, give a rapid appraisal of success or failure in meeting control standards. It is desirable and practical to adopt more flexible survey methods than those that have been used in the past. Survey techniques should be geared closely to control needs and to the long range plans of forest managers who must have a reliable estimate of disease impact. The motto should be--"Millions for biologically useful data but not one cent for file records."

High resolution color from aerial photography will be improved to the point where discrete infections such as dwarfmistletoe or cankered branches in open stands can be recorded quickly for subsequent automated counting and analysis. Other symptoms of damage in the crown of mature trees such as early chlorosis and changes in thermal radiation will be recorded on color film or by other sensors.

For survey data and records from field plots a radio telephone will be used to feed all pertinent information to a computer center. When the researcher returns to his laboratory he will find print-out sheets waiting with a preliminary summary and analysis of his observations. By establishing trends early in a study or a survey the chances of moving too far into a blind alley will be minimized.

An all-seeing X-ray camera or perhaps a sonar device will provide a record in profile of the decay pattern in trees. Also, such scrutinizers will show the location of compression wood or frost cracks and similar potential weak spots in live standing trees. Thus recreation and industrial foresters will simultaneously solve problems of safety and of economic values.

Other lines of research will keep us on top of the air pollution problem. By continuous flow chromatographic analyzers we can identify areas in which damaging levels of air pollutants occur. Also we will develop sensitive tissue tests as early warning signals and confirmation of unfavorable environment in advance of the obvious symptoms of damage.

These are but a few examples of what lies ahead of us for improvement of Survey. Now let's refer briefly to -

IDENTIFICATION

There will be a great need for the mycologist and taxonomist in the golden era of the 21st century. The mycologist must provide rapid identification of a more complete inventory of the ecologic complex. The mere presence of some specific fungus in the soil does not necessarily mean that it will proliferate to damaging levels. If we know the nutritional needs and the environmental preferences of competing organisms we can give nature a nudge in the right direction. Quick and positive identification of pathogens is the key to many of the practical problems faced by foresters in nurseries and in young forests. Also, we want more information on the impact of nonpathogenic agents on trees which results in low vigor, stagnation, and high susceptibility to endemic pathogens. By means of tissue tests that are precise and accurate we can identify levels of cell metabolism characteristic of pathogenic or nonpathogenic agents.

Now let's make some predictions about control that third wing of the house where dwells the split-level forest pathologist.

CONTROL

I could take the easy route of forecasting developments in control by saying that we shall develop biological or natural methods of control for all major forest pests. No question--much help will come from geneticists and silviculturists who will be providing elite seed and pest resistant lines of planting stock. For some time, however, their work will be be directed to a few of the destructive pathogens--perhaps some of the host specific obligate parasites, such as C. ribicola. I predict that some of this resistance will be induced or built into a plant species by chemical means. Progress in the study of the structure and nature of the genes may give us a leg up on this complex research. I am optimistic about chasing that old will-o'-the-wisp the biochemical nature of disease resistance. It is all so very simple. All that we need to know is how sporidia can tell the difference between a white pine and a yellow pine.

Given an understanding of the biochemical nature of disease resistance we can expect to make more rapid progress in the design of systemic and highly selective toxicants. These toxicants may be fungistatic or fungicidal. In some cases the best way to control a damaging pathogen is to work with an associated fungus. By the use of specific antibiotics we can shift the ecologic balance to favor the harmless entities.

In the plant sciences we can tap a vast reservoir of basic research on the improvements in yield and quality. This work will help us understand and control the biochemical nature of disease resistance. I believe that the amino-acid, phenolics, and associated enzymes are critical. For example, the brilliant work of biochemists at Purdue who discovered that a mutant gene called opaque-2 was directly related to the level of the amino-acid lysine (in corn). Corn lines, normally deficient in lysine and associated tryptophane, when bred with opaque-2 contain grain with 10 times the nutritional level of regular corn. The opaque-2 gene in corn is recessive and apparently was overlooked in corn although its identity had been on record for over 30 years.

Of interest to pathologists and silviculturists are the many microorganisms that have intriguing possibilities for increasing food supplies.

What better place to control and use micro-organisms than in the great outdoor (and mostly for free) laboratory of the manipulated forest? Algae and yeasts, leaf proteins, and bacterial transformations of presently unused plant material are tools for food production in the hands of the skilled biologists.

In the area of specific and systemic fungicides I strongly favor intensified study of the toxins and biogens produced by natural parasites of the pest. It is known that at cellular level fungi will, at any one time, absorb and react with more than one toxophore. Much information is needed on the role of enzymes and antimetabolites associated with the establishment of infection. Why are the sporidia and haustoria of C. ribicola so happy with the cell contents of white pine tissue and so revolted by those of ponderosa pine.

For all types of direct control by chemical methods there will be new and improved techniques for forecasting weather. When and how often to spray or treat are just as important as how to spray and what to use.

In addition to the new biological and chemical controls we shall have to hand a powerful battery of mechanical and physical aids. Logging by balloon, helicopter, etc. will minimize all types of injuries to the bole and to the roots of trees. To sanitize forest soil against root diseases all thinning will be done by a powerful extractor that snapsoff the stem and then will pull all of the root system out of the ground. In situations where this may not be economic we can instead deaden all weed trees by the use of a portable laser gun. This type of controlled heat may also be used to excise or cauterize the margin of cankers in the bole of crop trees.

For a concluding shot at the control target I wish to use my favorite weapon of "the package approach to forest pest control." Hopefully the pathologist, entomologist, and silviculturist will combine into that scientist for all seasons who understands the what, where, when, and how of pest control.

EPILOGUE

Under the aegis of sound planning the pathologist and silviculturist will have available many new and better tools and methods of growing, protecting, and harvesting forest products. The split-level pathologist is the biological scientist of the future who will focus imagination and technical competence on the forest manager's need for survey, identification, and control of forest diseases. Computer techniques of simulation and linear programming will evaluate all economic and biologic factors in the management of wildland areas. The economics of disease control will be clearly set forth and prescribed in the overall management plan; and the specifics of the impact of both native and exotic pathogens will be recognized and evaluated so that there will be few if any crash programs that fall short of goals.

As we conclude our forecast about the work of the split-level forest pathologist it is appropriate that we cast an Olympian eye on the factors and motivations that will prod him along the way.

High labor costs, machine handling and processing of wood fibre, and a supply of flexible and low cost energy will direct the management of forest lands along lines similar to that used on agricultural land. It will be essential and economic to maintain fully stocked disease free, fast growing forests. Also, there can be a new emphasis on quality and variety of forest products under the impetus of a leisure-oriented society. Recreation can help pay for an aesthetic and productive forest stand. The land is there, the product is needed, and the price will be right. Hire an extra pathologist and silviculturist and do a better job.

Moderator--Chuck fought hard, but I finally landed him. The main reason--his presence would preclude someone else from attending. However, now that attendance is at the discretion of the Director; well, all hands can be here. Chuck was my personal choice because he knows both sides; in fact, his knowledge of pathology is but a bit behind his stature as a silviculturist.

Grew up in Idaho. Undergraduate work at Idaho; graduate work at Yale; up through the research chairs; leader of the Inland Empire Research Center; Division Chief, Forest Disease and Silviculture Research; now Assistant Director, Forest Insect, Disease, and Timber Management Research. Has received Department's Superior Service Award; listed in "American Men of Science," and "Who's Who in the West."

Despite all this, he is a modest and unassuming person.

I talk a lot and say little, but Chuck is different -- he talks little, but says a lot. I suggest that you listen.

Chuck, Offord took us to outer space. Now it's up to you to bring us back to earth.

A SILVICULTURIST LOOKS AT THE FUTURE OF FOREST DISEASE CONTROL

Charles A. Wellner

Introduction

Ben picked the wrong man to give you a far out look by a silviculturist at the future of forest disease control. I'm sure many of you in the audience characterize me as a practical, conservative person, not given to flights of fancy. As a research administrator of many years I cannot claim to be conversant with all of the latest developments in silviculture, much less in forest disease control. This is not given as an apology but simply as a frame of reference. So, I will concern myself mainly with what is here and known rather than with poor guesses as to what the future may bring.

All fields of knowledge are advancing so rapidly and areas of applied science changing so swiftly that the control of forest diseases can move in only one direction--toward a vast improvement! You forest pathologists may take pride in the body of knowledge you have amassed. You are to be congratulated on the quality and quantity of results flowing from your research programs. Your record as scientists is outstanding. I only wish I could say as much for the application of your knowledge to the control of forest diseases. Although we can point to some examples of effective control of forest diseases, these are barely a beginning on the total job to be done. When concerned over the forest disease situation, I used to cheer up by recalling the achievements in control of white pine blister rust and the good beginnings in control of dwarfmistletoes. But the recent reverses in blister rust control and new trouble spots in the control of dwarfmistletoes leave no room for complacency. Frankly our record to this date in forest disease control is not good. That is why I say we can go in only one direction--toward improving the situation.

Why Isn't Our Record Better?

Why isn't our record better in view of existing knowledge of forest diseases? Soul searching here is worthwhile only if it gives clues to improve future action. I list the following as major causes:

1. The economics of disease control. Economics--costs and returns--determines what can be done. Measures to protect forests from diseases must be within the economic limits of total forest management. The relatively low value of forest crops has sharply circumscribed control measures. All the wishing in the world, all the insistence of pathologists, will not bring adoption of biologically successful control measures if they are not within economic reach. Perhaps when the economics of disease control is better understood--and I'll point out later that it needs to be--it may become a less significant stumbling block than it is today.

2. Knowledge of forest diseases has not gotten into the mainstream of management practice. It is discouraging at times to observe how little of the knowledge concerning disease control is actually used in managing forests. We see simple, well-proved practices, not to mention more sophisticated principles and methods of disease control, violated frequently and repeatedly.

I believe all of us--pathologists, silviculturists, educators, land managers--have contributed to this situation. Too many people consider forest protection as a thing apart, an activity only for specialists. The forest pathologist frequently has jealously guarded his specialty and claimed it was his and his alone. Long ago Pop Hawley at Yale insisted that forest protection was an integral part of silviculture and included a section on principles of forest disease control in his textbook on the practice of silviculture. Conversely, Smith's recent text on the practice of silviculture implies that forest protection is something separate from silviculture. Even our land management organizational structures help to perpetuate the situation when they frequently have separate units for forest protection and timber management. I suspect that the measure needed for direct control of introduced diseases such as white pine blister rust, like insect control projects, have encouraged some managers to consider disease control as a distinct and separate practice in forest management. I'm sure that insufficient training of silviculturists and forest managers is responsible too. Timber management guides that do not take forest diseases sufficiently into account contribute to this deficiency. The fact is, we are not using what we know.

3. Knowledge of damage by specific diseases has been lacking. It is one thing to know the identification, taxonomy, cytology, histology, host-parasite relations, and ecology of a disease and quite another to know its effects and the magnitude of its damage. Without a good estimate of damage we do not know how important each disease is and whether control is needed. In the United States the "Timber Resource Review" pioneered in estimates of mortality and growth loss, but any one who helped develop those guesstimates knows their weak basis. "Timber Trends" estimates are no better than those of the TRR. I'm very much concerned that current forest inventory procedures in the United States do not yet give us the information needed on mortality and growth loss for all diseases, let alone for specific diseases.

4. Basic knowledge about many specific diseases has been insufficient to develop control measures. Little needs to be said on this topic to a group that knows far better than I the deficiencies in knowledge of forest diseases. I'll only repeat that our knowledge, good as it is, is not good enough. We spent millions of dollars on control of white pine blister rust in the northern Rocky Mountains before we discovered our methods wouldn't do the job there.

The Future

The purpose of my apparent pessimism up to this point has been to emphasize that our first job in the future is to make sure we use what we already know. At the same time we need to draw on all the creativeness of forest pathologists and other scientists to develop new, imaginative, effective, economical methods that will prevent, control, or permit us to live with forest diseases.

The first job is to use what we know.

Don Leaphart and Larry Weir have reviewed the control methods that we now have. Some old, some new, some simple, some complex--they add up to an impressive, steadily increasing body of knowledge about how to combat forest diseases. Let's list some of the tried and true practices again:

- Maintaining vigorous trees and stands
- Selecting inherently resistant stock in planting
- Fitting the right species to the site
- Fitting the right seed source to the site
- Avoiding certain sites
- Avoiding certain soils
- Favoring natural over artificial regeneration
- Favoring mixed over pure stands
- Favoring native species
- Favoring natural stand conditions
- Using proved practices for site improvement
- Making timely thinnings
- Using proved control methods such as cleancutting for dwarfmistletoe control

This body of knowledge must be made a vital part of routine silvicultural practice. It must enter into every phase of silviculture from seed selection and nursery practice through stand establishment and stand culture to harvest of the stand. Each of us has the responsibility to work together to this end.

We have some things going for us and some against us. Economics is slowly going our way; increasing timber values permit practices not possible 30 or even 20 years ago.

In contrast, changing times are heading certain practices toward trouble. Although we have a very positive silvicultural control for dwarfmistletoe, clearcutting cannot be used in some situations, and public objections to clearcuts are mounting by the hour. Prescribed broadcast burning is another management practice that has long been favored as a desirable prophylactic measure by some forest pathologists. But the very real and widespread official concern over air pollution is forcing a search for alternatives. I'm not sure the disease implications of these alternatives are known or are often even being considered. What do such practices as chipping of slash or breaking and crushing slash into the soil mean in the control of forest diseases? I'm concerned, too, at the virtual discontinuance of tree pruning on the National Forests simply because dollar inputs did not produce sufficient clear wood values; the benefits of pruning in certain situations as a disease control measure were hardly considered.

The second job is to develop imaginative new methods for recognition, inventory, evaluation, and control of forest diseases.

Not many years ago control of forest diseases, because of costs, had to be limited mainly to what could be accomplished during the various cuttings to which a managed forest was subjected. The intensive measures for disease control used in agriculture had to be restricted in forestry to the nursery, again because of costs. Increasing value of wood products and recognition of the great values of forests for other uses and services are changing the situation somewhat. We should not limit our thinking on disease control measures but should explore all possibilities. Here are some needs and possibilities as I see them:

1. Better procedures for detection and inventory. The equation for predicting future timber supplies depends on reliable information on growth, cutting, and losses. No matter how good the information may be on growth and cutting, it is of limited value unless we have equally reliable information on losses. We need this information for other purposes. We need it to set priorities on research effort. We need it to support requests for funding for research and for control. Recent advances in remote sensing to recognize disease conditions look very promising. New forest inventory techniques developed by Al Stage emphasizing destructive sampling offer a means to improve markedly estimates of losses by heart rotting organisms. We seriously need procedures to determine the magnitude of losses caused by diseases--procedures that can be incorporated into inventory systems. We need more detailed evaluations of effects of individual diseases. And we need all this soon!

2. Economic evaluations of the effects of diseases. Management funding and action increasingly depend on sound economic evaluations of effects of diseases. The work of Flora and others on this problem is an excellent start. But it is only a start. Economic studies of many diseases are needed now in many regions. Unfortunately, those making economic evaluations usually find that the biological basis for evaluations is very inadequate.

3. Better control methods. As this is a major topic in itself, I'll concentrate on only a few needs:

(a) Root diseases. We need an immense amount of research on root diseases before we can seriously talk about control. Experience with *Fomes annosus* is a help, and the work of Hacskaylo, Zak, Marx, and others suggesting the role of mycorrhizae as a barrier to root pathogens is exciting. The root disease problem is urgent and needs study now.

(b) Chemical or biological controls. These are needed for many diseases but especially for white pine blister rust and for the mistletoes. A development in this field could open unlimited possibilities.

(c) Cultural measures. We need to know a lot more about the benefits and place of many intensive cultural means such as cultivation, fertilization, irrigation, fumigation, spraying, dusting, and crop rotation. We also need to know more about the benefits of burning, clearcutting, and other silvicultural operations in disease control.

(d) Genetic selection. Results of present programs in breeding for disease resistance are so encouraging that we need to move full speed ahead where this means offers opportunities. We need to be fully cognizant, however, of the dangers and pitfalls, and we must investigate them.

Challenges to Forest Pathologists

The future actively challenges forest pathologists. I know you will continue learning about forest diseases and developing new, unique, and better means of keeping diseases within bounds. As scientists most interested in forest diseases you have a special and major responsibility to put your knowledge to work. I suggest you need to give greater effort to:

1. Selling what you know to forest managers.

2. Bringing your knowledge of forest diseases more sharply to bear on the development of economically sound control methods.

3. Determining the effects of forest diseases and assessing the losses they are causing.

Despite the pessimistic tone of my opening remarks, I believe the future for forest disease control looks bright. The creativeness of forest pathologists and allied scientists will devise new and better methods to protect our forests. However, the brightness of the future depends in a large measure on how successful all of us are in cooperating to bring all available knowledge about disease control into the mainstream of forestry practice.

PANEL III. -- THE TEAM APPROACH IN FOREST PATHOLOGY

Keith R. Shea, Panel Moderator

THE TEAM APPROACH AND RESEARCH ADMINISTRATION

C. Gardner Shaw

The need for a new or expanded research program is usually recognized by a research worker long before an administrator becomes cognizant of the need. This individual, whom we might call the innovator, may take various steps to find financial support for the investigation. Let's assume that the innovator takes the logical initial step and consults the director of research of his organization. Let's also, for convenience, take as an example an existing line project in the Forest Service, "Breeding for Control of White Pine Blister rust." The director, as a result of his discussion with the innovator, decides that additional research along this line is justified, even though work is being done on the problem by research workers at other laboratories, academic and federal.

Depending on the background of the director and the innovator, they might first talk with a forest economist, a forest geneticist, or a forest pathologist. The immediate reaction of any one of these gentlemen probably would be to narrow the proposal. The forest pathologist might wish to devote all of his effort to searching for strain variants of the pathogen. The forest geneticist might propose a breeding program and ignore completely any possibility that the pathogen might vary. The forest economist, realizing that the actual conduct of a breeding program was completely beyond his capability, might modify the project to a study of the economic impact of the disease on the production potential of different white pine sites.

If the director is lucky enough to consult an extrovert first, his consultant might immediately recognize that the scope of the problem was beyond his ability to handle alone. The research worker dubbed by the director as the project leader would logically seek to interest qualified colleagues in the project and obtain their collaboration. Failing to stimulate interest initially, he might then seek funds internally or externally, sufficient to permit the organization of the research team he envisions. If the research director can assign additional funds to the project, this in itself might stimulate interest among colleagues whose initial reaction was but lukewarm at best.

If unable to find colleagues willing to cooperate (usually their plea is lack of time) or funds internally, the project leader in an academic institution is free to seek supporting funds elsewhere. The sources of these are many and varied and certainly beyond the scope of this presentation. But the basic point is that a faculty member at an academic institution has this prerogative; a prerogative not usually associated with research positions at federal laboratories and private industry.

Let me assure you that individual research workers are the initiators of grant requests. Rarely do administrators write up research proposals.

Deans and Directors, never; Department Chairmen, hardly ever. Even major grants from NSF and Public Health Service such as the Health Service Advancement Awards currently being made to major institutions are not written by administrators. An administrator may decide that his institution will go after $500,000 or $5,000,000, but the job of writing the proposal is assigned to a single individual or to a committee composed of research workers. The administrator may review and revise the proposal but he actually contributes little to its originality and actual content.

What is meant by a research team? Frankly, I don't know. I see many differences between research teams as organized in academic institutions, and those organized in industrial and federal laboratories. In an academic institution the project leader may be the only professionally trained individual devoting any appreciable portion of his time to a given project. Other members of the team will be technical, experimental and laboratory aides with varying degrees of experience, graduate students, and professional colleagues who have indicated their willingness to collaborate on the problem but whose contributions more often constitute lip service than actual effort.

With the size of research grants increasing, more and more proposals for grants include provision for post-doctoral fellows, and other individuals with full professional training. With such individuals included in the team within educational institutions we approach the situation found in commercial and federal laboratories. The leader of a grant supported project at a university usually has more control over other personnel working on the project than do project leaders in other laboratories. The project leader has obtained the funds by his own effort, and they have been assigned to him for a specific project. He finds and recommends for employment all those working with him on the project. Most important, the positions involved are without tenure and can be terminated at almost any time and certainly upon the renewal date for the grant. Thus, the project leader has considerably more authority in controlling personnel supported by grant funds than he could possibly have over his peers (who have tenure) or subprofessional help paid from appropriated state funds who have permanent appointments.

There is another significant aspect of research and research teams at academic institutions. Most scientists have both teaching and research responsibilities. The department of Plant Pathology at Washington State University has nine individuals located at Pullman paid from state funds. The effort of 2.58 faculty members is assigned to teaching, 1.50 to extension, and 4.92 to research. The fractions of an individual's time assigned to research range from 35 to 60%. There are 17 research projects currently active and funded that are directed by 8 of these 9 individuals. This figures out to 0.29 of one professional man's time per project. Thirteen half-time research assistants work on these 17 projects or an average of less than one-half man per project. Five technical aides are available - some assigned to a single project, others split between four and five projects. This adds another 0.29 man year giving 0.96 man year, or less than one full-time equivalent of all personnel, per project. In that two or three individuals do work on each of these projects, perhaps one can speak of a team working on each of them. Nevertheless, there isn't very much total manpower per project and, hence, some might question whether this actually constitutes a team approach to research.

A considerable amount of nonproject research also is conducted and supervised by these same individuals. A project is not set up unless the problem being investigated requires upwards of 10% of the principal investigator's time.

Eight of the nine individuals located at Pullman have fractions of their time assigned to teaching. These fractions, in my opinion, are unrealistically low. Supposedly they provide for all formal instruction and all thesis research. Those graduate students who are also research assistants normally write theses in connection with some aspect of the project to which they are assigned. However, a number of graduate students perform thesis research on subjects that are not the basis of experiment station projects. While the student has the primary responsibility for investigating a thesis problem, one faculty member always serves in an advisory capacity, or, depending upon the student's capability, in a more or less supervisory capacity. Two or three other professors serve on the thesis committee. Again the question arises, does one graduate student devoting approximately one-third of his total effort to a particular project and one faculty member devoting a small percentage of his time and the occasional advice of two or three other individuals constitute a research team?

One might at this point conclude there is little team research in universities and that administrators play a minor role in determining what projects will be activated and the constitution of the teams that work on these projects. Actually, the point I've tried to make is that in most instances our teams are small and understaffed.

There is no way to avoid having each member of a university faculty responsible for several projects. Each professional scientist can contribute to, and supervise, the work of two or three projects provided he has enough hands to work with him. This is the area in which research teams are weak at universities. A well-trained and experienced professional man should be able to supervise three or four individuals. Included in the individuals working with each professional should be technical aides (1-3), research assistants (up to 3 or 4), and several part-time employees. There just isn't enough money to build such teams on state allocated funds alone. However, with upwards of one-third of a university's total funds now coming from grants, there is increasing opportunity for each individual research worker, primarily through his own efforts,to build the team he needs to handle the projects for which he is responsible.

I do not want to leave an entirely negative impression of research teams in universities. In addition to the main campus of Washington State University there are four major research centers located elsewhere in the state. Each of these is staffed with 30-40 professional men and, surprisingly, more subprofessional positions per professional are available than at the main research center. Scientists at these research centers are assigned 100% to research. They do not have an assigned teaching or extension responsibility, although many do undertake such responsibilities voluntarily.

Certainly within Washington State University the best development of the team approach to research is to be found in these branch stations. Several of our pathologists, for example, head teams of 3-5 individuals all working on a single or, at most, two related projects.

There is another aspect to this question of what constitutes a research team. If you consider projects as the basis for organizing research teams, the latter may be very small. If, on the other hand, we think in terms of programs as the basis of organization, the size of the team increases proportionately. We have a very sizeable research team at Washington State working on wheat. The group includes cereal pathologists (3 state, 4 federal); breeders (2 state and 2 federal); agronomists; irrigation specialists; ag engineers; chemists and biochemists; entomologists; and perhaps others that do not come immediately to mind. From this standpoint, we have a comprehensive team of research workers involved in a coordinated and most productive program.

Well, now what about the role of the administrator? The administrator must first of all, recognize the potentials of the professional men on his staff. Some individuals can effectively divide their efforts between two or three projects; others are most effective if all of their efforts are directed to a single project. Some individuals by their interests are better adapted for basic research; others for attacking applied projects. Some can supervise the efforts of a relatively large group, whether working on one project or on several. Some command the respect and cooperation of their professional colleagues and can get good cooperation. Others work best as "lone wolves" and can neither effectively seek good cooperation from others or be good cooperators on projects supervised by others.

The administrator has to recognize both the capabilities and limitations of his faculty and, through both advice and directive encouragement, seek to obtain the utmost productivity from each member of his staff. By logical allocation or nonallocation of funds he can encourage or discourage certain lines of endeavor. By urging the members of his faculty to seek outside support for those projects that he believes should receive additional support he can lead the total program towards those problems he considers need the greatest emphasis.

The administrator must constantly consider de-emphasis or termination of existing lines of activity in order to make room for new endeavors. There are always more problems demanding research attention than can be attended to. Much of the routine testing and demonstrational type of research must be eliminated. Seed and soil testing, analysis of products, production of foundation seed stocks and purebred animals for breeding purposes, and many similar activities previously performed by experiment stations have evolved beyond the research stage and are now a service type operation. As such, they should either be eliminated from the programs of university research centers or handled by nonresearch personnel. Their cost should be fully reimbursed on a fee basis - with the fee high enough to cover all indirect costs that are involved in providing such services. The administrator must do everything he can to free the scientist for fundamental tasks.

The administration has the primary responsibility for long-range planning. Each individual research worker is wrapped up in his current project(s) and often is so engrossed in day-to-day details that he loses sight of his own original goals. Through annual reports and work plans for the next reporting period, the research worker is provided an opportunity to review and plan ahead for his own program.

If the annual reports mentioned previously are worth the scientist's time to prepare, they are certainly worth the study necessary to determine what progress is being made and where difficulty is being encountered. Each report must be reviewed by both department chairmen and the research director. The administrators, after review of these reports, must call the attention of the research worker to any deviations from original objectives. If revision or termination of a project or the initiation of a new project is necessary, these points must be brought to the attention of the project leader.

Long-range objectives are most difficult to establish, but we must accumulate the necessary information that will help us to predict the research needs of forestry, agriculture and industry 10 and 20 years from now. Evaluation is an essential step in identifying worthy programs, determining their needs and opportunities for their improvement.

The higher up the administrative ladder an individual is, the more difficult time he has maintaining close personal contact with all scientists on his staff. When these individuals are located 200 and 300 miles from home base, these problems are increased. A departmental chairman at Washington State University is responsible for the total research program within his department. He must determine when changes of program are necessary. He recommends employment of appropriate staff members to accomplish the desired program insofar as funds and facilities will permit. Each department chairman must work closely with the Director of Research to give proper consideration to the long-range goals of the entire center.

The quality and quantity of the research program is influenced in large measure by the characteristics of the scientist and the environment in which he works. From an intellectual standpoint, the scientist's principal motivating forces should be:

1. The desire to know and understand some mechanism or phenomena;

2. The desire to be able to control the phenomena;

3. The desire to be recognized by his professional colleagues as a creative scientist and a productive scholar.

These motivations must be safeguarded by sound administrative management and by an equitable reward system. A scientist has little incentive to excell if he knows that mediocre performance receives the same reward as outstanding performance.

Now let us consider how we build research teams for cooperative research. Basically this amounts to finding the scientists who have professional interests in problems of interdisciplinary nature. This requirement is the same whether one is building a cooperative team at a research center of one university; on an inter-university basis; between federal and state agents; or within industry or federal laboratories. We must bring together scientists interested in production phases, and geneticists, breeders, pathologists, economists, and chemists in order to develop balanced programs aimed at major problems of long-range importance. In selecting such teams I emphasize again the necessity of bringing together only those people who have an interest in, and are challenged by, the specific problem in question.

Research is most productive if done on a voluntary basis - voluntary, that is, in terms of the scientists visualizing the problem and formulating objectives and procedures to guide their own activities.

THE TEAM APPROACH AND THE RESEARCH SCIENTIST

Neil E. Martin

Team approach to research is a term to which each of us can attach a connotation based upon our experiences or contact with a team. Some of us have a mental picture of a group of white clad, pipe smoking, bearded, and bespectacled individuals in one congested laboratory. Some of us are also sure that somewhere in this picture of unorganized chaos there must be a quarterback, a timekeeper, and a scorekeeper. Whatever the picture, we are sure that the individuals are highly coordinated and display organized movements and that all are involved in this thing called research.

We are aware that the team derives its organization, and therefore its team effect, from within in the form of a board of directors or from without in the form of a project leader or similar director, or policy maker. Because of many diverse visions of what a team in research might be, a definition is necessary as a base for further discussion. In order to fit all of your connotations we can define a research team as a group of scientists deployed to solve a specific problem. However, one can extend the definition of team to include participants with compatible interests. In this light, it becomes extremely difficult to recall an instance that is not the result of team effort and consequently equally difficult to delimit a team on the basis of geography, funding agency, or discipline.

Due to the lack of a descriptive definition, perhaps we can better understand the team approach by looking from the inside through the eyes of a participating scientist. Such an analysis is possible due to the realization that a team and an individual are inseparable. The result is a mutual reflection of individual efforts we group into a team effect, and a team effect that satisfies the individual. To fully appreciate the interaction of the team approach and the research scientist, we will review how certain aspects affect the individual and, consequently, the team. Four aspects seem conclusive: 1) the training of a scientist, how his background prepares him for team participation; 2) the interaction of the facets of a problem and the scientist; 3) stimulation of productivity; and 4) coordination of efforts through communication.

Training of the scientist

The scientist has spent years preparing to be an expert in a narrow area of one of many areas of research. As a student, great personal effort was directed toward a solution to one problem, and submitting all data and conclusions in written form to a board of review. All this tends to mold a student into an individual with extremely narrow immediate application. At present, it is very difficult for an individual to be fully competent in two areas of research and this may become more difficult, if not impossible, in the future. The complexities of science and the depth to which a problem can be pursued with modern methods dictate that this trend toward a specialist will continue.

A highly specialized scientist is a vital component in a team. The fact that he is a specialist indicates that he is qualified to contribute his share to a coordinated research effort. He has learned to recognize the existence of a problem, to communicate in both written and verbal form, and to evaluate and recommend.

Although narrow in application, he has been exposed to other academic disciplines and has acquired a conversational level of achievement in areas related to his special area of research. Participation in seminars has provided the opportunity to evaluate the research of others and to formulate meaningful questions. During the period of graduate study the individual absorbs and practices the tactics of those with whom he has direct contact in the classroom and laboratory. How well he has learned and how well he can apply these talents can be evaluated only with time. Training does not cease with the granting of a degree. The past becomes a guideline upon which to formulate tomorrow's decisions. Through maturity that comes with experience the scientist strives to enhance his integrity, to develop discourse that enables exploration into the complexities of a problem, and to perfect methods, procedures, and processes of his trade.

The facets of a problem

Before we can appreciate what a scientist in a research team must accomplish, we must recognize the diversity and capability of the object of study. A disease in one of its many exemplifications is to be expected in a forest population. The requirements and tolerances closely approximate the environmental complex in which it exists. These requirements and tolerances are basically determined by the genetic constitution of the organisms involved. No species of pathogen can maintain membership in a microclimate unless it is provided with at least its minimum requirements on one hand and provided that its tolerances are not exceeded on the other (Roth, WIFDWC, 1966, p. 10). Because it is likely that effective control will be based on knowledge of such requirements and tolerances it appears as though an effective research team, from the team standpoint, would be one composed of experts in all facets of biological and related areas of research, namely, genetics, ecology, chemistry, meteorology, soil science, silviculture, etc. It is logical that the team coordinator should actively seek qualified personnel in research areas of high priority.

To a scientist the facets of a problem are usually limited to specific problem areas, rather than encompassing the over-all problem. Within the team he seeks an understanding of the over-all problem so that he can better evaluate his own problem area in proper prospective to others. He finds security in a well thought out approach to the over-all problem that integrates all research areas. Armed with this information the scientist can assign priorities to the facets of his specific problem area and request their acceptance on the basis of their harmony with related areas of research on the over-all problem.

Stimulation of productivity

Now that the scientist-in-the-rough represents an expert with potential limited only by his own initiative, what are the conditions most suitable for productivity and at the same time will satisfy the scientist's basic humanistic needs? The scientist seeks the freedom to exercise original thinking. He also seeks the freedom to utilize his specific talents, the freedom to be independent, yet have the freedom to satisfy a desire to aggregate with others, and the freedom to be active in formulating decisions (Pelz 1967, Science). These freedoms are a form of security to the scientist because they represent needs or goals and at the same time, a challenge, because they have no limits.

The scientist has been trained to observe and then integrate these observations, a process we term original thinking; a process that develops a discourse toward a conclusion and eventual solving of a problem. The scientist needs and demands freedom within the team to exercise his mental facilities. Without this freedom he would no longer be a scientist but a technician.

Application of specific scientific know-how gives the scientist confidence that he can make a contribution to the team effort and thus he achieves a feeling of stability and security. At the same time, the specific problem has no finite limit and absorbs the scientist's full freedom of exploration, thus presenting itself as a challenge.

A high value is placed by the scientist upon independence. Throughout the training period the scientist has been conditioned to associate reward with individual effort. The graduate program does little, if anything, to encourage team participation in solving a common problem. The scope of an instructional staff and the requisites of diversified funding agencies ensure the impracticality of any attempt to coordinate graduate student research efforts. Here the impracticality stops. Coordinated effort becomes the byword if not the first commandment to progress whether it be in a government organization or private industry. To measure progress is to measure the rate of creativity. Therefore, the team must provide reward for independence and at the same time integrate individual efforts.

Since childhood an individual is exposed to the directives of policy makers. As one increases in age and experiences, the more capable he is of making a sound decision and the more one desires the freedom to take an active part in policy making. The scientist is concerned about policy that affects himself and his colleagues and is more amenable to directives he has discussed and understood. To discuss one's opinions with persons in several positions is to run the risk of criticism and disruption. However, the more sources there are involved in decisions, the greater is the likelihood of challenge. The interaction of scientist and team should provide the scientist with the opportunity to initiate, discuss, and establish policy that harmonizes with the needs of the team.

<u>Coordination of efforts through communication</u>

Communication with fellow colleagues presents a challenge to a scientist in the form of direct evaluation of hypothesis and conclusions, in other words, a challenge and a prod. Other members of the team are actively requesting suggestions, evaluations, and reviews of everything from hypothetical models to manuscripts. The scientist in turn is stimulated through an underlying common interest to produce a solicited response that represents considerable mental effort rather than a shallow response associated with apathy.

The scientist desires a similar freedom within a team, but not restricted to the team boundaries. The willingness to work together toward a common objective and the capacity to integrate results is, of course, a key factor in productive interdisciplinary study, a factor that relies on communication as the coordinating machine. However, high performance of an individual is frequently associated with the frequency of conferring with others of compatible interests. A desire on the part of scientists for freedom toward increasing such contact, outside the team sphere, is exemplified in the numerous professional society meetings on the regional as well as the national level and in the establishment of informal discussions such as we are attending today. The scientist's scope of communication does not stop here but extends beyond a few or a few hundred colleagues and encompasses sources of information over an entire nation or nations.

Within this broad context several recent or probable forthcoming developments in the field of communications appear likely to affect research workers. However, their greatest impact will vary according to the type and stage of the research program where information retrieval is important. The formation of a few large information centers, equipped with large electronic memories containing references to many subjects and connected through rapid transmission facilities to a large network of libraries, appears highly probable.

The laboratory at Moscow is attempting to initiate a system of information retrieval of material vitally important to pathology. This can become a part of a larger system such as that proposed by the National Research Council for a biological sciences information system. This, in turn, may integrate with the system of communication under study by the International Council of Scientific Unions. [Scientific Research 2(4):27]. The three national libraries of the U.S. have formed a task force to plan the establishment of a national computerized catalog information center. The Library of Congress and the National Agricultural Library are conducting an automation study. The proposed data bank would serve as a reference source for all libraries in the Nation and could be used to produce indexes and bibliographies for other libraries. In the not too distant future, it should be possible not only to extract references pertaining to specific subjects but also locate a library from which the publication may be obtained [Science 157(3785):172].

Summary: A productive team is made up of highly trained individuals that have similar sources of motivation but are in constant intellectual conflict. Characteristics of an individual that make a coordinated team effort possible are: 1) be trained in a specific area; 2) have integrated knowledge of related areas; 3) have interests compatible with the over-all problem being emphasized, and 4) be able to communicate.

Literature Cited

1. News in Brief. 1967. National Libraries Coordination. Science 157:172.

2. Pelz, D. C. 1967. Creative tensions in the research and development climate. Science 157(3785): 160-165.

3. Roth, F.L. 1966. An ecosystem approach to forest disease prescription. WIFDWC, p. 10.

4. The Science World. 1967. Life sciences information group established by NRC. Scientific Research 2(4):27.

THE TEAM APPROACH AND THE LAND MANAGER

Benton Howard

You have heard Gardner Shaw and Neil Martin. They have ably and skillfully presented research problems and approaches. Now it's up to me to give another viewpoint. As you know I'm not a researcher, a pathologist, an administrator, nor a land manager; technically a forester, and certainly some sort of a "bastard" that associates with all four. Tries to understand, interpret, extrapolate, and when necessary prostitute science. All in the interest of forest management.

Let us look at the definition of a team. The book says, and I quote: "Team ... 2. Two or more horses, oxen, etc. Harnessed to the same vehicle or plow ... 4. A group of people working or playing together ... . V.I. (verb intransitive) ... 2. To join in cooperative activity ... ." Now to "teamwork," and again I quote the book. "N.1. joint action by a group of people, in which each person subordinates his individual interests and opinions to the unity and efficiency of the group; coordinated effort, as of an athletic team."

Please note "joint action" and that "each person subordinates his individual interests and opinions to the unity," and so forth. This does not and must not mean that the individual's thinking, imagination, nor integrity are impaired. The team approach does demand that goals be accepted, and that progress along generally agreed upon lines must be achieved.

Research administration, Research Pathologist, Control Pathologist, Land Manager--each of these units has its own team. Each has its objectives. Together they make a master team which has the over-riding and controlling goals.

The objective of any team is to reach its goal. To do so means that the goal must be defined. So who is to define the goal? Again I say that this should be a team effort. Too often goals have not been adequately defined. We have had research for research's sake, for the sheer pleasure of doing it, the intellectual exercise, and so on. Much of this has been the manager's fault. He has not participated nor defined his needs nor clarified his thinking, nor been demanding. He has been too content to live in the present, reluctant to face the future and only too willing to abdicate his responsibilities for decision making to Research.

For example, much research and not just in forest pathology, has and is being done in old-growth problems long past any reason. By the time the answers are known the only old growth will be in "museums;" i.e., parks, wilderness, etc.

This can be avoided. The master team can define goals, can program research, can execute research, can test and adopt research, and can reach the goal.

Basically our goal is well managed forest lands with a minimum loss of values, be them economic or aesthetic.

Each team has its proper place and I envisage it like this.

The research administrator team does the programming, the budgeting, the coordination. It outlines approaches, reviews progress, and provides the means. It adjusts the efforts among research teams.

The research team does the research--they find the answers. I mean a multi-discipline team. Too long have we been too functionalized. The manager will no longer accept partial answers. The interdisciplinary approach is a must. The basic answers are developed by these teams.

The control team bridges the gap between research and operation--tests, extrapolates, adopts, adjusts, and writes the guidelines for the foresters. This team has one foot in research, the other in operational practices. He must understand both. He must have the ability to talk to both, and to both make sense. No easy task! His is the task of devising and developing--the practicability of the basic factors involved.

The land manager uses the end result. Putsresearch to practice. If each team has done its job well the research is used; if it hasn't, it won't be used.

It is just as blunt and bold as that. Even if the administrator has done his work, the research brilliant, it won't be used unless it is properly translated so that the land manager can understand it and benefit from it.

Obviously each "team" makes its contribution, and as such should participate in defining the goals.

The manager should know his need; the research administrator knows what can be programmed, time tables, funding; the research team must decide on the approaches, balance efforts, and be bold, imaginative, and sound. It must have a sense of urgency.

The control team has to be realistic, flexible, and adaptable, and above all be able to communicate.

The land manager must be objective, define his needs, participate in the decision making; in fact he should provide the impetus and leadership in decision making.

Jointly these teams can define realistic goals, proceed to secure answers, provide procedures and put research into effective use.

Anything less should be unacceptable.

PANEL IV. -- HOST RESPONSE TO INJURY BY PATHOGENS

Robert V. Bega, Moderator

CULTURE OF BLISTER RUST INFECTED WESTERN WHITE PINE TISSUE

A. E. Harvey

Use of the plant tissue culture method as a tool for phytopathological research is relatively new. Although the technique is still in its infancy, it represents one of the most potent tools currently available to basic plant research. In my opinion, this technique will be particularly useful for the investigation of culture methods and the host-parasite physiology of obligate parasites.

Theoretically, tissue culture technology provides the researcher with an opportunity to study the host, the parasite, or the host-parasite system where it can be manipulated, under controlled conditions, to satisfy research objectives. Potentially, this is an ideal system for investigating pathological and host response physiology of plant pathogenic organisms. Progress made toward these ends on the study of white pine blister rust serve as an example.

Cambial explants of western white pine, infected with blister rust, can be cultured on a medium containing glucose, calcium nitrate, magnesium sulfate, potassium phosphate (monobasic), ammonium sulfate, ferric sulfate, maganese sulfate, and one of three auxins--IAA, NAA, or 2,4-D.

Invasion of newly formed callus tissue by the rust fungus is generally slow. Frequently dense, felt-like, aerial mycelia with clamp connections are produced. Intercellular mycelia and haustoria are typical and direct attachments to the aerial mycelia are frequently observed. Occasionally mycelia invaded the medium, but showed no tendency to become independent of the host. Production of pycnial and aecial sori occurs in many cultures. Aecia sori form immediately following maturation of pycnia and frequently have deposits of pycnial fluid containing spores on their peridium. Thus far, aecial sori have not produced spores.

Host tissue, when proliferating at the maximum rate, is able to prevent the rust fungus from inhibiting growth significantly. However, host tissue generally grows in a cyclic pattern and during the slow (dormant) periods the rust can gain sufficient advantage to completely take over the callus.

This proliferation by the rust inevitably results in the death of both organisms. In order to sporulate, the rust must gain a strong foothold in the callus tissue. Sporulation will not occur until the host tissue has begun to decline as a result of the rust infection.

Precisely what occurs, at the biochemical level, resulting in the decline of the host tissue is not yet known. However, it is felt that the tissue culture method described presents an unusual opportunity for the study of host responses to the blister rust organism.

CHANGES IN WOOD PROPERTIES AT THE BOUNDARY OF <u>POLYPORUS AMARUS</u> DECAY POCKETS

W. Wayne Wilcox and Baxter J. Garcia

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Abstract

Incense cedar heartwood infected by the pocket rot fungus Polyporus amarus was subjected to micro-tensile testing and microscopical examination to determine the effects of the presence of this fungus upon wood substance outside of advanced decay pockets. There was a general tendency for the springwood of the annual ring nearest the decay pocket to have a slightly lower tensile strength than springwood from the same ring but at a distance from the pocket. However, the variability within specimens and between similar specimens appeared as great as the effect being measured. Microscopical examination revealed the presence of small, sparse bore holes and hyphae in approximately the same numbers inside and outside of advanced decay pockets, and in discolored regions considered incipient decay. A few bore holes and hyphae were found in specimens from what was macroscopically considered to be sound wood. Polarized illumination gave no evidence of the diffusion of cellulolytic enzymes from the hyphae in regions other than within advanced decay pockets, but the small numbers of hyphae and bore holes observed in decay pockets indicated that considerable enzyme diffusion might be necessary for decay to proceed. The data suggest that possible alteration of physical, mechanical and chemical properties in wood substance surrounding decay pockets need not be a significant factor for consideration in utilization of this material.

Introduction

Incense cedar (Libocedrus decurrens Torrey), while a minor coniferous species in terms of total volume, is destined to increase in importance as management of California forest land intensifies. This prediction is based upon this species' rapid growth rate, ease of establishment and abundance of regeneration, high decay resistance of the heartwood and favorable physical and mechanical properties, similar in many respects to those of redwood. Before intensive management of incense cedar is feasible the standing volume of mature and overmature trees must be utilized. Much of the volume of such trees is cull: as high a proportion as 1/3 to 3/4 of the total volume has been estimated (Wagener and Bega, 1958; Kimmey, 1950). Almost all of this loss is due to pecky rot caused by Polyporus amarus Hedgcock (Boyce, 1930; Wagener and Bega, 1958). It has been estimated that 75 to 100 per cent of the mature incense cedar trees are affected (Meinecke, 1914). To facilitate efficient utilization of the timber extensively affected by this fungus, knowledge is necessary of the changes it produces in the attacked wood. The tissue adjacent to decay pockets presently is being utilized in the manufacture of pencil slats and as a fiber source; However, it is not known whether the properties of this tissue differ from those of wood some distance from the visible degradation.

Microscope technology has advanced considerably since the last detailed studies of the effects that P. amarus infection has upon the structure and composition of incense cedar cell walls. Furthermore, techniques have been devised for testing certain mechanical properties of small quantities of wood tissue. This study was undertaken to apply some of these techniques to incense cedar heartwood infected with P. amarus in order to increase the knowledge about this material which is necessary for its efficient utilization.

Literature Review

P. amarus is one of a small group of decay fungi which produce their deterioration in discrete pockets. Little is known about the mechanism by which such pockets are delineated. It has been hypothesized that the limitation of decay to pockets by P. amarus might be due to the sparse distribution of hyphae between pockets (Hubert, 1931) or variation in chemical or physical properties of the wood or the fungus (Boyce, 1920; von Schrenk, 1900).

Reported observations appear to have considered each decay pocket as a separate entity, rather than as a portion of a typical heartrot column. The boundary between decay and sound wood appeared very sharp and without gradation (Wagener and Bega, 1958; Boyce, 1920; von Schrenk, 1900; Harkness, 1879). Hyphae were common within the pockets but were rare outside the pockets (Hubert, 1931; Boyce, 1920; Harkness, 1879). Sometimes hyphae were rare both in advanced pockets and in the wood surrounding the pockets (Boyce, 1920; von Schrenk, 1900), or abundant only in slightly decayed wood and its surrounding tissue (Boyce, 1920).

The incipient stage of decay has been described as a faint yellowish-brown discoloration occurring in patches but with the wood remaining firm (Wagener and Bega, 1958; Boyce, 1930). The color gradually deepened and the wood became soft (Wagener and Bega, 1958; Boyce, 1930; Boyce, 1920).

Microscopical investigations revealed that, in advanced stages of decay, cellulose had been decomposed while lignin remained in the wall structure (Boyce, 1920; von Schrenk, 1900). Wood in advanced stages of decay, while lacking in hyphae, had numerous bore holes indicating that hyphae had been abundant (von Schrenk, 1900). Slightly decayed wood and surrounding tissues contained abundant hyphae (Boyce, 1920). However, Harkness (1879) found no microscopic evidence of the passage of hyphae through the apparently sound wood between decay pockets, and Boyce (1920) and Hubert (1931) found hyphae to be very sparse in this region. Hyphae exhibited no preference for pits in their passage from cell to cell (von Schrenk, 1900; Boyce, 1920), and formed bore holes in all directions, in either springwood or summerwood (Boyce, 1920). Hyphae were rare in ray cells and did not fill the lumina of the tracheids which they occupied (Boyce, 1920). Hubert (1931) found hyphae to be rare in all areas of wood tissue. The hyphae often were constricted when passing through the cell walls and bore holes were small (Boyce, 1920; Hubert, 1931).

Materials and Methods

Properties of the wood tissue adjacent to decay pockets were studied in two ways: micro-tensile testing and microscopical examination using various optical systems. Sound and decayed samples of freshly felled and sawn incense cedar heartwood were treated with about 10 ml formalin in a plastic bag for transport and then were kept in frozen storage at about -18°C until ready for use.

For micro-tensile testing the samples were thawed and sawn tangentially until small pockets were encountered which had curvature sufficient that test strips could be obtained with the center span closer to the pocket than either of the gripping ends. Blocks then were sawn approximately 1/2 inch wide in the tangential plane, 1-1/2 inches deep radially and 2 inches along the grain. A similar block was prepared from wood 2 to 3 inches tangentially distant from the decay sample and including the same annual rings. Rectangular test specimens were prepared and tested, following essentially the procedures of Ifju (Wilson and Ifju, 1965), with nominal dimensions of .003 x 1 x 1.75 in. Actual width was measured microscopically and actual thickness was measured with a micrometer designed by Schniewind (1959). The samples were tested green on a table model Instron with a gage length of 1.15 in. and a cross head speed of 0.01 in. per min. Constant gripping pressure of 25 p.s.i. was applied by means of rubber-faced pneumatic grips in order to minimize grip failures. The moisture content of the samples was maintained by transferring water from an eyedropper to them by means of surface tension. Because of the thinness of the summerwood in incense cedar it was excluded from the sample sections. The springwood samples were identified by a percentage scale with reference to their position within the springwood portion of the ring only, with 0 beginning at the boundary between the springwood and the previous year's summerwood and 100 occurring at the transition to summerwood within the same ring. Tensile strength was expressed as the maximum load to failure in pounds per square inch of cross-sectional area.

Samples for microscopical examination were thawed and sawn to a thickness approximately 1/4-inch along the grain. Blocks approximately 1/8-inch square were carefully split from the boundary of advanced decay pockets, from within yellowish mottled areas presumed to be incipient pockets and from apparently sound wood taken from specimens which contained no decay pockets. The small blocks were fixed in FAA (Sass, 1958) and embedded in celloidin (Wilcox, 1964a). The embedded blocks were microtome-sectioned at a thickness of 4μ. Sections were mounted unstained or after staining with safranin and fast green (Wilcox, 1964a). They were examined microscopically using bright field, phase contrast, and polarized illumination systems.

Results

The results of micro-tensile tests on 6 pairs of samples are shown in Figs. 1-6[slides]. In general, at least in the springwood of the annual ring nearest the decay pocket there was a slight tendency for the wood tissue near the decay pocket to be lower in tensile strength than wood from the same annual ring but tangentially distant from the pocket. However, a great deal of variability also was apparent. A test of the significance of the difference between the members of each pair of curves indicated that the differences in all but six of the pairs of curves were significantly different at the 1% level. Those in which the difference was not significant were Fig. 1, Ring 1; Fig. 2, Ring 2; Fig. 3, Ring 3; Fig. 4, Rings 1 and 2; Fig. 6, Ring 3 [slides]. However, it also is apparent that the difference in level from ring to ring was considerable and in some cases the strength in the decayed portion of the ring was greater than that of the sound portion. Grip failures occurred in 33 per cent of the sound samples and 48 per cent of the decay samples. In only several of the tests, however, did failure at the grips appear to have any effect upon the maximum load values; therefore all but these few values were retained in the data. In Figs. 1 and 3 [slides] the pocket was nearest the high end of the curves while in the other figures it was nearest the low end.

The results of microscopical examinations are shown in Figures 7-17 [slides]. In pockets with an advanced stage of decay, bore holes were very small and sparse; hyphae were rare (Fig. 7). Bore holes and hyphae also were found in the boundary zone (Fig. 8) and the tissue surrounding advanced pockets (Fig. 9). Bore holes, in fact, were more frequent in these regions than in material within the pocket, probably because they were easier to see due to the fact that the cell walls in the decayed tissue had undergone considerable distortion and structural collapse.

Most hyphae observed were approximately 1µ to 2µ in diameter (Figs. 7-9) while some were as small as 0.5µ (Fig. 10). Hyphae in a malt agar culture of P. amarus reached a diameter of 3 to 4µ. Most bore holes were approximately 2µ in diameter (Fig. 11) reaching a maximum of 3 to 4µ (Fig. 12). Their small size made bore holes very difficult to locate by techniques other than phase contrast illumination. The walls of cells in advanced pockets lacked birefringence (they appeared dark between crossed polarizing filters while the walls of cells in sound wood appeared bright), indicating that the crystalline cellulose had been disrupted or destroyed by the decay process. The boundaries of advanced pockets, while appearing very abrupt macroscopically, were found to include a zone of about 10 to 20 cells in which polarized light revealed a progressive loss of crystalline cellulose (Fig. 13). The boundaries of some pockets contained groups of cells which still retained some degree of birefringence surrounded by tissue which had lost all birefringence (Fig. 14). Bore holes appeared to run tangentially about as frequently as they ran radially, except that definite preferences for one direction or the other were apparent in different areas. Hyphae also were found running longitudinally within the cell lumina, but they were not numerous either. Bore holes appeared about as frequently in springwood as in summerwood but they were easier to locate in the summerwood due to the thickness of the secondary walls and smaller size of the cells. The hyphae appeared to have no preference for pits in their passage through the cell walls (Fig. 11).

Wood which contained no advanced pockets but had irregular yellowish, mottled regions was sectioned and examined. This material corresponded to descriptions in the literature of the early stages of pocket formation. However, because the discoloration often was irregular and not confined to discrete zones, it seemed doubtful that this could, in fact, be the irregularities in staining properties, when treated with safranin and fast green, which were similar to those of tissue from the boundary of advanced pockets and similar to changes observed in incipient brown rot in earlier studies (Wilcox, 1964[b]). This involved a greater tendency for the red to show in the secondary wall, turning the green to gray-green. Bore holes and hyphae were found and were similar in size and number to those in tissue surrounding advanced pockets, i.e., they were small and sparse (Fig. 15). Although hyphae and bore holes were as prevalent in this tissue as in the area of advanced pockets, there was no apparent change in birefringence of the cell walls, indicating that appreciable attack upon the crystalline cellulose had not yet begun.

Sections of sound wood, material without yellow mottling and some distance from any advanced pockets, contained almost as many bore holes as were present in the samples of early stages of decay (Fig. 16). When bore holes and hyphae were found in tissue other than that in advanced stages of decay there was no evidence, under polarized light, of any loss of birefringence in the material surrounding them (Fig. 17).

Discussion

The accuracy of micro-tensile testing is questionable, according to the procedures used in this study, in determining the effects of the presence of a decay pocket upon the properties of the wood substance adjacent to the pocket. Apparently the variability encountered, with respect to the testing methods and even between different specimens of wood, may be greater than the changes under observation. Examination of decay pocket boundaries under polarized light revealed a zone of progressive cellulose decomposition at the pocket boundary. Since tensile strength, as determined by micro-tensile tests, is very sensitive to cellulose content and chain length (Ifju, 1964; Kennedy and Ifju, 1962) it was presumed that a striking difference in tensile strength could be detected in the region near decay pockets. This was expected to be particularly true in springwood tissue since Ifju (1964) found that the relative loss in tensile strength due to cellulose degradation was greater in springwood than summerwood. In general, these data are consistent with such a hypothesis as far as the springwood band nearest the pocket is concerned. According to the information obtained from examination in polarized light, that is as far as it would be expected that an effect on strength could be measured. However, it would also be expected that a considerably greater effect would be noted, within a single springwood band, in the portion of the ring nearest the pocket. Such an effect is not apparent in these data. Furthermore, the range of differences between test and control values in second and third rings away from the pocket, is similar in magnitude to the difference between test and control values in first rings which yielded the expected results. In several samples the "decay" values even were consistently higher than the "sound" values. Another possible factor which may have been involved is that in moving tangentially away from the decay pocket to obtain the control sample, even though the same annual rings were sampled, the wood properties may have varied considerably.

The proportion of grip failures in this experimentation is considered too high. Wilson and Ifju (1965) reported a grip failure rate of less than 10 per cent. In preliminary tests the pneumatic grips, which allowed each specimen to be gripped with exactly the same force and without flexure during assembly, appreciably reduced the proportion of failures at the edge of the grip faces rather than at the mid-span of the specimen. It was still not possible, however, to refine the techniques to the extent that could produce on acceptable level of grip failure. Nevertheless, the range and variability of most of the values where grip failures had occurred appeared so similar to those mid-span failures that the grip failure data were retained as valid. The fact that grip failures were 1 1/2 times as frequent in the decayed specimens as in the sound may be significant in itself.

Although "necked-down" specimens (Schniewind, 1959) might have lowered the proportion of grip failures, rectangular specimens are considerably easier to work with and Ifju (Wilson and Ifju, 1965) found that rectangular specimens provided results which were more satisfactory than those from "necked-down" specimens without increasing the grip failure rate appreciably.

The microscopical results confirm that the yellowish, mottled areas in incense cedar heartwood can be considered incipient decay. However, apparently hyphae also may be present, and therefore incipient decay, where there is no macroscopical evidence of their presence, as indicated by the presence of hyphae in the samples selected as sound controls. But, the lack of any detectable effect upon the crystalline cellulose of the cell walls outside of the actual decayed tissue in advanced pockets is evidence that the difficulty in locating incipient decay should have little consequence in the utilization of incense cedar heartwood. Apparently, if cellulose decomposition had taken place at all, as the slight changes in staining properties suggested, it involved mainly the amorphous fraction.

In addition to the lack of evidence of significant celluloytic activity around hyphae outside of advanced pockets, there was no evidence of the action of celluloytic enzymes upon cell wall material immediately surrounding bore holes. This suggests that there was little tendency for cellulolytic enzymes to diffuse laterally from the penetrating hyphae. Conversely, however, the very small number of bore holes and hyphae in wood in any stage of decay, including advanced pockets, suggests that the decay process must be significantly dependent upon diffusion of cellulolytic enzymes to relatively great distances from the hyphae. This, along with the apparent widespread, although sparse, distribution of hyphae, would suggest that the mechanism of pocket delineation might involve, rather than a physical or chemical exclusion of hyphae from certain areas, an action which affects enzyme diffusion or activity. These two lines of evidence, at this point, appear contradictory.

Literature Cited

1. Boyce, J. S. 1920. The dry-rot of incense cedar. U.S. Dept. Agr. Bull. No. 871. 58 p.

2. ____________. 1930. Decay in Pacific Northwest conifers. Yale Univ. Osborn Bot. Lab. Bull. 1. 51 p.

3. Harkness, H. W. 1879. A foe to the lumberman. Pacific Rural Press 17(4): 49.

4. Hubert, E. E. 1931. An Outline of Forest Pathology. John Wiley and Sons, Inc., New York. 543 p.

5. Ifju, G. 1964. Tensile strength behavior as a function of cellulose in wood. Forest Products J. 14: 366-372.

6. Kennedy, R. W., and G. Ifju. 1962. Applications of micro-tensile testing to thin wood sections. Tappi 45: 725-733.

7. Kimmey, J. W. 1950. Cull factors for forest-tree species in northwestern California. U.S. Dept. Agr., California Forest Range Exp. Sta., Forest Survey Release No. 7. 30 p.

8. Meinecke, E. P. 1914. Forest tree diseases common in California and Nevada. U.S. Dept. Agr., Forest Service. 67 p.

9. Sass, J. E. 1958. Botanical Microtechnique. 3rd. ed. The Iowa State College Press, Ames. 228 p.

10. Schniewind, A. P. 1959. Transverse anisotropy of wood: A function of gross anatomic structure. Forest Products J. 9: 350-359.

11. Shrenk, H. von. 1900. A disease of _Taxocium distichum_ known as peckiness, also a similar disease of _Libocedrus decurrent_ known pin-rot. Missouri Bot. Garden Annual Rep. 11. 23-77.

12. Wagener, W. W., and R. V. Bega. 1958. Heart rots of incense-cedar. U.S. Dept. Agr., Forest Service, Forest Pest Leafl. 30. 7 p.

13. Wilcox, W. W. 1964a. Preparation of decayed wood for microscopical examination. U.S. Forest Service Res. Note FPL-056. 22 p.

14. Wilcox, W. W. 1964b. Progressive microscopical changes in wood caused by a white-rot and a brown-rot fungus. Ph. D. Thesis, Univ. of Wisconsin. 127 p. (to be published as U.S. Forest Service, Res. Paper FPL-70).

15. Wilson, J. W., and G. Ifju. 1965. Wood characteristics VI: measuring density and strength properties of minute wood specimens. Pulp and Paper Res. Inst. Canada, Tech. Rep. 423. 15 p.

REACTION OF LODGEPOLE PINE TO BARK BEETLES AND OTHER INJURIOUS AGENTS

D. M. Shrimpton

Blue stain fungi are inoculated into lodgepole pine (_Pinus contorta_ Dougl. var. _latifolia_ Engelm.) by the bark beetle (_Dendroctonus ponderosae_ Hopkins). The complex of blue stain fungi has been described (Robinson 1962). The total picture--insect, blue stain, tree interrelationships--has also been previously described (Reid and Whitney 1965), (Reid, Whitney, and Watson 1967). In this paper the response of lodgepole pine to inoculation with one of the blue stain complex (_Europhium_ sp.) will be described. It must be remembered throughout that the condition where the tree is fully resistant, in which neither insects nor fungi can become established, intergrades continuously with the nonresistant condition in which the insects become established and produce a brood and the blue stain fungi extensively colonize the sapwood of the tree.

Response by Resistant Trees

Resistance of lodgepole pine to invasion by blue stain fungi is effected by an initial flow of primary resin (oleoresin) followed by a gradual impregnation of the sapwood and inner bark adjacent to the wound with resinous substances. The zone of sapwood and inner bark impregnated with resins is termed the resistant reaction. The total process-oleoresin flow and then development of the reaction area and subsequent consolidation in the sapwood area--is termed the resistant response.

The reaction of resistant trees to inoculation with Europhium sp. is at a maximum from about 6 - 20 ft. above ground level. As this is the area usually attacked by bark beetles there is some advantage here for the tree. But the total response decreases in July, usually around the time bark beetles attack. This decrease in response occurs at approximately the same time as tip elongation ceases and just before the starch content of the stem decreases.

The form of the resistant reaction to inoculation with Europhium sp. has been described for trees 65 years of age and older (Reid, Whitney, and Watson 1967). The same reaction pattern occurs in trees approximately 15 years of age and younger. This pattern is evident even in portions of such trees having a single growth ring. It is also seen in 4- to 6-year-old seedlings.

After inoculation with Europhium sp. the fungus can be isolated from the region of the resistant reaction immediately adjacent to the inoculation and occasionally about half way to the margin of this reaction. Two years after inoculation, however, no fungus could be recovered from any part of the resistant reaction.

Extracts from both the resistant reaction and normal sapwood inhibited growth of Ceratocystis montia and caused a delay in the commencement of growth of Europhium sp. These effects upon the growth of each fungus were due to volatile components in the extract, the majority of which are terpenes.

The only significant difference observed to date between primary resin and secondary resin is in a somewhat greater percentage composition of terpenes and resin acids in the primary resin. Both types of resin contain a complete spectrum of heartwood substances. The resistant reaction is a zone of sapwood that has become impregnated with heartwood substances (secondary resin) including terpenes, resin acids, aromatics and waxes. The composition of heartwood substances in the reaction does not change with a change in the fungus used to inoculate the tree, but the rate of production alters with a change in fungal stimulant.

Response of Nonresistant Trees and Some Differences from the Resistant Situation

In contrast to the resistant situation fungi can be readily isolated from any part of the nonresistant reaction area. The fungi rapidly overgrow the reaction area, colonize large sections of the sapwood and the tree quickly dies. In spite of this very obvious contrast, the difference between the resistant and the so called nonresistant response is in degree only. Thus the nonresistant response is in point of fact an insufficiently intense response by the tree.

All lodgepole pines studied to date responded to Europhium sp. inoculation with an initial flow of primary resin but, in the case of nonresistant trees, this resin flow is negligible. All of these trees then deposit heartwood substances--secondary resin--in the tissues adjacent to the wound. The rate at which the nonresistant reaction develops is, however, much less than the rate of development of the resistant reaction. Also the concentration of heartwood substances in the nonresistant reaction is lower than in the resistant reaction. I am not yet sure whether some of the observed differences in percentage composition are significant differences in tree response or are due to fungal activity.

Literature Cited

Reid, R. W., and Whitney, H. S. 1965. Reactions in the stem of lodgepole pine to attacks by the mountain pine beetle and concomittant infection by blue stain fungi. Proc. of 13th Western International Forest Disease Work Conference.

Reid, R. W., Whitney, H. S., and Watson, J. A. 1967. Reactions of lodgepole pine to attack by Dendroctonus ponderosae Hopkins and blue stain fungi. Can. J. Bot. 45: 1115-1126.

Robinson, Robena C. 1962. Blue stain fungi in lodgepole pine (Pinus contorta Dougl. var. latifolia Engelm.) infested by the mountain pine beetle (Dendroctonus ponderosae Hopk.) Can. J. Bot. 40: 609-614.

PHYSIOLOGY OF BASIDIOCARP FORMATION IN *TRAMETES HISPIDA*

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Trametes hispida Bagl. is a wood-decaying fungus, attacking dead poplars and willows (2). In Colorado it also infects living apple trees and causes severe heart rots (3).

Isolates of this fungus were obtained from dead cottonwood in the Cache la Poudre Canyon west of Fort Collins, Colorado. These isolates produced basidiocarps when grown on potato dextrose agar, (PDA). But an incubation period as long as two months was required for obtaining basidiocarps. Attempts were made to stimulate a more rapid basidiocarp formation in culture.

(1) Effects of the orientation of culture plates. Culture plates containing PDA were inoculated with an isolate of *T. hispida* and maintained in the following positions: (a) upright, with agar-surface facing upward, (b) inverted, with agar-surface facing downward, (c) vertical, with agar-surface facing sideways. All plates were incubated at room temperature with alternating 12-hours light and dark periods.

After only five days of incubation, basidiocarps were formed in the plates kept in the inverted or in the vertical positions. All these sporophores were fertile, and basidiospores were formed and discharged.

The plates kept upright produced no basidiocarps even after two weeks of incubation.

The effects of the change of position of plates on basidiocarp development were also studied. After the formation of basidiocarps and before the discharge of basidiospores the cultures were changed from the inverted position to the upright position. No spores were discharged, and subsequent microscopic examination showed the transformation of basidia into vegetative hyphae.

(2) Effects of gravity. Culture plates were mounted on the vertical disc of a clinostat and incubated at room temperature under 12-hours alternating light and dark periods. The disc rotated at the rate of one rotation per every five minutes. Thus the culture plates were compensated for gravity. As a control, other plates were mounted vertically.

The vertical control produced basidiocarps after five days and spores were discharged. Plates on the clinostat, however, failed to produce any fruiting structure even after one month.

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(3) Effects of light. After inoculation, half of the plates were kept in continuous light and the other half were kept in the total darkness. A 100-watt incandescent light bulb was used as a light source and plates were kept approximately 36 inches away from the light source. All the plates were incubated in the inverted position.

The cultures in the light produced basidiocarps after five days. The plates kept in the dark did not produce fruiting structures. Inhibition of mycelial growth under the light was also found. Reduction as much as 10 per cent in mycelial growth was observed in the light.

To find out the minimum length of light period required for basidiocarp formation, the inoculates were kept under continuous light for the duration of 1, 6, 12, 24, 48, 72, and 96 hours and then placed into the total darkness. All the plates were in the inverted position throughout this experiment.

The plates placed in the darkness prior to 48-hour light period failed to produce fruiting bodies. After 48-hour light period, all cultures continued to develop basidiocarps even in the dark.

(4) Effects of aeration of the cultures. Culture flasks (1000 ml) containing PDA were inoculated, and these flasks were aerated with a small aquarium pump. The air was pumped through water for humidification and filtered through cotton before being introduced into the flasks. All the aerated flasks were kept in the upright position. As a control, flasks without aeration were kept in the upright and in the inverted position.

The inverted controls produced basidiocarps after five days. The aerated flasks started to produce basidiocarps after three weeks incubation. The non-aerated upright flask failed to produce fruiting structures even after a month.

(5) Effects of carbon dioxide. To facilitate the removal of carbon dioxide, a small sterile container with 1/10 N potassium hydroxide solution was placed in each culture plate. The plates were incubated in the upright position. The plates produced basidiocarps after three weeks incubation.

As the results of this study indicate, the period required for the formation of basidiocarps was much shorter when plates were inverted. This agrees with the results obtained by Cauchon in several species of wood-decaying fungi including *Polyporus cinnabarinus* and *Lenzites saepiaria* (1).

This stimulatory effect of the plate inversion and the failure for the fungus to form any fruiting structure under compensated gravity suggests the importance of gravity in the basidiocarp formation in *T. hispida*.

The exact mechanisms of how the gravity influences the formation of a fruiting body are not understood.

Aeration of the culture flasks stimulated the basidiocarp formation. This suggests that supply of fresh air or removal of injurious gas is a contributing factor. The observation that fruiting was also stimulated by the presence of potassium hydroxide solution in the culture plate suggests the importance of the removal of carbon dioxide.

The light was also found to be essential for the initiation of the fruiting. But which wave length of the light is most effective for this process is not known.

Literature Cited

1. Cauchon, R. 1963. Influence of plant orientation on fruiting in cultures of Hymenomycetes. Can. J. Bot. 41: 1275-1276.

2. Overholts, L. O. 1953. The Polyporaceae of the United States, Alaska and Canada. Univ. Mich. Press, Ann Arbor. 466 pp.

3. Smith, E. C. 1930. Trametes hispida, a destructive parasite in apple orchards. Mycologia 22: 221-222.

DWARFMISTLETOE ON UNDERSTORY RED FIR IN CALIFORNIA

Robert F. Scharpf

Red fir (Abies magnifica) is one of California's commercial timber species that is severely attacked by dwarfmistletoe (Arceuthobium campylopodum f. abietinum). Early investigators including W. W. Wagener and J. W. Kimmey noticed that small red firs were relatively free from dwarfmistletoe attack. But they did not study in detail the amount of dwarfmistletoe that did develop in stands of small trees.

This paper reports on a study to determine the relationships between tree height, tree age, and crown size on percentage of trees infected and on degree of infection.

Thirty-six plots, each 1 chain square, were established in red fir stands extending from the central Sierra Nevada into the southern Cascades of northern California. Five trees from each of 4 different height classes were selected on each plot, or a total of 20 trees per plot. Tree height, tree age, crown height, crown radius, and number of infections per tree were recorded.

Results of the study showed that the percentage of understory trees infected by dwarfmistletoe was directly related to tree height (Table 1), and--to a much lesser extent--to tree age (Table 2). The number of infections per infected tree also was directly related to tree height (Table 3). However, no marked relationship was evident between age of tree and number of infections (Table 4).

Target area, a function of tree height, was also investigated with regard to number of infections per square foot of crown surface area. The surface area of a cone was considered to most nearly represent the target area of a red fir crown (Figure 1). Marked differences in number of infections per square foot were not noted for trees of different heights (Table 5). This finding suggests that target area of the tree expressed as its crown surface area is an important factor that affects the amount of infection.

An "F" test was used to determine the significance of tree height and age on infection. In all cases, a function based on height alone was found to be adequate for predicting if a tree was infected or uninfected. The following model, based on tree height only, can be used for this purpose:

$$-.54 + .08\ (\text{height})$$

The discriminant function above classifies a tree as infected if the value is positive, uninfected if it is negative or zero.

The probability of proper classification was found to be 76 percent. For example:

For a 3-foot tree: $-.54 + .08(3) = -.30$

The probability is that, in about three out of four cases, any tree of this height growing under heavy overstory dwarfmistletoe will be uninfected.

For an 8-foot tree: $-.54 + .08(8) = +.10$

In this case the probability is that, in about three out of four cases, any tree of this height growing under heavy overstory dwarfmistletoe will be infected.

I concluded that percentage of understory red firs infected by dwarfmistletoe and number of infections per tree are more closely related to tree height than to tree age. With regard to tree height, target area of the tree crown--considered as the surface area of a cone--is probably the most important factor that determines the amount of infection.

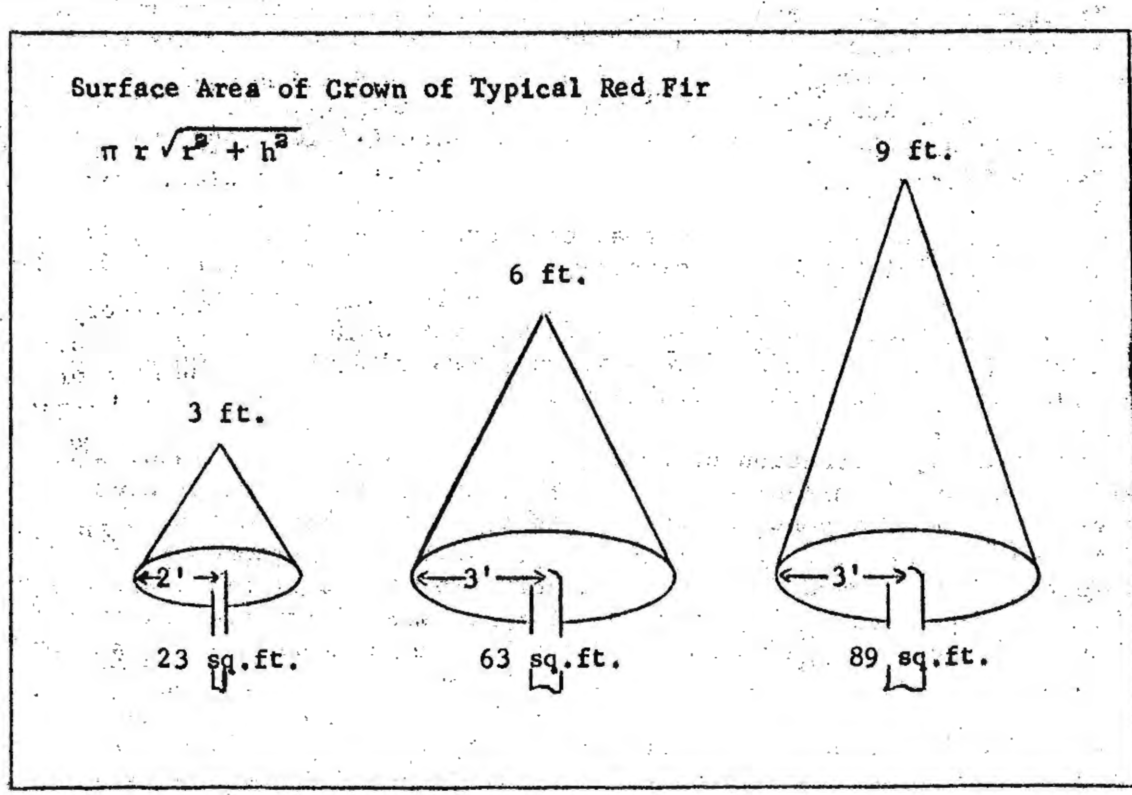

Figure 1.--Typical red fir crowns of given heights and radii and their respective surface areas.

Table 1.--Percentage of red fir understory infected by dwarfmistletoe (by height groups)

Height (feet)	Number of trees	Percent infected
0-3	182	07
4-6	187	30
7-9	187	65
10-12	184	79

Table 2.--Percentage of red fir understory infected by dwarfmistletoe (by age groups)

Age (years)	Number of trees	Percent infected
0-20	86	19
21-40	200	40
41-60	237	51
61-80	159	53
81-100	46	59
100+	12	67

Table 3.--Number of infections per infected tree (by height groups)

Height (feet)	Number of trees	Number of infections	Number of infections per tree
0-3	13	18	1.4
4-6	57	114	2.0
7-9	121	643	5.3
10-12	146	1,670	11.4

Table 4.--Number of infections per infected tree (by age groups)

Age group (feet)	Number of trees	Number of infections	Number of infections per tree
0-20	16	39	2.4
21-40	80	436	5.5
41-60	122	1,136	9.3
61-80	84	585	7.0
81-100	27	189	7.0
100+	8	60	7.5

Table 5.--Number of infections per square foot of crown surface area

Height groups (feet)	Total number infections	Number of infections per square foot
0-3	18	.18
4-6	114	.08
7-9	643	.12
10-12	1,670	.16

POWDERY MILDEWS OF OAK IN ARIZONA

Wilhelm G. Solheim[1]

In working with materials to be filed in the Phytopathological Herbarium of the University of Arizona, a collection of mildew on Quercus arizonica Sarg. was found. This collection was made by the late Paul D. Keener and Elizabeth H. Keener on 28 February, 1965, in Madera Canyon, Coronado National Forest, Santa Rita Mountains, Arizona. Mildewed oak was recollected in abundance at the same locality in March and April, 1967, by the author and several graduate students at the University of Arizona.

A study of the specimens revealed the presence of two most interesting fungi. One of these, including the material collected by the Keeners, was found to be a species of Typhylochaeta, a genus described by Ito and Hara in a paper by Ito (2) and is based on T. japonica Ito et Hara on Quercus glandulifera. The Arizona material is similar to T. japonica, but should probably be considered as a variety due to several points of difference. Hirata (1) gives the known range of T. japonica as Japan and China.

The second fungus, commonly found associated with the Typhulochaeta, belongs in the genus Saccardia. The latter genus was originally placed in the Erysipheae, but was transferred by Theissen and Sydow (3) to the Saccardiaceae of the Myriangrales based on the presence of an interascal stroma. Irrespective of the taxonomic placement of the genus, the disease produced by the species at hand is a typical powdery mildew and should be so designated. Two species of Saccardia have been reported on oak, S. quercina Cooke and S. martini Ell. et Sacc. The specific assignment of the Arizona fungus will require further studies.

Literature Cited

1. Hirata, Kohi. 1966. Host range and geographical distribution of the powdery mildews. Niigata, Japan. 472 p.

2. Ito, Seiya. 1915. On Typhulochaeta, a new genus of Erysiphaceae. Bot. Mag. Tokyo, 29: 15-22. Pl. II, Figs. 1-16.

3. Theissen, F. and H. Sydow. 1917. Synoptische Tafelin. Ann. Myc. 15: 389-491. pp. 433-447.

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PROMISING EARLY RESULTS OF PROGENY TESTING IN REGION 6 BLISTER RUST RESISTANCE PROGRAM

J. H. Thompson and H. H. Bynum

Blister rust has decimated much of the western white and sugar pine stocking in the Region. Attempts to control the disease by ribes eradication and antibiotic applications have proven generally unsuccessful. Accordingly, development of rust-resistant pines through applied genetics is recognized as the ultimate answer to the blister rust problem.

Western white pine producing areas have been divided into four geographical zones; sugar pine into two altitudinal zones. A goal of 400 rust-resistant candidate trees per zone or 2,400 in total is being pursued. To date, 400 western white pine candidates and 260 sugar pine have been selected - a total of 660 trees.

Expectations of the program include mass production of F_1 seed in five to six years. It is hoped that F_2 seed with a 10-15% increase in rust resistance over the F_1 seed will be mass produced in 20-25 years. F_3 seed with a further increase of 5-10% resistance is anticipated in time.

Progeny testing involves subjecting seedlings to three inoculations under artificially controlled conditions at ages 1-0, 2-0, and 3-0. Seedlings are distributed randomly within the inoculation chamber and infected ribes cuttings are inserted into the pots. About one year after each inoculation, seedlings are carefully examined for rust infection.

Seedlings which resulted from the 1962 seed sowing have been inoculated and examined three times. The results of this early progeny testing indicate an unexpectedly high level of rust resistance among some crosses. Of 30 sugar pine crosses, four resulted in greater than 60% transmittal of resistance. The highest was 81%. It is interesting that a single rust-resistant candidate parent is common to all four of these promising crosses. In addition to this parent, the general combining ability of three others appeared to be high.

Of 50 western white pine crosses, 16 resulted in greater than 50% resistance after artificial inoculations. Here again, several parent trees expressed a high general combining ability.

It is expected that final percentages of resistance will be somewhat lower than the figures mentioned above. It has been noted in fusiform rust-resistant breeding in the South that resistance from artificial inoculations does not always correlate closely with that from natural field inoculation. Nonetheless, these first progeny testing results are viewed with considerable optimism.

Because these early results indicate a somewhat higher level of resistance transmittal than is reported from the Region 1 rust-resistant program, there has been speculation as to the reason for the favorable results. There was some early doubt as to the efficacy of the artificial inoculations, but this was largely negated by the very low resistance transmitted by the majority of the crosses. Also, the three individual inoculations appeared to be equally effective because they resulted in comparable rates of infection by seedling age classes.

It is interesting to note that all of the western white pine crosses resulting in high resistance transmittal involved parent trees growing in south central to southern Oregon, or south of the 45th parallel. Crosses involving parent trees from north of the 45th parallel resulted in very low resistance transmittal at best. Perhaps there is a latitudinally related genetic or ecological factor which influences rust resistance or its transmittal.

Whatever the reason, early results of the blister rust resistance breeding program have been encouraging. It can only be hoped that future results will prove just as rewarding.

AN IMPORTANT UNEXPLAINED TYPE OF TREE FAILURE

Willis W. Wagener

I am not listed on the program, I have no prepared paper, and I am a very poor extemporaneous speaker. However, I do have some information about a particular type of tree failure that I consider important and which I would like to present to you. Your program chairman has kindly allowed me to do so.

At 3:20 a.m., July 2 of this year, on a windless night, a 39-inch diameter redwood fell in one of our State parks, instantly killing a 17-year-old boy sleeping directly in the line of fall, slightly injuring a companion sleeping close by, and flattening the personal car of a Navy dentist. About a month later, at 7:20 a.m., an overmature 5-foot diameter Douglas-fir fell under similar conditions, demolishing a tent in which a man and wife were asleep but fortunately causing painful but not serious injuries to them. Last summer a tanbark-oak fell during a windless summer night, seriously injuring a French student, temporarily in this country, who was sleeping in the line of fall.

These are three examples of half a dozen such cases known to have occurred in recreational areas in recent years. In every case, failure occurred during a windless night or early morning following a warm, dry day. More important, all of them occurred in the midst of the summer vacation season, when recreational areas are crowded. They occurred at night or in the early morning when most vacationers are in bed and unaware of what is happening. All resulted in deaths, injuries, or property damage. Accordingly they are much more important from the standpoint of consequences than failures occurring at any other time of the year.

Why did these failures occur when they did? As of now we do not know. So far as could be seen, the redwood had not changed appreciably since the last fire that had scarred the base in 1904. In the intervening 63 years the tree had been through countless storms without failing. Why then should it fail in the absence of wind?

I feel that the question must lie in the province of tree physiology and that cell turgor is involved. We need research to provide a basis for understanding this type of failure. Tree hazard is involved so the project could well comprise a joint effort. We certainly need something more than speculation to form a real understanding of this type of recreational hazard.

Now, just a couple of other comments bearing on the question of tree hazard. Tree failures are often basically of two types. These are:

(a) Rupture, in which the failure occurs primarily across the grain of the wood. It is usually initiated by the action of strong external forces on the tree, such as those accompanying very heavy storm winds. Failure occurs rapidly and frequently is preceded by a compression failure across the grain of the wood, consisting of the collapse of rows of wood cells across the grain because of excessive pressure, such as produced by high gusty winds. I am told by wood products specialists that compression failures were very common in connection with tree breakage accompanying the very high winds of the tropical storm of October 12, 1962 on the Pacific coast.

(b) Collapse, in which failure starts rather slowly and consists essentially of the buckling of a tree trunk at a weak point. The failure is usually initiated by some lateral force, such as a gust of wind, but not necessarily an extreme force. Typical cases involve trees with only a thin outer shell of sound wood, the rest having been destroyed by decay or some other destructive agency. Rupture of the outer wood consists typically of splitting with the grain rather than across it. A case of collapse is prima facie evidence that the tree was in a hazardous condition at the time of failure.

Cases of collapse are most readily recognized in relatively small diameter trees such as lodgepole pines. In large trees the two types of failure are less readily distinguished because simple mechanics dictate that the proportion of across-the-grain rupture of the wood will be greater in the case of collapse of large trees than in small ones. Also intermediate cases occur, in which failure is not definitely of one type or the other but is intermediate in causation and characteristics.

Switching to another point, I am somewhat worried over the dangers of the one-track-mind approach to hazard elimination in our recreational areas. Hazard reduction is almost certain to have multiple effects, and I feel that these should be clearly in mind before a hazard elimination program is carried out. Mistakes have already been made in such programs - as a result of ignorance of what really constitutes hazard in trees, or of the one-track-mind approach to the problem. In trees such mistakes are irreversible and posterity is the loser. I hope there will not be too many of them.

APPENDIX I

Active Projects, Old and New

(Addresses and affiliations for project leaders are given in membership list at end of these Proceedings).

A. Forest Disease Surveys--General

53-A-1 Forest disease surveys in Alberta, the Rocky Mountain National Parks, and Northwest Territories (R. J. Bourchier).

53-A-3 Disease survey in British Columbia (A. C. Molnar and W. G. Ziller).

56-A-1 Forest disease conditions in California (D. R. Miller).

57-A-1 Forest disease sampling studies (G. W. Wallis).

57-A-3 Exotic plantation studies (A. C. Molnar).

58-A-1 Taxonomic and biological studies of Ascomycetes and Fungi imperfecti (A. Funk).

58-A-2 Studies of native and introduced Populus spp. (W. J. Bloomberg).

63-A-1 Root disease survey of windthrown trees (D. R. Miller).

66-A-1 Development of statewide surveys to determine the impact of forest disease in Washington (K. W. Russell).

66-A-2 White pine blister rust incidence survey (H. V. Toko and D. A. Graham).

67-A-1 Fomes annosus survey in eastern Washington (K. W. Russell and C. H. Driver, cooperatively).
Objective: To determine the presence of F. annosus in the ponderosa pine type of eastern Washington and to determine if the increase in precommercial thinning of this type will increase the presence of this fungus.

67-A-2 Brewer spruce dwarfmistletoe survey (H. H. Bynum).
Objective: Survey the incidence and severity of dwarfmistletoe in Brewer spruce to determine whether control measures are warranted to protect this rare tree.

B. Non-infectious Diseases

66-B-1 Susceptibility of western conifers to air pollution (R. V. Bega).

C. Cone, Seed, and Seedling Diseases

53-C-5 Forest nursery diseases (W. J. Bloomberg).

60-C-1 Disease losses in redwood reproduction (J. R. Parmeter, Jr., and John Davidson).

60-C-2 Isolation of soil and root fungi (J. R. Parmeter, Jr.).

60-C-3 *Rhizoctonia* damping-off of nursery seedlings (H. S. Whitney).

64-C-1 Seedling diseases of forest nurseries in Hawaii (E. Trujillo).

D. Root and Soil Diseases or Relationships

53-D-2 *Poria weirii* root rot of Douglas-fir (T. W. Childs).

53-D-3 *Poria weirii* root rot of Douglas-fir (G. W. Wallis).

53-D-4 Characteristics and ecology of the root system of western white pine (R. G. McMinn).

53-D-8 Root diseases of forest conifers (W. W. Wagener).

55-D-2 Soil moisture in relation to the pole blight disease (C. D. Leaphart).

55-D-3 Mycorrhizal relationships of healthy and pole-blighted western white pine (R. L. Gilbertson).

59-D-1 Nursery and replant diseases (R. V. Bega and R. S. Smith, Jr.).

59-D-2 Root diseases--general (R. V. Bega, and R. S. Smith, Jr.).

61-D-1 Nursery and reforestation diseases (R. V. Bega, and R.S. Smith).

61-D-2 *Fomes annosus* (R. V. Bega, and R. S. Smith).

62-D-1 Diseases of marginal ponderosa pine, pinon and juniper in Arizona and New Mexico (J. W. Riffle).

62-D-2 Diseases of seed, seedlings and young trees (R.V. Bega and R. S. Smith).

62-D-3 The epidemiology and host parasite relations involved in the charcoal root disease complex of *Pinus lambertiana* (R. S. Smith).

63-D-1 Factors governing the rapid buildup of *Armillaria mellea* in conifer stands (O. K. Miller, Jr.).

64-D-1 The ecology of *Phytophthora cinnamomi* Rands in forest soils (E. Trujillo).

64-D-2 The host range of *Armillaria mellea* on subtropical species (E. Trujillo).

64-D-3 Armillaria root rot of Ceanothus spp. (J. Terry, J.D. Rogers, and C. G. Shaw).

65-D-1 The pathogenicity of Verticicladiella wagnerii to Pinus spp. (R. S. Smith).

65-D-2 Fomes annosus, is it a threat to immature stands in British Columbia? (G. Wallis).

65-D-3 Inter-relationships between root-pathogenic fungi, especially Fomes annosus, and bark beetle infestations in coniferous forests (F. W. Cobb, Jr., with J. R. Parmeter, Jr., R. W. Stark, D. L. Wood, and E. Zavarin).

66-D-1 Investigations on the occurrence and control of Fomes annosus butt- and root-rot of intensively managed stands (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).

66-D-6 Nutrient supplying potential of freshly fallen litter from alder, conifer, and alder-conifer stands (K.C. Lu and W.B. Bollen).

66-D-7 Comparison of Poria weirii survival in soil of a pure conifer stand to that of a mixed alder-conifer stand (E. E. Nelson).

66-D-8 Effect of nitrogen fertilizers on Poria weirii survival and populations of antagonistic microorganisms (E. E. Nelson).

66-D-10 Secondary metabolic products of mycorrhizal fungi (J. M. Trappe, and P. Catalfomo).

66-D-11 Characterization and classification of mycorrhizae of Douglas-fir and other northwestern conifers (B. Zak).

66-D-12 Fungi associated with deterioration of unsuberized roots of forest-grown Douglas-fir (B. Zak).

66-D-13 Field trials to test effects of thinning on spread and intensification of Armillaria root rot in young ponderosa pine (U.S. Forest Service, Region 6, Portland, Oregon).

67-D-1 Microbial and mycorrhizal factors inhibiting pathogenic root fungi in red alder-conifer associations of the Douglas-fir region (W.B. Bollen, K.C. Lu, and J.M. Trappe).
Objective: To determine the role of red alder and associated microflora in biological control of root diseases.

67-D-2 Selective culture of Poria weirii (E. E. Nelson).
Objective: To develop techniques for recovering Poria weirii from soil or wood habitats often containing other fungi more competitive than P. weirii on standard media. This, in turn, would allow more exacting determinations of presence or absence of P. weirii in experimental materials.

67-D-3 Effects of phenolic compounds on *P. weirii* and their location in forest trees and soils (K. C. Lu).
Objective: To determine the inhibitory effects of certain phenolic compounds on *P. weirii* and where they can be found in nature.

67-D-4 Investigation of the chemical thinning for control of *Fomes annosus* butt and root rot of precommercial western hemlock (K. W. Russell, and C. H. Driver, cooperatively).
Objective: To develop an alternate method of precommercially thinning western hemlock with reduced chances of further infection of the stand by *Fomes annosus*.

67-D-5 A survey of the coniferous root rots in Montana and northern Idaho (O. J. Dooling, and D. H. Brown).
Objective: To determine (1) Distribution; (2) Incidence of the various root rots in the major coniferous species in the area.

E. Foliage Diseases

53-E-4 Needle diseases of white pine (W. W. Wagener).

53-E-5 The Hypodermataceae of conifers in British Columbia (W. G. Ziller).

54-E-1 Rhabdocline on Douglas-fir (C. W. Waters).

57-E-1 *Elytroderma deformans* (C. W. Waters).

57-E-4 Life history studies of *Elytroderma deformans* (L. F. Roth).

62-E-1 Survey of needle cast diseases in the central Rocky Mountains (J. M. Staley).

62-E-2 Ontogeny and cytology of the species of Hypodermataceae of conifer in North America (C. C. Gordon).

63-E-1 Foliage disease of Christmas trees (J. D. Rogers).

64-E-1 Diseases of Christmas trees (E. Trujillo).

66-E-1 A study of moisture and temperature relationships governing the establishment of pathogenic conifer foliage fungi. (A cooperative project between the Rocky Mountain Forest and Range Experiment Station and Colorado State University, Department of Botany and Plant Pathology under direction of Dr. Nagayoshi Oshima).

67-E-1 Foliage diseases of conifers in the Southwest (P. C. Lightle).
Objective: To determine (1) the organisms causing foliage diseases of conifers in Arizona and New Mexico; (2) Their distribution, and (3) their relative frequencies.

F. Stem Diseases (Malformations, Witches-Brooms, Dwarfmistletoe, etc.).

53-F-3 Biological studies of dwarfmistletoes in Alberta (J.A. Muir).

53-F-4 Dwarfmistletoe of ponderosa pine (L. F. Roth).

55-F-2 Chemical control of dwarfmistletoe of conifers in California (C. R. Quick-1960).

55-F-3 Dwarfmistletoe of western larch and lodgepole pine (C. W. Waters).

57-F-1 Dwarfmistletoe of western larch, lodgepole pine, and Douglas-fir (C. W. Waters).

57-F-2 Some studies of Douglas-fir mistletoe and tissue culture of the host (L. Blakely and C. W. Waters).

57-F-4 Dwarfmistletoes on ponderosa pine and Douglas-fir (K. R. Shea).

57-F-5 Environmental and host-parasite relationships of dwarfmistletoes (J. R. Parmeter, Jr., and R. S. Scharpf).

57-F-6 Fungus diseases of dwarfmistletoes (R. S. Scharpf).

60-F-3 Losses resulting from growth reduction and mortality in lodgepole pine stands infected by dwarfmistletoe, Arceuthobium americanum Nutt. ex. Engelm. (J. A. Baranyay).

60-F-4 Impact of dwarfmistletoe on ponderosa pine (K. R. Shea and T. W. Childs).

60-F-5 Life tables for ponderosa pine and lodgepole pine dwarfmistletoe (F. G. Hawksworth).

60-F-6 Effects of dwarfmistletoe on yields in young lodgepole pine (F. G. Hawksworth).

61-F-1 Eradication and thinning studies for dwarfmistletoe control in infected stands in western larch, Douglas-fir, and lodgepole pine (E. F. Wicker).

62-F-1 Ballistics of dwarfmistletoe seeds (T. E. Hinds and F. G. Hawksworth).

62-F-2 Field cultures with dwarfmistletoe (F. G. Hawksworth and T. E. Hinds).

62-F-3 Taxonomy, hosts, and distribution of the mistletoes in the Rocky Mountains and the Southwest (F. G. Hawksworth).

62-F-4 Cytotaxonomic study of Arceuthobium campylopodum and A. vaginatum (Del Wiens).

62-F-5 Barriers to dwarfmistletoe infection in non-susceptible hosts (F. G. Hawksworth).

62-F-6 Effects of dwarfmistletoe on growth and mortality in immature ponderosa pine (F.G. Hawksworth, P.C. Lightle, and T.E. Hinds).

62-F-7 Ecology of lodgepole pine dwarfmistletoe (F. G. Hawksworth).

62-F-8 Ecology of ponderosa pine dwarfmistletoe (F. G. Hawksworth).

62-F-9 Resin disease of lodgepole pine dwarfmistletoe (F.G. Hawksworth).

62-F-10 Silvicultural control of ponderosa pine dwarfmistletoe (P.C. Lightle)

62-F-11 Physiological studies of seed of dwarfmistletoe of ponderosa pine (K. Beckman).

62-F-12 Physiological studies of dwarfmistletoe of ponderosa pine. A comparison of metabolites in host and parasite (L. McDowell).

62-F-13 Ontogeny of the aerial shoots of _Arceuthobium douglasii_ and _A. campylopodum_ f. _laricis_ (B. Jones).

62-F-14 A study of the factors influencing seed production of _Arceuthobium americanum_ on lodgepole pine (J. E. Nighswander).

62-F-15 Use of aerial photography in detecting dwarfmistletoe-infected lodgepole pine stands (J. A. Baranyay).

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

63-F-2 Epidemiology of dwarfmistletoes on firs in California (R.F. Scharpf).

64-F-1 Cytology and ontogeny of _Arceuthobium douglasii_, _A. americana_, and _A. campylopodum_ f. _laricis_ (C. C. Gordon).

64-F-2 Development and pathogenicity of Hypoxylon fuscum on northwestern species of alder (_Alnus_) (J. D. Rogers).

65-F-1 A study of the factors influencing reproduction and parasitism by _Arceuthobium americanum_ Nutt. (J. A. Muir).

65-F-2 The effect of dwarfmistletoe on growth of western hemlock (K. W. Russell).

66-F-1 Field tests on cultural control of dwarfmistletoe by understory destruction and by species manipulation (B. Howard, J. H. Thompson, and D. P. Graham).

67-F-1 Computer classification of _Arceuthobium_ (Coop. study with CSU) (D. J. Rogers).
Objective: To develop a computer-based classification for _Arceuthobium_ using morphological, anatomical, phenological, pollen, host, and other data provided by F.G. Hawksworth.

G. Stem Diseases - Stains and Decays

53-G-13 "Red stain" and decay of lodgepole pine injured by fire in Alberta (V. J. Nordin).

57-G-1 Loss factors for a continuous forest inventory in British Columbia (W. Bradshaw).

57-G-3 Significance of the black heartwood stains of yellow cedar (H. W. Eades and J. W. Roff).

57-G-4 Studies on Idaho wood-rotting fungi (R. L. Gilbertson).

57-G-10 Ecology of branch stub infection in aspen (D. E. Etheridge).

59-G-1 The influence of site on variations in red heartwood stain of lodgepole pine (J. E. Nighswander).

59-G-4 Red rot in young ponderosa pine (S. R. Andrews).

59-G-5 Heart rots of upper-slope species (P. E. Aho).

59-G-6 Pathology of advanced reproduction on upper-slope clearcuts (P. E. Aho).

61-G-4 The Indian paint fungus in northern Idaho (A. D. Partridge).

62-G-2 Red rot in merchantable ponderosa pine in the Black Hills (S. R. Andrews and T. E. Hinds).

62-G-4 Decay in advanced alpine fir regeneration in British Columbia II. In the Kamloops District (R. B. Smith and H. M. Craig).

62-G-5 Decay in young grand fir (O. C. Maloy).

62-G-6 Bark decomposition by four species of wood decay fungi (L. Reesman M.S.U. Graduate Student).

63-G-3 A study of (Ophiostomaceae) wood staining fungi in North America (R. W. Davidson).

64-G-1 Heart rots of upper slope species (P. E. Aho).

65-G-1 Factors influencing resistance of lodgepole pine to attack by the bark beetle-blue stain fungi complex--pathological aspects (H. S. Whitney).

66-G-1 The effect of extracellular enzymes of lodgepole pine heartwood inhabiting fungi on the molecular structures of the fungitoxic stibenes pinosylvin and pinosylvinmono-methylether, and of the flavanones pinobanksin and pinocembrin (A. A. Loman).

66-G-2 Hazard in red fir on federal recreational lands in California (L. A. Paine).

67-G-1 Causes of cull in the mixed conifer stands of the Southwest (P. C. Lightle).
Objective: To determine the causes of heart rot in the commercial forest trees of the Southwest, their distribution, and the damage each causes.

67-G-2 Microorganisms associated with brown stringy rot decay (*Echidontium tinctorium*). (O. D. Maloy, J.D. Rogers, and D. Hudson).
Objective: To attempt to determine if initial invasion by and development of other microorganisms predispose coniferous species to attack by *Echidontium tinctorium*.

H. Stem Diseases - Rusts and Cankers

53-H-2 Testing progeny of resistant pines for susceptibility to white pine blister rust in the Inland Empire (R. T. Bingham).

53-H-4 Blister rust canker development following artificial and natural inoculation (A. D. Partridge).

53-H-6 Forest tree rusts of British Columbia (W. G. Ziller).

54-H-2 Rate of growth of white pine blister rust cankers on sugar pine in California (D. R. Miller).

54-H-3 White pine blister rust (G. M. Harvey).

54-H-4 Status-of-control plots (D. R. Miller).

54-H-5 Summary of disease survey and scouting data (D. R. Miller).

54-H-7 Test fungicides for protection of white pines (C. R. Quick).

54-H-8 Search for blister rust-resistant white pines (C. R. Quick).

54-H-9 Appraisal and prediction of timber losses caused by blister rust (D. R. Miller).

54-H-10 Analysis of physical and ecological site factors contributing to high rust hazard (C. R. Quick).

55-H-2 Tests of fungicides to inhibit blister rust development on the leaves of ribes bushes (C. R. Quick).

55-H-4 Effect of microclimatic conditions on the distribution and spread of white pine blister rust in California (R. V. Bega).

56-H-4 Distance spread and rust losses to sugar pine associated with prescribed control standards (D. R. Miller).

56-H-6 Etiological, host-parasite, and ecological investigations of the Atropellis disease of lodgepole pine in Alberta (J. C. Hopkins).

57-H-2 Vegetative propagation of sugar pine for disease resistance tests (C. R. Quick).

57-H-4 The relation of bark moisture to the development of Cryptodiaporthe canker on willow (J. E. Bier).

58-H-2 Western tree rusts of Coleosporioides complex (W. W. Wagener).

58-H-3 Cronartium top-kill in ponderosa pine (K. R. Shea).

59-H-1 Cronartium comandrae rust on lodgepole pine in Wyoming (E.A. Andrews).

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 adaptation in inter-species hybrids, toward accumulation of a pool of rust-resistance genes from other white pines of the world (R. T. Bingham).

62-H-1 Life cycles of western conifer rusts (R.G. Krebill and D.L. Nelson).

62-H-2 Taxonomy and identification of native pine-stem Peridermia (R. G. Krebill and D. L. Nelson).

63-H-1 Differentiation of live from dead hyphae of Cronartium ribicola. I. Supravital staining (J. W. Koenigs).

63-H-2 Differentiation of live from dead hyphae of Cronartium ribicola. II. Tissue culturing (J. W. Koenigs).

63-H-5 Epidemiology of Cronartium comandrae. II. Relation of infection to environment (R. G. Krebill).

64-H-1 Derma dieback of Douglas-fir in the Cariboo Region, British Columbia (A. K. Parker).

64-H-2 Conifer stem rusts: pathological histology (R. G. Krebill).

65-H-2 Treatment and sampling of western white pine trees for bioassay of Acti-dione (C. D. Leaphart).

65-H-3 Western gall rust epidemiology, symptomatology, and life cycle (F. W. Cobb, Jr.; J.R. Parmeter, Jr.; and D. L. Nelson).

65-H-4 Cytology of western gall rust fungi (J. G. Christensen).

65-H-5 Cytospora canker associated with dieback of dwarfmistletoe infected branches of red fir in California (R. F. Scharpf, and R. S. Smith).

66-H-1 Feasibility of establishing white pine tissue explants and/or sub-cultures infected with Cronartium ribicola in axenic culture (A. E. Harvey).

66-H-2 Nutritional and environmental requirements of axenicly cultured western white pine tissue (A. E. Harvey).

66-H-3 Comparative physiology of varieties of western white pine with respect to their reaction to the blister rust fungus (R. J. Hoff).

66-H-4 Level and trend of natural inactivation of blister rust cankers (R. D. Hungerford and C. D. Leaphart).

66-H-5 Persistence of natural inactivation of blister rust cankers (R. D. Hungerford and C. D. Leaphart).

66-H-6 Numbers and kinds of resistance-genes and their relation to rust symptomatology (G. I. McDonald).

66-H-7 Precise estimates of heritability and combining ability of rust resistance (G. I. McDonald).

66-H-8 Development and pathogenicity of Hypoxylon fuscum on northwestern species of alder (Alnus) (J. D. Rogers).

66-H-9 Biological control of western white pine blister rust (E. F. Wicker, R. Williams, and C. G. Shaw).
Objective: To investigate the fungi, Tuberculina maxima, associated with blister rust cankers, and to determine what effect these fungi may have on canker development.

66-H-10 Secondary screening of chemicals for control of white pine blister rust on sugar pine (H. H. Bynum).
Objective: Self-explanatory.

67-H-1 Etiology of aspen cankers (T. E. Hinds).
Objective: To determine what agencies are responsible for the development of black cankers and nectria-like cankers on aspen and how these canker producing agents are transmitted and introduced into healthy trees.

67-H-2 Epidemiology of Cronartium comandrae. III. Ecology of comandra (R. G. Krebill).
Objective: To understand factors influencing the abundance of comandra so that this alternate host might be reduced in areas where comandra rust of pines is a problem.

67-H-3 Establishing Cronartium ribicola in axenic culture (R. D. Hungerford).
Objective: To grow C. ribicola on artificial media (using spore forms, refined techniques employed with other obligate parasites, or developing new techniques) and to assess the nutritional and environmental requirements for its growth.

67-H-4 Development and implementation of an assessment program for screening potential therapeutic agents against white pine blister rust (A. E. Harvey).
Objective: Self-explanatory (to be developed as a three-stage program starting with a laboratory screen and carrying promising candidates successively through greenhouse tests to field tests prior to operational pilot testing).

67-H-5 Blister rust physiology. I. Extraction of protein from white pine tissue (N. E. Martin).
Objective: To develop an extraction procedure for the proteins of white pine bark which will provide: (1) proteins in their native state, (2) high protein yield, and (3) an extract representative of a) the protein spectrum of the bark, and b) a proportionate concentration of each member of that spectrum.

67-H-6 Blister rust physiology. II. Concentration change of metal cations associated with infection in western white pine (N. E. Martin).
Objective: 1) To establish concentration differences between diseased and healthy white pine trees and tissues for the metal cations: potassium, sodium, calcium, and magnesium; 2) To map concentration gradients of these metal cations in the tree; and 3) To establish a concentration difference between current year needles and a composite of needles older than one year.

67-H-7 Controlling level and uniformity of inoculations and infection by Cronartium ribicola toward reducing number of replicates and seedlings required for progeny testing (R. T. Bingham).
Objective: To eliminate patchy inoculation that has increased inter- and intra-block variance of infection in progeny tests, thereby reducing costs of the tests.

67-H-8 Stem rusts of pine--basic cytological, taxonomic and life history studies (Y. Hiratsuka, and J. M. Powell).
Objective: To study aspects of the cytology, taxonomy and life history of the pine stem rusts of the region (Alberta Territories) and related species in the world.

67-H-9 Taxonomy and biology of forest tree rusts in western North America with special emphasis on the species of Pucciniastrum, Chrysomyxa, and Melampsora (Y. Hiratsuka).
Objective: Self-explanatory.

67-H-10 The epidemiology and aerobiology of the Comandra blister rust, Cronartium comandrae Peck (J. M. Powell).
Objective: To investigate the factors influencing the spread and intensification of the rust in Alberta. To obtain a better understanding of the incidence and damage of the rust.

67-H-11 A study of larch canker (Phomopsis sp.) in seedling and sapling stands of western larch (H. V. Toko, and C. E. Carlson).
Objective: To determine: (1) distribution; (2) incidence; (3) impact of phomopsis canker in regeneration of western larch in northern Idaho and western Montana.

I. Wilt and Blight Diseases

53-I-1 Pole blight of western white pine (C. D. Leaphart).

53-I-3 Pole blight of western white pine (R. G. McMinn, and A. C. Molnar).

J. Defects and Decays of Forest Products

53-J-1 Deterioration of slash of lodgepole pine in Alberta (V. J. Nordin).

58-J-1 Deterioration of beetle-killed Engelmann spruce in Colorado (F. G. Hawksworth and T. E. Hinds).

60-J-1 Relation of wood inhabiting fungi to preservatives (J. W. Roff, and A. J. Cserjesi).

63-J-1 Deterioration of Douglas-fir logs (R. B. Smith, and H. M. Craig).

65-J-1 Incense cedar pocket rot: effects of infection and biology of the causal fungus (W. W. Wilcox).

65-J-2 The use of gas chromatographic methods for carbon dioxide detection in the respiration of fungi attacking wood (R. S. Smith).

66-J-1 Effectiveness of Boron diffusion treatment vs. advance of heartrots in stored unseasoned lumber (J. W. Roff).

66-J-2 Interaction of fungi and chemical preventives (J. W. Roff).

66-J-3 Microbiology and pathology of wetwood in California firs (W. W. Wilcox).

K. Miscellaneous Studies

53-K-1 Survival of fire-damaged trees (W. W. Wagener).

54-K-1 An undescribed disease on Rocky Mountain juniper (C. W. Waters).

54-K-2 Vegetative propagation of disease-resistant western white pine (C. W. Waters).

56-K-2 Disease surveys of pathogenicity tests on selected clones of poplar, including introduced varieties (J. E. Bier).

57-K-2 Bio-assay of "Agri-mycin-100"-treated Pacific silver and noble firs (K. R. Shea).

58-K-1 Tests of systemic fungicides (G. M. Harvey).

59-K-2 Control of forest disease by systemic fungicides (G. M. Harvey).

60-K-2 Systemic fungicides in Douglas-fir (L. C. Weir).

62-K-1 The taxonomy, comparative morphology and ecology of fungi of arid lands (P. D. Keener).

63-K-1 Fungus flora of the State of Washington and the Pacific Northwest (C. G. Shaw and H. Goree).

63-K-2 The isolation, purification, and measurement of the antibiotic Phytoactin or its derivatives from treated plant tissues (S. O. Graham, C.G. Shaw, A.E. Harvey, M. Stoner, and S. Gurusiddaiah).

63-K-3 Life cycles of Ascomycetes with special emphasis on cytology (J. D. Rogers, and S. Jong).

64-K-3 The time required for Tuberculina maxima to inactivate blister rust infections on western white pine (J. W. Kimmey).

65-K-2 The incubation period of Tuberculina maxima Rostr. (J. W. Kimmey and E. F. Wicker).

65-K-3 Fungi associated with Peridermium cerebroides Meinecke on Pinus radiata D, Don. (J.W. Byler, F.W. Cobb, Jr., and J.R. Parmeter, Jr.).

65-K-5 Survey of naturally inactivated lethal-type blister rust cankers on young western white pine in the Inland Empire (J. W. Kimmey).

65-K-6 The effect of treating western white pine stands with antibiotics on the normal fungal population (O. K. Miller, Jr.).

66-K-1 The isolation, purification, and measurement of the antibiotic, Phytoactin, or its derivatives from treated plant tissues (S.O. Graham, C.G. Shaw, and M. Stoner).

66-K-2 Physiological impact of treatment of western white pine seedlings with Phytoactin (A. E. Harvey, cooperative study with Washington State University, Pullman).

66-K-3 Effect of Phytoactin on several Basidiomycetes in culture (O.K. Miller, Jr., and J.W. Koenigs).

66-K-4 Investigations of cultural problems in the production of Christmas trees from natural stands (J.P. Nagle, R.W. Dingle, J.D. Rogers, E.P. Breakey, D.O. Turner, and B. Jones).

66-K-5 Investigation of nuclear phenomena in selected Ascomycetes (J.D. Rogers, C.G. Shaw, R. Duran, and Shung-Chang Jong).

66-K-6 Biologic control of western white pine blister rust (C.G. Shaw, C.D. Leaphart, E.F. Wicker, and R. Williams).

66-K-7 Fungus flora of the State of Washington and the Pacific Northwest (C. G. Shaw, and L. Grand).

66-K-8 Ecology of Tuberculina maxima (E. F. Wicker).

66-K-9 In vitro reaction of Tuberculina maxima to cycloheximide and Phytoactin (E. F. Wicker).

66-K-10 Nutritional requirements of Tuberculina maxima (E. F. Wicker).

66-K-11 Overwintering capabilities of Tuberculina maxima on white pine blister rust cankers (E. F. Wicker).

67-K-1 Evaluation of boron treatment of lumber against decay (J. W. Roff).

67-K-2 Prevention of Sapstain and mould on lumber (J. W. Roff).

67-K-3 Old growth western hemlock dieback in Northwest Washington (K. W. Russell).
Objective: To determine the extent of damage and identify the organisms or site factor causing the dieback. The study will also provide management guides for the District Forester and will help in planning the logging schedules for diseased areas.

67-K-4 Host-parasite relationships (A. K. Parker).
Objective: To obtain a better understanding of the nature of resistance to a number of diseases occurring in British Columbia. Initially, the diseases selected for study are: needle rusts of conifers, needle blight of Douglas-fir, needle blight of pines, and decay in spruce caused by Stereum spp.

67-K-5 Silvicultural control of Acropellis piniphila (L. C. Weir).
Objective: To determine the effect of various degrees of thinning in overstocked stands of lodgepole pine on the rate of spread of Acropellis piniphila.

67-K-6 Silvicultural control of Poria weirii (L. C. Weir).
Objective: To determine the effect of food base reduction on subsequent infection of regeneration.

67-K-7 Impact of hazardous tree failure on forested recreation sites (L. A. Paine).
Objective: To investigate the frequency of mechanical failure of trees by species, the types of failures, and the impact of failures in terms of type of accidents caused by each type of failure.

67-K-8 Factors involved in mechanical failure of hazardous trees in recreation sites (L. A. Paine).
Objective: To establish the visible characteristics and defects predisposing a tree to failure and the environmental factors most often contributing to in-season failure for aid in hazard control and planting programs in site management.

67-K-9 In-season tree failure on California recreational areas (L. A. Paine).
Objective: To provide statements of annual property losses and injuries caused by defective trees on recreation sites in California for use in recreation management.

67-K-10 Effectiveness of hazard reduction programs on recreation sites - losses and various costs of protection (L. A. Paine).
Objective: (1) To evaluate the current effectiveness of hazard control programs, (2) to evaluate the benefit-cost relationships for control of each type of hazard, and (3) to indicate budget figures and changes in control emphasis which will result in more effective use of available resources.

67-K-11 Application of immunofluorescent method for identification of forest fungi (Y. Hiratsuka).
Objective: Self-explanatory.

67-K-12 An information service for biochemistry and control of plant parasite systems.--A research project in automatic storing, searching, and information retrieval (T.K. Burgess, S.B. Locke, C.G. Shaw, A.E. Harvey, and C.D. Leaphart).
Objective: To develop a retrieval system to provide relevant scientific and technical information on the specific fields of chemotherapy, host-parasite, physiology, and biochemical differentiation in host-parasite complexes.

APPENDIX II. -- TERMINATED PROJECTS

57-A-2 Field key of forest diseases in California (H. H. Bynum).

53-C-2 Control of diseases of seeds and seedlings of forest trees in California (R. V. Bega).

53-C-4 Nursery and plantation diseases (G. M. Harvey).

57-C-1 Molds of forest tree seed (K. R. Shea).

58-C-1 Effect of storage on seedling vitality (E. Wright).

62-C-1 Pathological failures of true fir seed (G. M. Harvey).

56-D-1 Influence of mycorrhizae on survival and development of Douglas-fir seedlings (J. E. Bier).

57-D-1 Armillaria mellea on ponderosa pine (J. Hunt).

57-D-3 Poria weirii root rot of Douglas-fir (A. K. Parker).

58-D-1 Eastern Oregon regeneration (E. Wright).

58-D-2 Fungi-bark beetle study (E. Wright and D. Allen).

58-D-3 Soil microbiology study (E. Wright and W. Lowry).

59-D-3 "X" disease (J. R. Parmeter and R. V. Bega).

61-D-3 Soil microbiology with special reference to forest and nursery root diseases (K. C. Lu).

66-D-4 Assessment of root pathogens (K.C. Lu, J.M. Trappe, C.Y. Li, and W. B. Bollen).

66-D-5 Comparison of rhizosphere microorganisms of mycorrhizal and non-mycorrhizal roots of red alder and Douglas-fir (K.C. Lu, J.M. Trappe, J.L. Neal, and W.B. Bollen).

66-D-9 Seasonal distribution of fungi in a Douglas-fir forest soil (E.E. Nelson).

53-E-1 Elytroderma needle cast of ponderosa pine (T. W. Childs).

53-E-2 Rhabdocline and Rhabdogloeum needle casts of Douglas-fir (A. K. Parker).

57-E-3 Taxonomic studies of principal fungi associated with ponderosa pine needle cast in Arizona (P. D. Keener).

53-F-2 Dwarfmistletoes of the Rocky Mountain Region (L. S. Gill).

56-F-1 Dwarfmistletoe of ponderosa pine (K. R. Shea).

57-F-7 Fungi associated with branch dieback in mistletoe-infected conifers (J. W. Parmeter, Jr.).

60-F-1 Studies of dwarfmistletoe in British Columbia (R. B. Smith).

63-F-3 Dwarfmistletoe (Arceuthobium spp.) (R. B. Smith).

53-G-10 Some aspects of the ecology of the Indian paint fungus in British Columbia (G. P. Thomas).

55-G-1 Correlation of the rate of extension of heart rots in living California trees with air temperature (L. A. Paine).

55-G-2 Cull indicator factors for red fir in the Sierra sub-regions (H. H. Bynum).

56-G-1 Heart rots of young-growth Douglas-fir (G. M. Harvey).

56-G-2 Decay of lodgepole pine (J. Hunt).

56-G-3 Decay of Pacific Coast alder (G. M. Harvey).

57-G-6 Cull indicators in Engelmann spruce (F. G. Hawksworth).

57-G-7 Navajo defect study (S. R. Andrews and P. C. Lightle).

59-G-2 Studies on blue stain of bark beetle-infested lodgepole pine (R. C. Robinson).

59-G-3 The role of decay in scar-damaged Douglas-fir in the interior dry belt region of British Columbia (H. M. Craig).

60-G-1 Study of decay in spruce, balsam, and lodgepole pine in the wet belt of the Kamloops Forest District of British Columbia (J. E. Browne).

61-G-2 Decay of hemlock in the Kitwanga area of British Columbia (J. E. Browne).

57-H-1 Canker and dieback fungi on Sequoia, Libocedrus, Ericaceous species, and other California tree species (J. R. Parmeter, Jr., and R. V. Bega).

60-H-1 Studies of forest tree rusts in Alberta (J. E. Nighswander).

60-H-2 Induction of resistance to Cronartium ribicola in Pinus monticola seedlings by seed treatment with chemical mutagens (J. W. Hanover).

60-H-3 Comparative biochemistry of resistant and nonresistant Pinus monticola to infection by Cronartium ribicola (J. W. Hanover).

65-H-1 Inoculation of sugar pine with Cronartium ribicola through bark grafts (H. H. Bynum).

55-I-2 Effect of silvicultural measures on the white pine pole blight disease (D. P. Graham).

56-J-1 Decay of killed Douglas-fir in interior British Columbia (G. P. Thomas).

57-J-4 Deterioration of Chermes-killed Pacific silver fir (K. R. Shea).

57-J-5 Post-logging decay in western hemlock and Sitka spruce (K. R. Shea).

57-K-1 Fungicidal properties of western redcedar extractives (J. W. Roff).

64-K-4 Diseases of *Pinus lambertiana* (H. Offord).

65-K-1 Environmental requirements of *Tuberculina maxima* in culture (H. H. Bynum).

65-K-4 *In vitro* testing of fungicides on spores of *Cronartium ribicola* (H. H. Bynum).

APPENDIX III. -- NEW OR MODIFIED TECHNIQUES

TISSUE CULTURE - A TOOL FOR PHYTOPATHOLOGICAL RESEARCH

Alan E. Harvey

The tissue culture technique represents one of the most potent tools available to basic plant research today. Theoretically, tissue culture provides the plant pathologist an opportunity to study the host, the parasite, or the host-parasite system under conditions designed to satisfy specific research objectives. Potentially, this is an ideal system for investigating pathological and host response physiology of plant parasitic organisms.

This technique will be particularly useful in the investigation of culture methods and host-parasite physiology of obligate parasites such as forest tree rusts[1/]. New information concerning the biochemical interrelationships of these rusts will, hopefully, render them controllable via artificial means.

1/ Harvey, A. E. 1967. Tissue culture of Pinus monticola Dougl. on a chemically defined medium. Canad. J. Bot. 45: 1783-1787.

Harvey, A. E. 1967. Axenic culture of white pine cambial explants infected with blister rust. (Abstr.) Phytopathology 57: 10.

Harvey, A. E. 1967. Culture of blister rust infected western white pine tissue. Report for Western International Forest Disease Work Conference, Sept. 1967.

APPENDIX IV. -- PUBLICATIONS

Baranyay, J. A. 1967. Shelterbelts in the Great Plains regions of the United States. Can. Dept. of For. and Rural Dev. Inf. Rept. A-X-5.

__________. 1967. Notes on Hypoxylon canker of aspen in Alberta. For. Chron. (In press).

Baranyay, J. A., and Y. Hiratsuka. 1967. Identification and distribution of Cirborinia whetzelii (Seaver) Seaver in western Canada. Can. J. Bot. 45: 189-191.

Bega, Robert V., and Richard S. Smith, Jr. 1966. Distribution of Fomes annosus in natural forests of California. Plant Disease Reptr. 50(11): 832-836.

Bega, Robert V., and Howard A. Scott. 1966. Ultrastructure of the sterigma and sporidium of Cronartium ribicola. Can. J. Bot. 44: 1726-1727.

Bingham, R. T. 1966. Breeding blister rust resistant western white pine. III. Comparative performance of clonal and seedling lines from rust-free selections. Silvae Genetica 15: 160-164.

Bollen, W. B., K.C. Lu, J.M. Trappe, and R. F. Tarrant. 1967. Primary microbiological succession on a landslide of alpine origin at Mt. Rainier. U.S. Forest Service, PNW Forest & Range Expt. Sta. Res. Note No. PNW-50. 8 p.

Callaham, R. Z., R.E. Goddard, H.M. Heybroek, C.M. Hunt, G.I. McDonald, J.A. Pitcher, and J.A. Winieski. 1966. General guidelines for practical programs toward pest-resistant trees. P. 489-493. In: H. D. Gerhold, 505 pp. Oxford: Pergamon Press.

Childs, T. W., and J. W. Edgren. 1967. Dwarfmistletoe effects on ponderosa pine growth and trunk form. Forest Sci. 13: 167-174.

Childs, T. W., and K. R. Shea. 1967. Annual losses from disease in Pacific Northwest forests. U. S. Forest Serv. Resource Bull. PNW-20, 19 p.

Deneke, F. J., and G. F. Bratton. 1966. A study of host crossing by dwarf mistletoes. Colorado-Wyoming Acad. Sci. Jour. 5: 74-75.

Elliott, D. P., and J. M. Powell. 1966. A cage for collecting insects from tree stems and branches. Can. Entomologist 98: 1112-1113.

Graham, Donald P. 1967. A training aid on dwarfmistletoe and its control. U.S. Forest Service, U.S.D.A., Pacific Northwest Region, Portland, Oregon.

Hanover, James W. 1966. Studies on the nature of resistance of Pinus monticola Dougl. to infection by Cronartium ribicola Fischer. Pp. 165-177. In: Gerhold, H.D., et al. (Eds.), Breeding Pest-Resistant Trees. 505 pp. Oxford: Pergamon Press.

Hanover, James W. 1966. Tree improvement for disease resistance in western United States and Canada. Pp. 53-56. In: H. D. Gerhold, et al. (Eds.), Breeding Pest-Resistant Trees. 505 pp. Oxford. Pergamon Press.

Hanover, J. W., and R. J. Hoff. 1966. A comparison of phenolic constituents of Pinus monticola resistant and susceptible to Cronartium ribicola. Physiologia Plantarum 19: 554-562.

Hanover, J. W., and R. J. Hoff. 1966. Pollination of western white pine with water suspensions of pollen--a technique for chemical mutagen treatments. Forest Sci. 12(3): 372-373.

Harvey, A. E. 1967. Axenic cultures of western white pine cambial explants infected with blister rust. Phytopathology 57(1): 1005. (Abstr.).

____________. 1967. Effect of Phytoactin treatment on mycorrhizae-root consociations in western white pine. Plant Disease Reptr. 51(12): 1012.

____________. 1967. Tissue culture of Pinus monticola on a chemically defined medium. Can. J. Bot. 45: 1783-1787.

Harvey, A.E., S.O. Graham, and L.D. Becker. 1967. Extraction and assay of the antibiotic Phytoactin from western white pine tissue. (Abstr.) Phytopathology 57: 99.

Harvey, George M. 1967. Growth rate and survival probability of blister rust cankers on sugar pine branches. U.S. Forest Serv. Pacific Northwest Forest and Range Expt. Sta. Res. Note PNW-54, 6 p.

Hawksworth, F. G., and D. Wiens. 1966. Observations on witches'-broom formation, autoparasitism, and new hosts in Phoradendron. Madroño 18:218-224.

Hawksworth, F.G., D. Wiens, and D.P. Graham. 1962. Dwarfmistletoe on brewer spruce in Oregon. Northwest Science 41: 42-44.

Hiratsuka, Y., W. Morf, and J. M. Powell. 1966. Cytology of aeciospores and aeciospore germ tubes of Peridermium harknessii and P. stalactiforme of the Cronartium coloeosporioides Arth. complex. Can. J. Bot. 44: 1639-1643.

Hiratsuka, Y., L.E. McArthur, and F.J. Emond. 1967. A distinction between Pucciniastrum goeppertianum and P. epilobii with clarification of status of Peridermium holwayi and P. ornamentale. (In Press) Can. J. Bot.

Hoff, Raymond J. 1966. Blister rust resistance in western white pine. Pp. 119-124. In: H. D. Gerhold, et al (Eds.), Breeding Pest-resistant Trees. 505 pp. Oxford: Pergamon Press.

Koenigs, J. W. 1966. Studies of the cytochemical localization of specific dehydrogenases in frozen, diseased tissues of Pinus. Amer. J. Bot. 53(10): 1101-1108.

Krebill, R. G. 1966. Functional pycnia of Pucciniastrum goeppertianum. Abstr. Utah Academy of Arts, Sciences and Letters Proc. 43(Part 2): 78.

Krebill, R. G. 1967. Gymnosporangium clavariiforme in witches'-brooms of Juniperus communis. Plant Disease Reptr. 51(2): 143-147.

Laycock, W. A., and R. G. Krebill. 1967. Comandra, grazing, and comandra blister rust. U.S. Forest Service Research Paper INT-36. 9 p.

Laycock, W. A., and R. G. Krebill. 1967. Comandra is not an invader on most western ranges. U.S. Forest Service, R-4 Range Improvement Notes 12(3): 1-5.

Li, C. Y., K.C. Lu, J.M. Trappe, and W.B. Bollen. 1967. Selective nitrogen assimilation by Poria weirii. Nature (London) 213(5078): 814.

McMinn, R. G. 1965. Further observations on pole blight of white pine. Can. Dept. Forestry, Bi-monthly Progress Rept. 21(6): 3.

Martin, Neil E. 1967. Histochemical analysis of the blister rust fungus, Cronartium ribicola. Phytopathology (Abstr.) 57(8): 820-821.

Mielke, J. L., and R. S. Peterson. 1967. Albino Peridermium harknessii in ponderosa pine. Plant Disease Reptr. 51: 306-309.

Neal, J.L., Jr., K.C. Lu, W.B. Bollen, and J.M. Trappe. 1967. Two simple time-saving techniques for studies of soil microbial populations and subsequent culture characterization. Soil Biology-International News Bull. International Soc. of Soil Sci. Comm. III(6): 38.

Neal, J.L., Jr., J.M. Trappe, K.C. Lu, and W.B. Bollen. 1967. Sterilization of red alder seedcoats with hydrogen peroxide. For. Sci. 13: 104-105.

Neal, J.L., Jr., K.C. Lu, W.B. Bollen, and J.M. Trappe. 1967. Two simple time-saving techniques of studies of soil microorganisms. U.S.D.A., For. Serv. Res. Note PNW-57, 3 pp.

Nelson, Earl E. 1967. Factors affecting survival of Poria weirii in small, buried cubes of Douglas-fir. For. Sci. 13(1): 78-84.

Offord, Harold R. 1966. Sequential sampling of ribes populations in the control of white pine blister rust (Cronartium ribicola Fischer) in California. U.S. Forest Service, U.S.D.A. PSW Station Paper PSW-36, 14 pp.

Paine, Lee A. 1967. Mechanical failures of hazardous trees on recreation sites--evaluation of losses and costs of protection. U.S. Forest Service, U.S.D.A. Res. Note. (In review).

___________. 1966. Accidents caused by hazardous trees on California forest recreation sites. U.S. Forest Service, U.S.D.A. Forest Service Res. Note PSW-133).

Parmeter, J.R., Jr., H.S. Whitney, and W.D. Platt. 1967. Affinities of some Rhizoctonia species that resemble mycelium of Thanetephorus cucumeris. Phytopathology 57: 218-223.

Peterson, Glenn W. 1967. Botryodiplodia disease of Russian-olive. Phytopathology 57: 825-826 (Abstr.)

__________. 1967. Control of Cylindrosporium leaf spot disease of Rhus trilobata and Shepherdia argentea seedlings. Plant Disease Reptr. 51: 700-701.

__________. 1967. Dothistroma needle blight of Austrian and ponderosa pines: epidemiology and control. Phytopathology 57: 437-441.

__________. 1967. Dothistroma needle blight of pines in North America. International Union of Forestry Research Organizations Congress, Munich, 1967. Papers Volume V. Section 24, Pp. 269-278.

Peterson, R. S. 1967. Cypress rusts in California and Baja California. Madrono 19: 47-54.

__________. 1967. Studies of juniper rusts in the West. Madroño 19: 79-91.

Peterson, R. S., and R. Salinas Quinard. 1967. Cronartium conigenum: Distribucion y efectos en los pinos. Instituto Nacional de Investigaciones Forestales. Boletin Tecnico No. 19.

Powell, J. M. 1967. A study of habitat temperatures of the bark beetle Dendroctonus ponderosae Hopkins in lodgepole pine. Agri. Meteorology 4: 189-201.

Powell, J. M., and W. Morf. 1967. A 24-hour impaction spore collector. Can. J. Plant Sci. 47: 222-224.

Powell, J. M., and W. Morf. 1966. Temperature and pH requirements for aeciospore germination of Peridermium stalactiforme and P. harknessii of the Cronartium coloesporioides Arth. complex. Can. J. Bot. 44: 1597-1606.

Quick, Clarence R. 1966. Experimental inoculation of ponderosa pine with western gall rust. Plant Disease Reptr. 50(8): 550-552.

Raski, D. J., and J. W. Riffle. 1967. Two new species and further notes on Criconemoides Taylor, 1936 (Criconemotidae: Nematoda). Proc. Helminthological Soc. of Washington, D. C. 34: 212-219.

Reid, R.W., H.S. Whitney, and J.A. Watson. 1967. Reactions of lodgepole pine to attack by Dendroctonus ponderosae Hopkins and blue stain fungi. Can. J. Bot. 45: 1115-1126.

Riffle, J. W. 1967. Effect of an Aphelenchoides species on the growth of a mycorrhizal and pseudomycorrhizal fungus. Nematologica 13: 151 (Abstr.).

__________. 1967. Effect of an Aphelenchoides sp. on the growth of a mycorrhizal and a pseudomycorrhizal fungus. Phytopathology 57: 541-544.

Riffle, J. W., and J. E. Kuntz. 1966. Nematodes in maple blight and maple dieback areas in Wisconsin. Plant Disease Reptr. 50: 677-681.

Riffle, J. W., and J. E. Kuntz. 1967. Nematodes in maple blight and maple dieback areas of Wisconsin. Trees Mag. 27(3): 8-11.

Riffle, J. W., and J. E. Kuntz. 1967. Pathogenicity and host range of Meloidogyne ovalis. Phytopathology 57: 104-107.

Scharpf, R. F., and F. G. Hawksworth. 1966. Hosts and distribution of Uredo phoradendri. Mycologia 58: 811-812.

Scharpf, R. F., and J. R. Parmeter, Jr. 1966. Spread and buildup of dwarfmistletoe in a 22-year-old plantation on Jeffrey pine in California. Fourteenth Western Int. For. Dis. Work Conf. Proc. P. 87.

Scharpf, R. F., and J. R. Parmeter, Jr. 1967. The biology and pathology of dwarfmistletoe Arceuthobium campylopodum f. abietinum parasitizing true firs (Abies spp.) in California. U.S.D.A. Forest Serv. Tech. Bull. 1362, 42 pp.

Shea, Keith R. 1967. Effect of artificial root and bole injuries on diameter increment of Douglas-fir. Weyerhaeuser Forestry Pap. 11, 11 pp.

Smith, A. H., and S. M. Zeller. 1966. A preliminary account of the North American species of Rhizopogon. Mem. N.Y. Bot. Gard. 14(2): 1-177.

Smith, Richard S., Jr. 1966. Foliage disease control. 10th Biennial Western Forest Nursery Council Meeting Proc. 10: 70-72.

Smith, Richard S., Jr., and S. L. Krugman. 1967. Control of the charcoal root disease of white fir by fall soil fumigation. Plant Disease Reptr. 51: 671-674.

Tripp, H.A., R.E. Stevenson, and J.A. Baranyay. 1967. Forest insect and disease survey, Alberta-Northwest Territories-Yukon Region. In: Ann. Rept. of the For. Ins. and Dis. Survey, Can. Dept. of Forestry. (In press).

Whitney, H. S., and J. R. Parmeter, Jr. 1963. Synthesis of heterokaryons in Rhizoctonia solani Kuhn. Can. J. Bot. 41: 879-886.

Whitney, H. S., and J. R. Parmeter, Jr. 1964. The perfect stage of Rhizoctonia hiemalis. Mycologia 56: 114-118.

Wicker, E. F. 1967. Appraisal of biological control of Arceuthobium campylopodum f. campylopodum by Colletotrichum gloeosporioides. Plant Disease Reptr. 51(4): 311-313.

______________. 1967. Collection and storage of Arceuthobium spp. U.S. Forest Service, U.S.D.A. Res. Paper INT-33. 13 pp.

______________. 1967. Seed destiny as a klendusic factor of infection and its impact upon propagation of Arceuthobium spp. Phytopathology 57(11): 1164-1168.

Wicker, Ed F., and J. W. Kimmey. 1967. Mode and time of infection of western white pine blister rust cankers by Tuberculina maxima. Phytopathology 57: 1010. (Abstr.).

Wicker, Ed F., and C. G. Shaw. 1967. Target area as a klendusic factor in dwarf mistletoe infections. Phytopathology 57(11): 1161-1163.

Wiens, D. 1967. Chromosomal relationships of the New World mistletoes. Am. Jour. Bot. 54: 654.

Young, R. A. 1966. Dwarfmistletoe control in the National Forests. Colorado Outdoors 15(1): 42-44.

Zak, B. 1967. A nematode (Meloidodera sp.) on Douglas-fir mycorrhizae. Plant Disease Reptr. 51(4): 264.

APPENDIX V. -- MINUTES OF THE BUSINESS MEETING

The business meeting was called to order by Chairman Alex Molnar at 0945.

I. Secretary's Report (Ed F. Wicker)

Minutes of the 14th meeting were adopted as they appeared in the Proceedings.

The financial report follows:

	Credit	Debit
Balance, 14th Conference	145.97	
Registration	53.00	
Banquet and Bus	540.50	
Coffee expense		25.20
Banquet expense		259.00
Bus expense		228.24
Postage		4.51
Jack Bier memorial fund		25.00
Total	$739.47	$541.95

Balance to incoming secretary-treasurer R. G. McMinn $197.52

II. Committee Reports

Dwarf mistletoe--Chairman Molnar appointed F. G. Hawksworth as new committee chairman. A desire was expressed by new committee chairman to expand, change title and redefine purpose of committee. Copies of committee report were circulated and appear in the Proceedings in Appendix VI.

Recreation--Lee Paine reported for the recreation committee. The report appears in the Proceedings in Appendix VI.

Interim Program Report--Hart Bynum reported as interim program chairman. List of suggestions was impressive and will be passed on to the 16th program chairman and his committee.

III. Old and New Business

1. Joint Meeting with Insect Work Conference

Alex Molnar commented on joint Insect-Disease Conference meeting. It was decided to await further contact by entomologists.

2. New and Terminated Projects

C. D. Leaphart reported and asked for a total response to previous inquiry to bring 15th Proceedings up to date.

3. Membership

Secretary read honorary membership list. Following a discussion from the floor, L. F. Roth moved that honorary membership begin with the termination of principal employment and that such designation be irrevocable. Seconded by T. W. Childs. Motion carried unanimously. Members previously removed are to be restored.

L. F. Roth asked for discussion on definition of student members. C. G. Shaw replied as to A.P.S. policy. Bob Bega commented in reference to 5th Proceedings. C. D. Leaphart passed out copies of policy statements.

C. G. Shaw moved that student member be defined as any individual enrolled in pathology in a university and who is not more than 50% gainfully employed. Dick Smith seconded motion. Motion passed by voice vote.

4. In Memorium

Chairman Molnar discussed the Paul Keener memorial fund and Mrs. Betty Keener's non-attendance at 15th Conference.

C. G. Shaw suggested that Chairman delegate someone to prepare a letter of Jack Bier's contributions for inclusion in 15th Proceedings. (Ray Foster forwarded such letter).

C. D. Leaphart moved that Conference provide a donation of $25.00 to the Jack Bier memorial fund. Seconded by T. W. Childs. Carried by voice vote.

5. Historical Records

Historian G. W. Wallis was not present. Chairman Molnar reported that copies of the 3rd, 6th, 7th, 9th, 10th, 12th, and 14th Conference Proceedings were available from the historian.

Chairman Molnar reported that work was progressing on the binding of past Conference Proceedings. Costs would be about $4.50-$5.00 per volume. Discussion on numbers of volumes was tabled because this was covered in 14th business meeting.

6. Conference Proceedings

Discussion on changes in Conference Proceedings was tabled by Chairman. Continue in present manner.

7. Chairman Molnar appointed Ken Russell as chairman of a standing committee. Ken read the proposal and moved for adoption. Bob Bega seconded. Carried by voice vote.

"A committee has been proposed to present annually to WIFDWC members, the results of all Forest Disease Control Tests conducted by the Conference membership.

"Forest Disease Control Tests would be reported in three categories:

1. Chemical
2. Biological
3. Silvicultural

"This standing committee would operate similarly to the Committee on Status and Needs of Research on Dwarfmistletoes.

"It is proposed that the first report be made to the membership at the 1968 meeting. The first report could include results of Forest Disease Control Tests from as much as five years back. Succeeding reports would be on an annual basis.

"This committee will not include results of dwarfmistletoe control. Refer to the annual Dwarfmistletoe Committee report for dwarfmistletoe control results.

"The term of the committee chairman will be two years.

"Current members of the committee for 1967-68 are:

Ken Russell, Chairman
Al Tegethoff
James L. Stewart
Hart Bynum
Larry Weir
Harvey Toko".

8. 1968 Meeting

C. D. Leaphart invited the group to meet in Coeur d'Alene, Idaho, with a second alternative in the Moscow-Pullman area.

R. V. Bega invited the group to meet at the University of Hawaii, Honolulu.

Leaphart's invitation was accepted for Coeur d'Alene, Idaho; R. V. Bega only exception.

A discussion on time of meeting followed. Comments from floor by Bega, Scharpf, and Shaw. R. V. Bega moved for meeting date not before mid-October. R. Scharpf seconded. Vote: 22 for; 6 opposed.

9. <u>Election of Officers</u>

R. S. Smith reported on nomination as Chairman of Committee (of one).

Chairman -- Stuart Andrews
Secretary-Treasurer -- R. G. McMinn

Carried unanimously.

10. <u>Commendations</u>

Chairman Molnar asked for recognition of Local Arrangements Committee, Program Chairman and Committee, panel moderators, Park Service, and State Land Office. (Membership applause).

11. R. V. Bega moved to close business meeting; seconded by E. F. Wicker. Meeting adjourned.

COMMITTEE ON STATUS AND NEEDS OF RESEARCH ON DWARFMISTLETOES

F. G. Hawksworth, J. R. Parmeter, K. R. Shea,
and J. A. Baranyay, Chairman

Highlights of 1967 Research

I. Taxonomy, hosts and distribution

a. Forest Insect and Disease Ranger Stan Allen has collected A. campylopodum (probably f. tsugensis) from Sitka spruce near Kitimat on the north coast of British Columbia. This is the first record of this combination in Canada.

An infestation of Douglas-fir dwarf mistletoe (A. douglasii) was located five miles east of Lytton, British Columbia, a town situated at the confluence of the Fraser and Thompson Rivers. The location is approximately 80 miles from the nearest previously known infestation and 65 miles further west. The extent of the infected area has not been determined accurately but covers at least one square mile. (Smith-Victoria).

b. Monterey pine (Pinus radiata) was successfully inoculated in the greenhouse with A. c. f. tsugensis seed collected from western hemlock. Inoculations were carried out on April 28, 1966 with pre-germinated seed. On August 18, 1967 the sole swelling produced was 26 mm in length and possessed 4 aerial shoots, one of which was 22 mm long. A report on infection of ponderosa, Scots, and Monterey pines by hemlock mistletoe has been prepared for the Canada Dept. of Forestry Bi-Monthly Research Notes.

Artificial inoculations in the plantation have failed to produce infections of western hemlock by larch dwarf mistletoe (A. c. f. laricis). As evidenced by swellings, infection of larch by hemlock dwarf mistletoe (A. c. f. tsugensis) is readily obtained. Twenty-five swellings of this latter combination have been produced. Of particular note is that only one of the 25 has so far produced aerial shoots even though the swellings are up to 72 mm in length. In contrast, of 17 infections of western larch by larch mistletoe, 11 have produced aerial shoots. In most of these, aerial shoots were produced very soon after the swellings were first observed. (Smith-Victoria).

c. Several mountain hemlock stands infested with dwarfmistletoe have been examined on the Coeur d'Alene National Forest in northern Idaho. In all cases, the causal organism has been identified as Arceuthobium campylopodum f. laricis. The inoculum source was traced to infected, overstory western larch residuals. (Wicker-IM).

d. Computer analyses are being used to help develop a taxonomic classification of Arceuthobium. The results have enabled us to structure the first sub-generic classification of the genus. The analyses also verify the distinctness of some previously undescribed mistletoes (for example, the Digger pine species). (Hawksworth RM & Wiens-University of Utah).

e. A trip to the Sierra Del Carmen, Coahulia, Mexico (just across the Rio from Big Bend, Texas) was made in cooperation with the Mexican Forest Services. Three dwarf mistletoes were found: A. douglasii on Pseudotsuga menziesii, A. campylopodum f. blumeri on Pinus strobiformis and A. vaginatum subsp. cryptopodum on Pinus ponderosa var. scopulorum and P. arizonica var. stormiae. In general, damage caused by the dwarf mistletoe was relatively minor. (Hawksworth and Lightle--RM).

II. Physiology and anatomy.

a. Chromatographic studies of the shoot pigments in Arceuthobium have been started. No reportable results to date. (Wiens - University of Utah).

III. Life cycle studies (seeds, infection, endophytic system, pollination, flowering, etc.).

a. Build-up of dwarf mistletoe in inoculated red fir has been surprisingly rapid since initial inoculation in 1957. For the first generation infections, abundant fruiting occurred only on 5 infections in 1963, 1964, and 1965. In the fall of 1966, 113 second generation infections were noted. Since dwarf mistletoe on red fir takes at least two years before symptoms appear, we can assume that the 113 new infections resulted from the 1963 and 1964 seed years. (Scharpf-PS, and Parmeter-Univ. of California).

b. Studies on chromosome analyses and flowering times in Arceuthobium have been summarized in an article submitted to the American Journal of Botany. It was found that there are three types of flowering: Group I, (e.g., A. americanum) undergoes meiosis in the fall and flowers in the spring; Group II, (e.g., A. campylopodum) flowers in summer to fall and undergoes meiosis immediately prior to flowering, and Group III, (e.g., A. vaginatum) flowers in the spring and undergoes meiosis immediately prior to flowering. (Wiens-Univ. of Utah).

IV. Host-parasite relations

a. Inoculation and dissection studies with mistletoe on red and white firs indicate that the region of the branch whorl ("girdle" as defined by Kuijt) is more liable to infection than is the region between branch whorls ("segment" as defined by Kuijt). Of 616 seeds placed at whorls and 656 placed at interwhorls, 34 and 13 infections respectively were obtained. Examination of 355 natural infections on 8 trees showed that more than 40% were centered at branch whorls. Since the area of branch whorls constitutes much less than 40% of the available target, a disproportionately high percentage of infections occur in the region of the whorl. (Parmeter and Platt-Univ. of California).

V. Effects on hosts (damage, mortality)

a. A study was started to enable us to predict yields in lodgepole pine stands on various sites and with various intensities of mistletoe. Satisfactory yield equations are already available for healthy stands so our study involves determining the impact of mistletoe so that the factor can be entered into the prediction equations. (Stewart-Region 2, U.S.F.S., and Hawksworth-RM)

b. Field work was completed on a study to determine the effects of dwarf mistletoe on growth and yields in even-aged ponderosa pine in Arizona, New Mexico, and Colorado. Data have been obtained on 7,376 trees on 107 transects in 14 national forests. Analyses of results have not been completed. (Lightle, Hawksworth, and Hinds-RM).

c. Preliminary studies on the effects of A. americanum on the wood properties of lodgepole pine have been started at Colorado State University. A McIntyre-Stennis proposal to continue this work has been submitted. (Don Crews-Colorado State University).

d. A project is being launched during the fall of 1967 to study the growth impacts of dwarfmistletoe on western hemlock. Basic attack will be by checking total volume production of infected and healthy trees in the same stand. Completion of this project may not be for 1½ to 2 years. (Russell, DNR-Washington, Olympia).

VI. Ecology - no reports

VII. Control - Chemical

a. The first treatment evaluation of Bandelier National Monument dwarfmistletoe chemical control test plots was made in the fall of 1966. Present indications reflect very little difference, if any, in control achieved from an application of fuel oil carrier alone and/or any of the herbicide concentrations listed in the 1966 report. The 1967 conferees will visit the test area during a field trip to Bandelier National Monument on September 13. (Lampi-National Park Service-Santa Fe).

b. The California Region has been testing herbicides for the control of dwarfmistletoe on ponderosa and Jeffrey pine since 1962. At present we have two active series of tests.

1962 TESTS
The first series, begun in 1962, is a secondary screening of three herbicides (2,4-DA, 2,4-DP, and 4-CPA), which were previously tested by Clarence Quick at the Pacific Southwest Forest Station. Each herbicide was applied at three concentrations (0.1%, 0.2%, 0.3% in stove oil), and at seven times during the period from May 2 to October 16. Each test consisted of 10-15 selected dwarfmistletoe

plants, each of which was on the bole of a separate tree. In all, 711 trees and plants were treated. Results after five growing seasons are summarized below and in Table 1.

1. All herbicides at all concentrations killed some trees, and often a prohibitive number. However, all were far less damaging during the dormant period. (Some test trees were severely infected, and perhaps were more susceptible to the herbicides than trees treated in operational control work would be.)

2. Considering only trees that were not killed, all herbicides at all concentrations suppressed shoot production on some and often most of the dwarfmistletoe plants treated.

3. According to present results, 2,4-DP is the most promising of the three herbicides. In general, 4-CPA was less damaging than 2,4-DP but also less effective. At the two higher concentrations, 2,4-DA was much more damaging than 2,4-DP.

4. Except for 2,4-DA, there were not great differences between the low and the high concentrations.

Although further development work would be needed, it seems likely that an operational chemical-control procedure employing these herbicides could be developed. Such a procedure would include the following features: 1. Direct treatment only. 2. Restrictive limits as to how severely infected the tree could be. 3. Treatment restricted to the late summer and winter.

1966 TESTS.
The second series of tests, begun in 1966, is a secondary screening of two additional herbicides: 2,4,5-TB applied directly to the dwarfmistletoe in an oil spray, and 2,4,5-TP applied directly in an oil-water emulsion and requiring translocation. Both herbicides were applied at two concentrations at two growth periods (dormant and prior to the spring growth surge) at two locations (Klamath and San Bernardino National Forests); and on two host species (ponderosa pine on the Klamath, and Jeffrey pine on the San Bernardino). A repeat test of the 2,4-DA was included to tie the two series of tests together. Each test consisted of 20 dwarfmistletoe plants on 20 separate trees; in all, 640 trees were treated, including controls.

Although first-year results will not be read until the fall of 1967, an informal examination of the Klamath plots indicate little, if any, tree killing.

Table 1.--Test Results in 1967, 2,4-DP, All Concentrations

Treatment date 1	Trees treated 2	Trees killed 3	Dwarfmistletoe plants treated 4	Dwarfmistletoe plants suppressed 5
5/2/63	30	33%	20	95%
7/3/62	30	27%	22	91%
7/11/62	30	43%	17	94%
7/24/62	45	36%	29	100%
8/2/62	36	36%	23	91%
8/23/62	36	17%	30	90%
10/16/62	30	17%	25	64%

Column 2: Total number of trees and dwarfmistletoe plants treated; one treated plant in each tree.
Column 3: Includes some trees in which only the top was killed.
Column 4: Does not include plants on killed trees.
Column 5: No visible shoots since treatment.
(? Div. of Timber Management-California).

VIII. Control - Biological--no reports.

IX. Control - Silvicultural

a. All permanent sample plots in the Grand Canyon dwarfmistletoe control area and the three plots scheduled for 5-year reexamination on the Mescalero Apache Indian Reservation have been re-examined. The data will be analyzed this winter. (Lightle-RM).

b. The first cleaning of the plots established in 1962 on the Colville National Forest to study the effects of thinning and eradication (reported in the 10th Proceedings) has started. All infections removed in this process are being stored for study of latent development, primarily in Douglas-fir and western larch. (Wicker-IM).

X. Surveys - No report.

XI. Miscellaneous - no report.

Needed Research

No suggestions.

FOREST DISEASE RECREATION HAZARD COMMITTEE

G. W. Wallis, H. R. Offord, and L. A. Paine, Chairman

Report to the 15th Annual Work Conference

Consequences of Recreation Site Tree Failures

Annual use-season property damage was estimated to be in excess of $25,000 on Federal recreation sites in California. Every reported tree failure represented an average property loss of $261, including both accident and non-accident failures.

One injury or fatality was reported for every 21 failures, with property damage in one out of every two to three failures (1964-65-66). Reported conifer failures resulted in one injury or fatality out of every 13 failures--including limb failures.

Guidelines for Removal of Hazards

The optimum allocation of hazard control efforts was evaluated on the basis of type of hazard, frequency of failures, damage caused by failures, and costs of control. Reduction of bole hazard was found to be the single most effective form of control for species other than oaks. Control of limb hazard, except in oaks, was least effective. Uprooting was the most common type of failure and caused the greatest number of accidents and property losses, but efforts at control, except for leaning trees, are only slightly more effective than limb hazard control.

Success in Detection and Removal of Hazard

A new study will attempt to measure practical success in detection removal of each type of hazard through a comparison of hazards removed and failures which occur despite hazard control. This is intended to provide a basis for elaboration of needed detection techniques.

Needed Research

After definition of conditions which constitute high risk, the problem of in-season uprooting is most important. Conditions which lead to uprooting need to be defined and evaluated, and techniques for field detection of these conditions are needed. A total list of known current projects in the recreation hazard field is included in the appendix.

Comments on Responsibilities of Management and Pathology

As Mr. Carlson noted at last year's meeting, the California Supreme Court has held that the owner is required to have and exercise a superior knowledge of hazardous conditions. This implies a responsibility for careful inspection by qualified personnel with appropriate training and techniques, as well as for the removal of detected hazard Pathologists share in the responsibility through their obligation to provide better tools and techniques to management and to point out problems requiring special attention.

Are we discharging these responsibilities? Timber sale procedures are set out in minute and voluminous detail with provisions for conformance. Control of hazardous trees may be disposed with in a couple of lines without guidance in methods of hazard detection or evaluation of risk, without goals in terms of acceptable levels of risk, and without means of attaining a reasonable uniformity in control practices. If administrative guidance leaves something to be desired, our own contributions are in the same category. Management badly needs detection and evaluation techniques as well as control methods which can be applied to valuable trees and stands without destroying the values of the site. As informed specialists, we have the additional obligation of bringing both inadequate administrative procedures and management needs to the attention of recreation administration. (Paine, USFS, Berkeley.)

INTERIM PROGRAM CHAIRMAN'S REPORT

(H. H. Bynum)

Suggestions for subject matter of future meetings

a. Disease problems of the meeting area.

b. Air pollution, including the legal aspects.

c. Biological control of forest diseases.

d. Fungus physiology.

e. Host-parasite relationships as regards the mistletoes.

f. Remote sensing.

g. Epidemiology of forest diseases.

h. The role of plant pathogens in forest ecosystems dynamics.

i. Information retrieval systems.

j. Population dynamics.

k. Impact of foliage loss on tree increment.

l. Original research - individual papers.

m. Research on mycorrhiza.

n. Tree breeding for disease resistance.

o. Modern techniques of pathogen identification.

p. The pathologist's role in managing stands for other than timber values.

q. Pathological data required for modern computer economic analysis of how to manage forest lands.

r. Elytroderma - taxonomy, host-parasite relationships, and control.

s. Role of the researcher, pest control pathologist, and land manager in forestry.

APPENDIX VII. -- PROVISIONAL MEMBERSHIP LIST

(* Indicates members present at Fifteenth Conference)
(** Indicates guests at the Fifteenth Conference)
(+ Indicates honorary life member)

Adams, Mr. David, Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon 97331

Aho, Mr. Paul E., Forestry Sciences Laboratory, 3200 Jefferson Way, Corvallis, Oregon 97331

Andrews, Dr. Ed A., Plant Science Division, School of Agriculture, University of Wyoming, Laramie, Wyoming 82070

* Andrews, Dr. Stuart R., Forest Disease Research, Rocky Mountain Forest & Range Expt. Sta., 240 Prospect Street, Fort Collins, Colorado 80521

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\+ Bedwell, Dr. Jess L., 1908 N.E. Schuyler, Portland, Oregon 97212

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Bloomberg, Dr. Wm. J., Forest Research Laboratory, 506 W. Burnside Road, Victoria, British Columbia

** Bond, Leroy, U.S. Forest Service, 517 Gold Avenue, SW, Albuquerque, New Mexico 87101

Bongberg, Jack W., Division of Forest Pest Control, U.S. Forest Service, South Building, Washington, D. C. 20250

Bradshaw, Mr. R., Survey Division, British Columbia Forest Service, Victoria, B.C.

Brandt, Dr. R. W., Forest Disease Research, U.S. Forest Service, Washington, D. C. 20250.

Bravo, Senorita Rosario Vasquez, Forest Mycology Section, Instituto Nacional de Investigaciones Forestales, Progreso 5, Coyacan 21, D.F. Mexico

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Mr. Conrad P. Wessela
Dr. Ernest Wright

PAST MEETING PLACES AND EXECUTIVE COMMITTEES

Meeting		Executive Committee		
Date	Place	Chairman	Secretary-Treasurer	Program Chairman
1953	Victoria, B. C.	R. E. Foster	- - -	- - -
1954	Berkeley, California	W. W. Wagener	P. C. Lightle	- - -
1955	Spokane, Washington	V. J. Nordin	C. D. Leaphart	G. P. Thomas
1956	El Paso, Texas	L. S. Gill	R. W. Davidson	V. J. Nordin
1957	Salem, Oregon	G. P. Thomas	T. W. Childs	R. L. Gilbertson
1958	Vancouver, B. C.	J. W. Kimmey	H. R. Offord	A. K. Parker
1959	Pullman, Washington	H. R. Offord	R. E. Foster	C. G. Shaw
1960	Centralia, Washington	A. K. Parker	F. G. Hawksworth	J. R. Parmeter
1961	Banff, Alberta	F. G. Hawksworth	J. R. Parmeter	A. C. Molnar
1962	Victoria, B. C.	J. R. Parmeter	C. G. Shaw	K. R. Shea
1963	Jackson, Wyoming	C. G. Shaw	J. E. Bier	R. F. Scharpf
1964	Berkeley, Calif.	K. R. Shea	R. F. Scharpf	C. D. Leaphart
1965	Kelowna, B. C.	J. E. Bier	H. S. Whitney	R. V. Bega
1966	Bend, Oregon	C. D. Leaphart	D. P. Graham	G. D. Pentland
1967	Santa Fe, New Mexico	A. C. Molnar	E. F. Wicker	L. C. Weir