Proceedings of the Eighth Western International Forest Disease Work Conference
Centralia, Washington November/December 1960

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FOREWARD

The Eighth Western International Forest Disease Work Conference was held at Centralia, Washington from November 29 to December 2, 1960. Fifty-eight members and 7 guests attended. The attendance was the highest for any work conference to date, and greatly exceeded the previous high of 40 members at the Fifth Conference (1957) in Salem, Oregon.

The theme of the Eighth Conference was "Problems and Possibilities." Three topics under this general heading were ably discussed by four panels.

The highlight of the meeting was on November 30, when J. E. Bier, on behalf of conference members, presented a portable rostrum and gavel to Mrs. Kirstine Buckland in memorium of Donald C. Buckland. Mrs. Buckland thanked the conference and turned the rostrum and gavel over to Chairman Art Parker. These will be brought to all future meetings of our conference, and will be a continuing inspiration of the thoughts and ideals of Don Buckland.

The annual banquet was held on November 31 in the Lewis and Clark Hotel. Dr. Vidar Nordin was the after dinner speaker and entertained everyone with an interesting illustrated talk on his "European Impressions," particularly in reference to poplar growing and other popular activities in Italy.

On December 1 trips were made to the Weyerhaeuser Company's Forestry Research Center at Centralia and to the large sawmill, plywood plant, and pulp mill complex of the Weyerhaeuser Company at Longview, Washington.

At the opening session, Robert McMinn was appointed interim program chairman. Bob canvassed the members during the conference and at the final business meeting presented a report on proposed guidelines for future meetings (see Appendix V). This information will be extremely useful to those concorned with planning future meetings.

CHAIRMAN'S WELCOME

Fellow members and guests, I take great pleasure in welcoming you to our Eighth Western International Forest Disease Work Conference. As in the past, the program prepared for us this year provides a hub of semi-formality around which free and informal discussion revolves. Your program committee, consisting of J. R. Parmeter, Jr. as chairman, K. R. Shea, J. W. Roff and R. V. Bega, have focused our attention on three topics, two of which have received relatively little attention at previous conferences and frequently in research programs; namely, sporadic disease outbreaks in young stands, and nursery and transplant disease problems. The third topic, covered by two panels, the deterioration of dead and felled timber and timber products, is one you have requested on several occasions in the past. Because the speakers are expected to raise more questions than they answer we may well call the theme of this conference "Problems and Possibilities." Although your program committee and panelists have put a great deal of thought and effort into providing the hub of semi-formality for us, the success of the conference as a whole depends on your active participation in the discussions.

A. K. Parker
Conference Chairman

PANEL I. SPORADIC DISEASE OUTBREAKS IN YOUNG STANDS

John R. Parmeter, Jr., Moderator

SPORADIC DISEASE OUTBREAKS IN YOUNG STANDS

John R. Parmeter, Jr.

The attitudes and concerns of foresters have changed greatly in the past few years. Old-growth orientation has given way rapidly to second-growth orientation. Each year large numbers of small landowners are investing in forest enterprises, especially in Christmas tree production and in small tree farms. Increased land values and increased operating costs necessitate placing an ever greater premium on maximum utilization of land and the crop it supports. These developments make it increasingly important that each acre of forest land support as near to the optimum number of trees as is possible and that each tree be as near to the maximum quality as is possible. Diseases that reduce either the number or the quality of trees in young stands increase in importance accordingly.

We can anticipate many of the disease problems that will affect young stands in a given area. White pine blister rust, dwarf mistletoes, many root diseases, and similar "perennial" disease problems often occur with predictable regularity and predictable consequences. Growers can, therefore, be prepared to undertake control or to sustain calculated losses from such diseases. Many other disease problems are at present unpredictable. The Christmas tree grower who tends his plantation for ten years and then suddenly finds most of his trees rendered unsaleable by a canker or needle disease sustains a loss he cannot anticipate. The timber grower who manages a well-stocked stand for 40 years and then suddenly loses many trees to a disease that he did not know existed is understandably disturbed Such unpredictable, sporadic disease outbreaks can be very serious.

The line that divides those diseases that occur regularly from those diseases that occur sporadically is difficult to distinguish. It is likely that all diseases fluctuate in intensity from year to year. In addition, damage from a given disease may be usual in one region and extremely rare in another. We can recognize in a general way, however, those diseases that occur at such infrequent intervals as to be unpredictable. It is with these diseases and the damage they cause that we are concerned this afternoon.

Damage resulting from sporadic disease outbreaks varies according to the intended use of the crop and to the type of disease. In general, Christmas trees are most liable to damage, since any

extensive loss of needles, branch dieback, or deformation of the crown may render trees unsaleable temporarily or permanently. Diseases which kill trees or deform the main stem may be equally serious in Christmas tree or timber stands. We have little information as to the magnitude of such losses in the West. This is information that I think we should have.

We need also to know something about the occurrence of these diseases. We often accept as a fact that a given disease occurs during drought years, and yet we find that it does not occur during all drought years. We may accept as a fact that another disease occurs during years of exceptional rainfall, and yet we find that it does not occur during all such "wet" years. Why do these diseases show up only during some "wet" or "dry" years but not during others? Our inability to predict such outbreaks points to deficiencies in our present knowledge of epidemiology. Elimination of these deficiencies might provide a basis for reducing losses from sporadic diseases.

It seems to me that control of sporadic diseases depends in large part on our ability to anticipate disease outbreaks. Yearly protective treatment of Christmas tree or timber stands is clearly impractical. Protection might be quite practical, however, if the treatment were restricted to those years in which outbreaks were imminent. Success would depend on the accuracy with which outbreaks could be predicted.

These problems and their possible solution bring up several questions worth discussing:

1. What are the major sporadic diseases of young stands in the West?

2. What is the impact of these diseases on forest productivity?

3. Are these diseases receiving adequate attention?

4. If increased attention is advisable, what direction should it take?

5. Is control of these diseases practical, and if so, is it justifiable?

6. If control is justifiable, what are the control possibilities and how should they be explored?

Our panel of experts has been brought together to discuss some of these problems in various regions of the West. Alex Molnar will discuss first some general aspects of sporadic diseases. Gardner Shaw will follow with a detailed discussion of two

specific disease problems, and Willis Wagener will complete the panel with a discussion of examples of sporadic disease outbreaks and factors associated with these outbreaks.

SPORADIC DISEASES IN YOUNG STANDS OF BRITISH COLUMBIA

A. C. Molnar

In considering sporadic diseases, rather than providing a list for British Columbia, with annotation of their importance and what we know about them - the list would be long, the annotations skimpy - I decided to make a rather generalized evaluation of how we stand with them. In doing so I would like to consider them in two groups: those which we readily recognize as primary agents (Group I) and those which are secondary agents requiring host weakening (Group II).

Group I

A. Characteristics

1. Obligate parasites or those sufficiently aggressive that host symptoms are marked and generally specific.

2. The symptoms and signs add up to the identity of the causal agent, generally.

3. The factors promoting outbreak levels of infection do **not include host weakening as the major item, more important are weather factors (macro- and micro-climate),** alternate host status, stand conditions as they affect weather factors, and stand composition. Host weakening is not excluded, of course.

To satisfy a craving for lists I submit a brief list of diseases or types of diseases which would fit in this group.

Native blister rusts

Foliage rusts

Douglas fir needle blight (Rhabdocline)

Yellow pine needle blight

Other Hypodermataceous needle casts

Atropellis canker

There are others but these are perhaps our more important groups.

In considering where we stand in regard to understanding our sporadic diseases I would like to examine Group I briefly under the following headings: detection, damage appraisal, life history, infection and epidemiology, and control.

Detection (Mycology, symptomatology, pathogenicity)

We are in a fairly good position on this phase. Most of the diseases in Group I have been studied in some detail; their nomenclature has been clarified, they have been adequately described; pathogenicity tests have been reported and confirmed. We have accumulated considerable information on their geographical distribution.

Damage appraisal

We recognize at least our currently important and damaging diseases by their signs and symptoms. We know how much damage some can cause at least on the individual tree and individual acre and with reference to particular products. But there are still many questions and requirements.

1. Just how damaging are our foliage diseases in terms of increment loss, and predisposition to secondaries?

2. Beyond total enumeration how can we estimate losses at reasonable cost in time and money?

3. What are the effects of broom rusts; can we ignore them?

4. Etc.

Life history

With a few gaps, the life histories of most are quite well understood. Exceptions will no doubt come to mind.

Infection and epidemiology

Here we come upon rockier ground. In general terms, we understand the factors governing spore dispersal and infection. Details have been worked out for a few diseases. The factors controlling epiphytotic infections are less well defined, however.

Questions

1. What are the conditions for successful spore dispersal and infection?

2. We have a good idea, particularly with foliage diseases, that micro-climate is critical. But we have little control, as yet, over weather. What about stand factors, how do they affect micro-climate?

Control

Nothing is being done to control sporadic diseases in British Columbia at present.

I have selected one example of a sporadic disease in Group I on which some research progress has been made, and which even at our present level of management holds some promise for control. I have to admit it is a special case but others will, in time, fall into this category.

A. Detection

1. Where do we stand

The disease has been adequately described and the nomenclature clarified. This aspect is under control.

B. Damage appraisal

In terms of Christmas tree management the basic information for damage survey is available. Sampling studies have not been carried out in the Interior but it is likely that sequential sampling can be used. Damage can be predicted for a year ahead, which may prove extremely important for control of this disease.

In terms of stands other than Christmas trees the picture is less clear. In recent years it has come to our attention that pole-sized stands in the Kootenays may suffer severe defoliation from Rhabdocline. Appraisal on material of this size is much more difficult. Many questions require clarification:

1. What are the effects of defoliation in terms of increment loss and how does this loss relate to the degree of defoliation.

2. Perhaps even more important is the impact of defoliation as it affects host vigor.

a. Does it commonly result in direct mortality?

b. Predispose trees to secondaries?

c. And conversely are low vigor trees in the stand more susceptible to infection by Rhabdocline than high vigor trees?

There seems to be plenty of scope for crafty infestigation here.

C. Life history

The life history of Rhabdocline appears to be well understood with perhaps the exception of its development within the host needles.

1. Is there delayed development to carry symptoms and fructification over to older needles?

2. Is there sufficient carry-over to carry appreciable inoculum over from a poor to a good year?

D. Infection and epidemiology

Some heartening progress has been made on this phase of studies on Rhabdocline.

1. The requirements for dispersal of the bulk of the spores during maturation of apothecia is a period of 48 to 96 hours at 100% humidity.

2. It seems probable these conditions also provide suitable environment for infection but this still remains to be proved.

3. Outbreak levels of infection depend on adequate inoculum (a sufficient number of mature apothecia) maturing during a period of suitable conditions for dispersal and infection. Thus, outbreak levels would not be expected to follow a single year favoring spore dispersal and infection, for inoculum would have to build up.

There are still questions awaiting answers:

1. Conditions for infection require to be positively defined.

2. Phenological development of the host in relation to infection. I believe Dr. Parker has made some progress on this aspect of the problem.

3. Are there resistant strains, and if so, what is the nature of resistance? Can this be used for control. With Christmas tree stock there may be scope for stand manipulation.

E. Control

Nothing positive can be reported concerning control, but the prospects are reasonably bright. The industry seems willing and anxious

to spend money on control and since outbreak years can be predicted direct control holds some possibilities. Acti-dione is currently being tested and inevitably will bring forth further questions. Other fungicides and antibiotics may be worth testing. Some of the questions concerning acti-dione may be:

1. Does it prevent infection?

2. Will it arrest or prevent development of existing infection and thus prevent the development of symptoms? That is, would it be worth spraying once infection has taken place?

3. What are the possibilities of application to older stands under sawlog management?

4. Not all stands appear to be equally susceptible to infection or at least to outbreak build up. What is the effect of stand form? - composition? - topography? Can we rate stands according to hazard?

If we answer all these questions for Rhabdocline it seems probable that our progress on other similar diseases will be much easier.

Group II

From a research point of view as well as from the viewpoint of control, the diseases in Group II are more troublesome and I suppose more challenging. They have the following characteristics:

1. They are facultative parasites and ordinarily quite weakly so, with their symptoms often poorly defined, masked or confused.

2. The association of signs (fructifications) and observed disease symptoms do not necessarily add up to cause of the condition.

3. Host weakening or at the least definite local injury is required for successful infection and disease development.

4. Because these organisms and their symptoms often develop in association with direct host injury, it is difficult and often impossible to determine where direct injury leaves off and the damage caused by the organism starts.

Nevertheless, few pathologists would dispute that a great many organisms recognized by these characteristics are extremely damaging under some, often little understood, conditions. We can point to many examples which would lead us to believe that they shall

become even more important with more stand manipulation and increasing reliance on artificial regeneration. But we really don't know how important, nor have we adequately defined the conditions under which they become a problem.

Just a few examples:

1. Armillaria mellea and probably a number of other root rots.

2. Cytospora sp.

   Cenangium ferruginosum

   Pullularia pullulans

   Myxosporium sp.

   Phomopsis lokoyae

   Stereum sanguinolentum

And probably a great many other fungi we have found associated with disease symptoms but for which we have not demonstrated pathogenicity.

How do we stand with them research-wise? Obviously on pretty shaky ground.

Detection

The presence of a great many we can recognize on the basis of fructifications, while many others we encounter only in the form of cultural isolates.

Questions to resolve:

1. Techniques of pathogenicity testing, particularly standard methods for different types of host weakening.

2. Descriptions and keys for more rapid identification of cultures.

3. Mycological studies particularly in the Fungi Imperfecti.

Damage appraisal

We can appraise damage on the basis of visible symptoms but we cannot assign cause with any degree of assurance. We are constantly being forced back to first principals. Much more research needed on other phases first.

Life history

We understand many in general terms but few are clearly defined. We know their hosts by association and find that they are most commonly not host specific to add to the complexity of the problem.

Infection and epidemiology

We have a long way to go. However, Dr. Bier's and Dr. Bloomberg's work on bark moisture conditions in relation to infection have opened up at least one promising line of approach. But -

1. Are there other important influences or types of host weakening which may be operative even when bark moisture relations are favorable.

2. Is host predisposition the only condition controlling epiphytotic build up. Can we assume that inoculum is always plentiful. What about microclimate. Regardless of host weakening; may losses be reduced if microclimatic conditions do not favor infection?

Control

Despite the dearth of factual evidence on the behavior of organisms in this group and on their relation to their hosts, we have some weapons for their control based on their broad characteristics. In particular, the fact that they depend on host weakening or injury for successful invasion gives us a weapon for their control, namely maintaining stand high vigor and keeping injury to a minimum. Unfortunately, even under intensive and highly informed forest management, this is not always possible for poor sites, weather injury, and, insect damage, among other uncontrollable factors may provide predisposition. It is necessary, therefore, to be prepared to devise methods for reducing losses if we cannot prevent predisposition.

SPORADIC DISEASES IN YOUNG STANDS OF THE INLAND EMPIRE REGION

Charles Gardner Shaw and B. D. Thyr

The symptoms of two serious foliage diseases, needle cast and blight of western white pine, in the Inland Empire have recently been described (1). Needle cast appears to be sporadic in its occurrence, while blight is chronic and endemic.

The severity of needle cast appears to be correlated with the amount of precipitation occurring during the relatively short period in which the only spore stage produced by the needle cast fungus matures. In contrast, the fungus associated with blight produces spores throughout the year and no correlation between environmental factors and the severity of the disease has been noted.

The hysterothecia of the needle cast fungus, Hypodermella arcuata Darker, mature during July and August. The time apparently depends to some extent on the locality and the site. Examination of hysterothecia in late June failed to give any indication that ascospore discharge had as yet occurred. Water mounts of hysterothecia collected between mid-July and mid-August characteristically contained abundant ascospores free from the asci as well as many asci still containing ascospores. Similar mounts made from material collected in September disclosed very few ascospores and the asci still discernible were empty. During September and October the dead needles upon which empty hysterothecia are present are cast. In severely infected stands the branch terminals have a "lion's tail" resulting from the contrast between the full complement of current season's foliage at the branch terminal and the denudation of the remainder of the branch by the needle cast. Frequently the defoliation is so severe that only the current season's needles remain in the upper crowns. In 1959 some white pine stands showed evidence that severe casting of needles had occurred for three or more previous years.

On the basis of the tremendous amount of inoculum obviously present during the 1959 growing season, one might have felt safe in predicting a heavy infection again this year. Such did not prove to be the case.

In some areas where needle cast was prevalent in 1959, it could not be found in 1960. In the Clarkia-Bovill and Headquarters areas of Idaho where the disease was severe in 1959, it was much less prevalent in 1960. While in 1959 whole stands had a reddish-tan discoloration in the upper crowns, this year this discoloration was in most instances limited to individual trees or at most small groups of trees. Furthermore, the percentage

on infected needles was obviously much reduced. In 1959 it was not uncommon to find trees, the upper terminals of which had almost 100% of the current season's needles browned and bearing hysterothecia. Examination this year of branch terminals on some of the same trees examined in 1959 disclosed percentages of infection from as low as 2-3% to about 30% but not higher.

For an explanation of this marked decrease in the amount of infection in spite of the large amount of inoculum present a year ago, climatological factors were considered. We have just started to analyze weather data for north-central Idaho. The precipitation records for the past four years may provide a partial explanation for fluctuations in the severity of the disease. Figure 1 shows that several showers occurred between July 10 and August 20 in 1956, 1957, and 1958, but that in 1959 there was a prolonged period between July 10 and August 19 with essentially no precipitation. This prolonged dry spell in 1959 coincided with the maturation of the ascospores of the fungus, and probably prevented their dissemination.

The spring of 1959 was cool and, as indicated by direct observations, the asci had not matured sufficiently to be ready to discharge ascospores during the month of June, when several intermittent showers occurred.

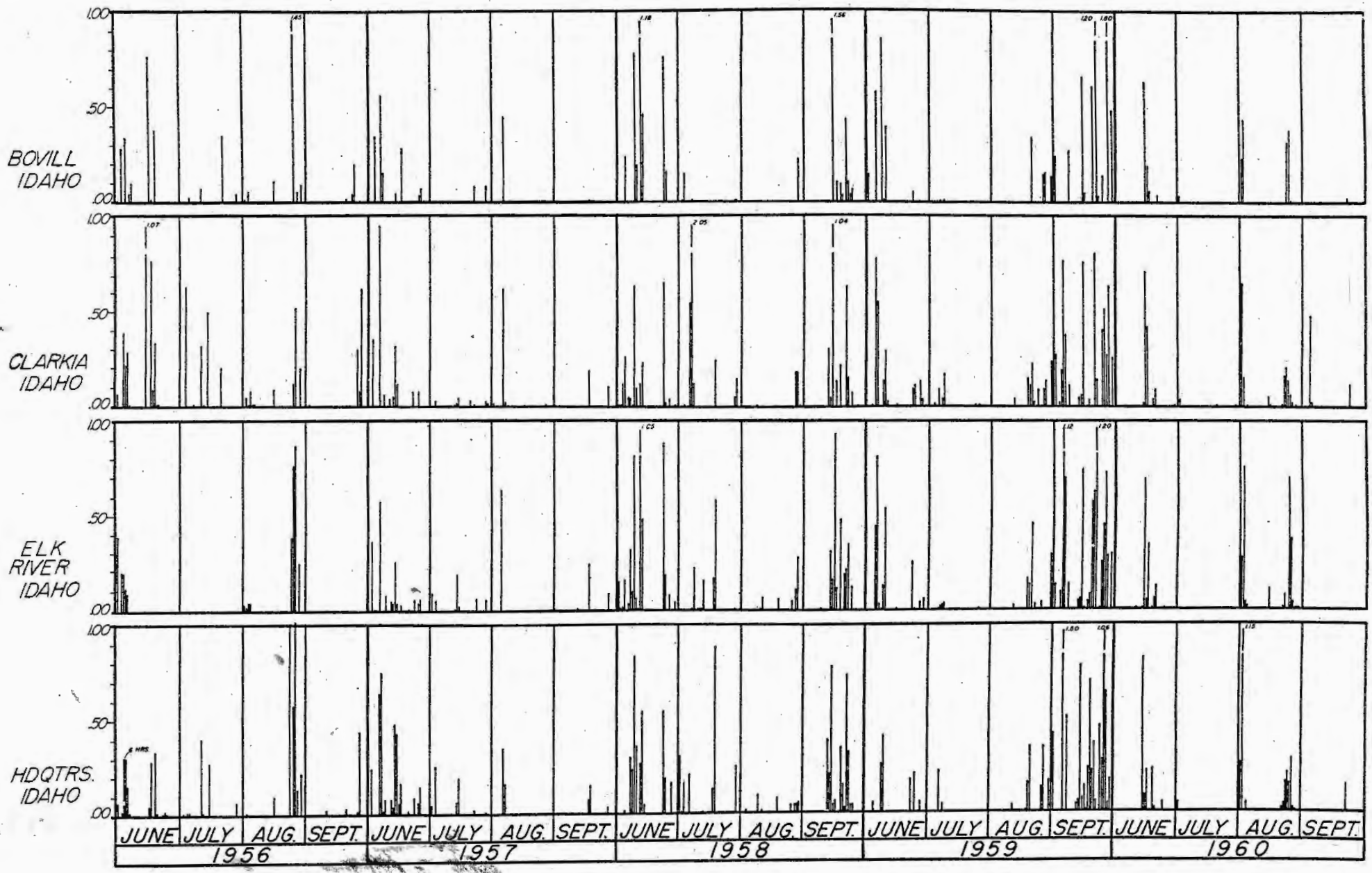
An additional point of importance is that the temperatures were extremely high during the prolonged dry spell in June and July of 1959, and humidities extremely low. Nothing is known as yet concerning the maximum temperature that the fungus can endure.

The fungus consistently associated with needle blight, tentatively identified as a species of Lecanosticta, has a markedly different sequence of development from Hypodermella arcuata. The erumpent stromata first appear on the discolored tips of needles 11-13 months old. As infected needles subsequently become discolored for their entire length, additional spore-producing stromata develop. Infected, two-year old and older needles bear stromata over their entire length. Blight infected needles are not cast; furthermore sporulation occurs on the stromata at all times of the year on infected needles from ten months to four years of age.

Obviously we have an entirely different situation in connection with this fungus than with the needle cast fungus. The inoculum of the needle cast fungus matures at a particular time of the year, namely during July and August, spore discharge and infection apparently occur during this period and then cease. If conditions are not favorable for spore discharge or infection, or both during this short interval, infection does not occur and cannot occur at other times of the year.

The blight fungus, however, produces inoculum all year long. This suggests that infection can occur at any time of the year when

PRECIPITATION RECORD JUNE – SEPT., 1956–1960



climatic conditions are favorable for infection.

These differences in the life cycles of the two fungi involved are reflected in the prevalence of the two fungi this year. The marked decrease in cast has already been discussed; in contrast blight is as prevalent this year as last. Since spores are produced throughout the year and hence inoculum is available whenever environmental conditions are favorable marked changes in the amount and severity of infection from year to year are not to be expected.

These observations can probably best be summarized by stating that: (1) the life cycle of a pathogen, and particularly the duration of the period of sporulation, may provide a clue as to whether the occurrence of the disease it causes will be sporadic and spectacular, or chronic and more or less static; (2) for a pathogen producing but one type of disseminule during a brief period of its annual cycle, climatological factors must be favorable for spore liberation, dissemination and germination, and for penetration during the period of sporulation for heavy infection to result. For an actual epidemic to develop, favorable environmental conditions apparently must coincide with sporulation for at least two or more years in a row. If these factors do not coincide, a marked reduction in the prevalence may be expected the next year.

Literature Cited

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SPORADIC DISEASES IN YOUNG STANDS IN CALIFORNIA AND NEVADA

Willis W. Wagener

The category "sporadic diseases" ordinarily suggests to the forest pathologist diseases of an infectious nature that occur at irregular and widely-spaced time intervals. In addition to these, however, we have a number of injuries of non-infectious origin that may sporadically damage our forests. I will begin my topic by citing a few types of damage of this sort that have occurred in the California-Nevada area and may occur elsewhere.

Injuries of Non-Infectious Origin

Top dieback of conifers: The injury specifically referred to under this heading is a dieback of the tops of conifers that becomes evident in the spring and may affect trees of various sizes, although most commonly seen in thrifty young-mature and mature specimens (7).

The first case that brought this type of injury to our attention was a top dieback of Coulter pines and a few bigcone Douglas-firs in a recreational area on the Angeles National Forest in southern California. It was presumed by local forest officers to have resulted from bark beetle attacks in the affected tops, but there were enough non-standard aspects of the situation for me to be invited to accompany forest entomologist F. P. Keen for an on-the-ground examination. When we arrived we found that the dead tops had been cut from some 315 affected trees as an insect control measure.

Almost without exception the tops contained bark beetles of some type, but Keen pronounced them to be species of Ips seldom regarded as primary. Moreover, in some tops the infestation was so light that it seemed questionable whether the beetles present could be solely responsible for the dieback. Another feature that did not fit in well with the beetle-causation hypothesis was the presence below the dead portion of a zone in which beetles were scarce or absent and the inner bark was heavily resin-infiltrated. We also found that in some of the younger trees the bark below the dead portion could be stripped off easily in ribbons several feet long. On the under side these ribbons or strips showed a layer of white, thin-walled, large-celled tissue, apparently parenchymal in character.

Another noticeable aspect of the dieback was that only tops fully exposed to the sun were affected. Shaded or part-shaded tops were not killed back.

There is not time to dwell on all the interesting aspects of the case. Suffice it that we found typically affected tops without any beetles present and I climbed and investigated one tree with a double top, the one on the south side dead and the one on the north side still alive. However, the latter top showed injury to the inner bark of the same type as that in the south top but not as severe.

Fortunately, weather records for the entire preceding year were available from a lookout within the affected area, as this was during World War II and the lookout was kept manned as part of the aircraft warning system. From these records it was found that nearly two weeks of very warm weather for that time of year had occurred in mid-January. This was followed by a drop to 20° F. in a little more than a day's time and one of the heaviest snowfalls ever recorded there. Without much question it was this period of abnormally mild temperatures followed by the quick drop to below freezing that had caused the injury.

We have since found that this type of dieback is not uncommon in pines and associated conifers in California. More than once it has been ascribed by entomologists to beetles such as Pityophthorus spp. because these insects later invaded the injured tops. Invariably the affected trees have been making rapid growth and the tops are fully exposed to the sun. Residual stands of pine that have been selectively logged, resulting in growth release, are particularly susceptible. Often the injury occurs in the autumn, in consequence of a sudden cold wave following warm fall temperatures.

There also have been cases of top dieback affecting more than one conifer species that apparently are not the result of sudden cold. We still do not have an adequate explanation for them. A noteworthy case occurred above Placerville, California in 1946. Ponderosa pine and incense-cedar were the chief species affected, along with a few Douglas-firs and digger pines. At the Institute of Forest Genetics over 200 bagged cones were lost on a single ponderosa pine top. A hygrothermograph record from within 200 feet of this tree gave no indication that temperature changes were responsible for the damage. In one ponderosa pine top examined in considerable detail the cambium and adjoining phloem of the inner bark were darkened in a gradually diminishing degree for about 12 feet below the lower limits of dying.

Closely related to the top dieback from sudden cold is a type of so-called winter injury to plantations or rapidly growing natural reproduction. As a rule only scattered trees are affected, always in locations fully exposed to the sun. The inner bark of such trees is heavily resin-infiltrated near the lower limits of the dying and the damage seems to be the result of incomplete dormancy of the inner-bark tissues, resulting in their damage by cold.

Spring dieback of conifers following fall drought:

Our attention in California was first drawn to this type of damage in the spring of 1930. The preceding fall was extremely dry, with no precipitation until December 8 in many parts of the state. Early the following spring dieback began to show up in a number of coniferous species, including Monterey pine and western juniper, but was most extensive in Douglas-fir and incense-cedar. Most of it occurred at fairly low elevations in the North Coast Ranges and in the Siskiyou Mountains.

In most of the affected Douglas-fir saplings and poles the dieback became arrested about the end of April and the remaining portions of the crown and stem subsequently recovered. In incense-cedar, however, the dieback continued, once it was started, until the entire tree succumbed.

In 1937 a similar dieback occurred but in a more restricted area, including parts of the Rogue drainage in southern Oregon. Following this, no similar dieback was noted until the spring of this year, when it again appeared in parts of northern California and on the Kern plateau. On the basis of our experience with the previous cases we were able to predict in advance that this type of dieback might be expected in the spring of 1960.

A feature of this dieback is that at the time it occurs there is usually plenty of moisture in the soil and has been for several months. Why, then, does it take place? Some of you are familiar with my hypothesis on this point. For those who are not I will reserve its presentation for the discussion period.

Heat scorch of deciduous foliage: In 1942 Mielke and Kimmey (5) reported a foliage scorch of California black oak and occasional other species the previous summer in parts of the Sacramento and Klamath drainages in northern California. No evidence could be found of an associated pathogen, and after a study of the weather records from within the affected area they concluded that the scorch was the result of a sudden hot spell in early July, following an unusually cool and moist spring.

No similar injury as extensive as this has since been noted in the State, but there is every reason to believe that the conditions responsible for the scorch in 1941 will sometime recur, with similar effect.

Salt spray damage: Redwood is a species relatively sensitive to salt damage of the foliage. However, the species extends to marine headland situations in a number of places along the north coast of California.

Following heavy on-shore winds in February 1959, conspicuous reddening of redwood foliage was noted on trees in exposed locations along the coast north of Eureka, California. Often all foliage on the windward side of the trees was killed. Associated Sitka spruces showed little or no injury.

Not all excessive on-shore winds carrying salt spray result in salt injury, but only those not accompanied by sufficient rain to wash the salt deposits off before they can inflict damage. These occur at relatively infrequent intervals, and as a result the damage is sporadic in occurrence.

Sporadic Diseases of Infectious Origin

Diseases of this type may be divided into two classes: (1) Outbreaks resulting from particularly favorable or unfavorable climatic conditions occurring at infrequent intervals, and (2) outbreaks developing from highly favorable but non-permanent host conditions or associations. Combinations of the two sometimes occur.

Examples of the two classes can be cited from the history of occurrence of western forest tree rusts in California and Nevada.

Pinyon rust: This rust, Cronartium occidentale, is endemic in pinyon pine at various locations in the pinyon woodland, extending southward from the Carson River in California and Nevada, wherever favorable associations occur with the desert gooseberry, Ribes velutinum. In some locations new infections in the pinyon pines occur at sufficiently frequent intervals to maintain a relatively constant but not severe infection level; in others, waves of infection by the rust are highly sporadic.

In the mid-1920's our attention was called by the local Forest Supervisor to pronounced flagging of pinyon branches along the highway through the pinyon-dotted hills south of Gardnerville, Nevada. On examination the flagging was found to trace to thousands of pinyon rust infections that had become established around 1920.

Old pinyon rust cankers can still be found in those stands, but since 1925 nothing remotely approaching the infection level of the early 1920's has developed although there has been little change in the pinyon-ribes association.

This infection wave must have resulted from a season of very favorable weather conditions, first for the infection of ribes and the intensification of the rust on that highly susceptible host, and later, unusually favorable weather for pine infection.

Comandra rust: A similar instance is provided by the rust, Cronartium comandrae, on ponderosa pine in Kyle Canyon of the Charleston Mountains in southern Nevada. By the latter 1930's mortality was heavy there in young ponderosa reproduction from a wave of infection by the rust either in 1932 or 1933. Old stem cankers from that wave are still present and the pine-Comandra association was still about the same when I last had an opportunity to make a check, but there has been almost no new infection on pine since 1932.

Here again we have an example of a disease able to maintain itself between very widely and irregularly spaced seasons when climatic conditions are favorable for infection of the pine host.

Western gall rust: An example of the second class is provided by the cerebroides form of this rust in plantations of Monterey pine in the Presidio of San Francisco during the mid-1920's. This form is confined to coastal areas within the summer fog belt in California and differs from typical Peridermium harknessii in having a non-continuous aeciospore layer from which the bark covering does not exfoliate as it does in the typical form.

During the early part of the present century the unoccupied portions of Presidio lands were planted to Monterey pine and Monterey cypress. Planting was in blocks of a single species and was continued over a period of years before World War I.

Monterey pine is most susceptible to damage by the rust when it is from 2 to 8 feet high. During this stage it is not only highly susceptible to infection but main stem infections are common, resulting in later high mortality. The first plantations in the series came through this critical period without much gall rust, but the infections that did become established provided local sources of aeciospores to spread the rust to later plantations. As you may remember, this rust is an autoecious form, spreading directly from pine to pine in the aecial stage.

By the mid-twenties some of the later plantations were in bad shape, with thousands of galls, many of them on main stems. By that time, however, planting had been stopped and there were no new blocks of trees reaching the highly vulnerable stage to carry on the infection chain. The older stands closed in and the lower branches, carrying many rust galls, were either shaded out or pruned off. The survivors in the heavily infected blocks also grew past the stage in which stem infections were likely to occur. Mortality from the rust practically ceased. Today gall rust is scarce in these Presidio stands because their age and condition is no longer favorable for infection by the pathogen.

An example of an intermediate situation comes from the Little Shasta River drainage, not far from the Oregon line in northern

California. J. S. Boyce examined a Comandra rust outbreak here in 1914, and from his field notes we can get a good idea of conditions as he found them. He reported that the advance growth of ponderosa pine, from seedlings to small poles, was badly infected, with many young trees recently dead. On a 100-foot square plot, which he stated represented average rather than the worst infection conditions, his tally showed 35 percent of the trees dead or dying from the rust and another 17 percent infected but still living. Comandra was very common among the pines and badly infected (4).

Today the rust is still common in the area but primarily as trunk cankers of the older trees. The remains of pines killed by the rust are scattered through the stands. Comandra plants and young pine infections are scarce. Trees or tops will continue to die from old-established trunk cankers for many years to come, but the prospects for the resurgence of the rust seem poor. There is no clue as to what may have caused the reduction in the Comandra population unless it was from the closing in of the young ponderosa stand. However, there are still many open situations which should be favorable for the alternate host.

Lokoya canker: Turning from the rusts, there are several examples of sporadic diseases from fungi of other groups that deserve mention. The Lokoya canker, induced by the fungus Phomopsis lokoyae, is one.

When this disease first came to our attention in an outlying stand of young Douglas-fir on a ridge adjoining the Napa Valley of California in 1930, it looked rather ominous and raised the possibility that it might represent another unwanted introduction to threaten an important timber species. However, after a season's investigation J. S. Boyce concluded that the outbreak was the result of intensification of a hitherto unrecognized native organism under unusually favorable conditions brought about by late spring rains followed by an unusually prolonged fall drought. Boyce found the canker at various locations in northern California and into southern Oregon, always on rather marginal sites for Douglas-fir (3). Subsequent experiences with the disease have confirmed Boyce's estimate of it. In the Lokoya area the incidence subsided as quickly as it had risen, once the rainfall pattern had returned to near normal.

Cytospora canker of true firs: During the summer of 1930 many inquiries were received concerning the dying in crowns of young white firs around Lake Tahoe. Along with this there was a pronounced increase in dying of older trees from attacks by Scolytus bark beetles. Damage was most pronounced along the north and east sides of the Lake, but was also very noticeable along the roads between Tahoe and the town of Truckee. Some trees were killed completely, but in others the damage was concentrated in

the lower crowns. After 1930, dying from the canker continued to extend slowly in trees in which the disease was established, but there was almost no involvement of additional trees. A study of the canker by Wright indicated that the causal fungus, which he identified as Cytospora abietis, was a weak parasite, requiring a nucleus of injured tissue from which to start (9).

It seems likely that the sudden upsurge in the disease in 1930 stemmed from the dry fall of 1929. At Tahoe City, which was just on the edge of the affected area, precipitation between July 1 and December 8 was only .97 inch as against a normal of approximately 6 inches. When precipitation did start it was on cold ground and part fell as snow. Physiologically, the moisture probably had little effect until spring.

Evidences of the disease gradually abated and in recent years there has been very little of it. Wright concluded, from examinations of trees that appeared to have borne cankers for some time, that most of them started in 1924, the driest year on record during the present century in much of California. The circumstantial evidence ties in with the findings of Bier and his associates concerning Cytospora cankers in hardwoods and their relation to a low moisture content of the inner bark (2).

Septoria disease of black oak: During the summer of 1958 pronounced browning of the foliage of California black oak was noted in a number of places in the mountains of northern California (1). Examinations of affected foliage indicated that Septoria quercicola was the chief fungus concerned. From field notes of Meinecke and Boyce it was found that a similar browning of oak foliage from this fungus had been noted in parts of the Sacramento River drainage in 1913. In the districts where the incidence of the disease was observed to be pronounced in 1958 the condition of the oak foliage was entirely normal in 1959, indicating that the occurrence of weather conditions unusually favorable for infection by the fungus was probably responsible for the sudden upsurge of the disease the previous year.

Leaf blight of California-laurel: The most recent disease of a sporadic nature to be reported for California has been a leaf blighting of California-laurel described by Parmeter, Bega, and Hood in the Plant Disease Reporter for August 15, 1960 (6). They found a bacterium and two fungi to be associated with the blighting, the chief agent apparently being Colletotrichum gloeosporioides. A branch dieback associated with Botryosphaeria sp. followed.

The authors concluded that the blighting had probably occurred because of two winters of unusually heavy precipitation following one another, the first building up the supply of infective material from a very low endemic level and the second inducing the pronounced blighting.

Fomes annosus killing of pines: To the above examples may be added one that is at least semi-sporadic under our conditions: the death of pines, principally ponderosa and Jeffrey, from basal killing by the fungus Fomes annosus (8). Some dying from this fungus at centers where it has become established occurs almost every year but the mortality incidence shows pronounced fluctuations, with peaks at irregular and widely spaced intervals. Although a connection has not been demonstrated experimentally, there are indications that the peaks in mortality incidence follow periods of prolonged spring saturation in soil moisture on sites that are normally quite dry. Conversely, during the latter part of a series of years with subnormal precipitation the mortality incidence from the fungus drops to a very low level.

The examples cited above should be sufficient to indicate that forests and woodlands of California and Nevada are subject to a variety of sporadic injuries and diseases, some of them occurring at very widely spaced and irregular intervals. They also suggest that with a sufficient background of observations and experience some of them are predictable before the actual damage becomes evident.

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PANEL II. DETERIORATION OF DEAD STANDING TIMBER

James W. Kimmey, Moderator

PROBLEMS AND POSSIBILITIES IN DETERIORATION OF FIRE-KILLED TIMBER STANDS IN THE WEST

James W. Kimmey

INTRODUCTION

The merchantable volume in a standing tree does not change appreciably upon the death of the tree. If it were not for subsequent deterioration, the only volume loss to the stand would be that of potential growth caused by cessation of growth in the dead tree. Even this growth loss might be compensated for, at least in part, by accelerated growth of adjoining younger trees.

Unfortunately, deterioration starts in a tree soon after its death. This deterioration is the source of practically all volume loss attributed to mortality in merchantable stands. It is also the source of practically all of the problems to be defined and discussed in this morning's panel.

Two broad phases or degrees of deterioration concern us: (1) that which reduces the usefulness and value of wood, and (2) that which renders wood unsuitable for use. The most important aspect of deterioration is the rate of change from normal sound wood to the first stage and then to the final stage in various parts of the tree bole.

Many things influence the rate of deterioration of sound wood in a dead tree. Within a single tree species, two of the more

important factors influencing deterioration are: (1) the cause of death, and (2) the location of the dead tree.

Trees may be killed by fire, insects, disease organisms, natural suppression, or other means. A single tree or a small group of trees may be killed in a green stand; or fire or insects may kill entire stands. The rate of deterioration is affected not only by these variations but also by geographic location.

Our panel, this morning, has divided the topic of deterioration of dead trees according to these general variations I have pointed out. My category is deterioration in fire-killed stands; Dr. Ernie Wright's is insect-killed stands in the Cascades and Coastal areas; Dr. Jim Mielke's, insect-killed stands in the Rocky Mountain areas; and Dr. Ray Foster will clean the board by including deterioration in trees killed by disease.

PROBLEMS IN FIRE-KILLED STANDS

Although trees contain approximately the same sound volume immediately after being killed by fire that they contained before the fire, this sound volume soon deteriorates in value and eventually will be lost entirely unless it is salvaged while still economically practicable. The greatest and most complex problem created by fire-killed timber is salvage.

Time is the most important element in salvage, especially in extensive fire-killed stands. Salvage on the Tillamook Burn continued more than 25 years, although many trees had deteriorated to uselessness in 10 years. In the West, burns often involve stands that include many topographic and climatic variations. To enable the maximum salvage, we should know how much time is required to bring on the two important degrees of deterioration at the lower elevations, at the higher elevations, and on the southern and northern slopes even within the same burn. And we should know the difference in deterioration rates between the wet climates and the dry climates of the West. Thirty-two years after the Yacolt fire in southern Washington, most of the large old-growth fire-killed Douglas-fir trees at the lower elevations were deteriorated beyond use. In this same burn at high elevations in the Cascades, most fire-killed trees (many smaller and of the ordinarily more perishable species) still were standing full length with mostly sound boles. They contained some arrested decay in the sapwood, and some slowly developing decay near the groundline. These standing trees were barkless, bleached, case-hardened, and badly checked; but the pulpwood volume was nearly the same after 32 years as it had been before the fire.

In such large burns immediate salvage of all fire-killed timber is a physical impossibility. And, even were immediate salvage

possible, it might not be desirable because of allowable-cut quotas and log market conditions. If we were able to properly delineate the areas of fast and slow deterioration within the burn, orderly salvage could proceed without undue loss. Proper delineation could not, however, be based on any single factor such as elevation. Research is needed to determine the species of stain and decay fungi involved and their general temperature and moisture requirements. Then, microclimate on slopes of different aspects at various elevations and latitudes should be determined. It is important to know for a particular area whether the faster deterioration will occur in the low-grade logs of the tree tops or in the high-grade logs of the lower bole, or whether serious decay will occur only at the groundline. We need studies to determine the role in deterioration performed by the heart rot fungi such as Fomes laricis, Polyporus schweinitzii, and others that may occur in the trees before their death.

The time of year that the fire occurs influences the rate of early sapwood deterioration, especially at lower elevations, and the severity of the burn itself may affect the rate of subsequent deterioration. Of course all these factors must be determined for each commercial tree species, and for each species we should know the effects on rate of deterioration of such factors as tree size or age and the rate of growth in the heartwood.

Research other than pathological, or joint research with the participation of pathologists, is needed on such problems as the relation of climate to deterioration by the various cambium and wood-boring insects. And research is needed on the interrelations of these insects and the development of stain and decay fungi. We should know the effect on pulpwood yield and value of casehardened wood, wood containing deep weather checks, stained wood, and wood containing early stages of decay caused by various fungi. The effect of early degrees of deterioration on lumber grades and yields should be determined. The possibilities of other uses for partially deteriorated wood should be investigated. Logging costs and excessive breakage should be studied in relation to degree of deterioration in fire-killed stands of each commercial species on various sites. The rate and manner of breakup of fire-killed stands of various tree species is important in establishing new stands and in their subsequent management.

POSSIBILITIES FOR RESEARCH

I shall not review the literature on western deterioration studies because Dr. Phil Thomas did this in his excellent paper presented at our Seventh Conference last year. Phil pointed out that probably our greatest accumulation of dead timber in the West has resulted from fires, yet our research in this class of material probably has been the most neglected. Most research on

fire-killed timber has been done in the Coast States, chiefly on Douglas-fir. The earliest study made in the West was reported by E. R. Hodson of the U.S. Forest Service in 1907 in Forest Service circular 113. This was a general study and included several tree species in the southern Rocky Mountain region. Especially noted was the much slower deterioration in fire-killed timber above 9,000 feet elevation.

Analysis of past research attests needs for the following studies by forest pathologists; suggested priority is indicated by the number following each problem:

Studies east of the Cascade and Sierra summits - 1:

Douglas-fir - 1; ponderosa - 1; lodgepole - 1; other commercial species - 2

Rate of deterioration and determination of causal fungi - 1:

Sapwood thickness and percent of tree volume - 1
Tree size and age - 1
Rate of tree growth in heartwood - 1
Elevational effects - 1
Aspect and exposure effects - 2
Other microclimatic factors - 2
Time of year fire burned - 3
Severity of burn - 3
Interrelation of fungi and insects - 3

Activity of heart rot fungi after tree's death - 2

Studies west of the Cascade and Sierra summits - 2:

Douglas-fir - 2; other commercial species - 1

Rate of deterioration and determination of causal fungi - 1:

Sapwood thickness and percent of tree volume - 1
Tree size and age - 1
Rate of tree growth in heartwood - 1
Rainfall effects - 1
Elevational effects - 2
Aspect and exposure effects - 2
Other microclimatic factors - 2
Time of year the fire burned - 2
Severity of burn - 3
Interrelation of fungi and insects - 3

Activity of heart rot fungi after tree's death - 2

An important part of each study should be to determine the location within the tree bole of the most rapid deterioration. All research programs should be carefully planned to take advantage of the possibilities for combining objectives. Cooperative research with entomologists, mensurationists, economists, or meteorologists is highly desirable for some studies of pathological deterioration. Such joint investigation not only makes research more efficient and authoritative, but it permits the combining of more objectives.

Research on some problems created by fire-killed timber, such as those involving wood uses, excessive breakage, and stand breakup, may require only consultation with pathologists. Other research, such as logging cost studies, or some entomological studies, may be entirely independent.

SUMMARY

Deterioration of fire-killed timber in the West is an important but neglected field of research. The principal problems involve rate and degree of deterioration. The ultimate objective of needed research is to provide information that will enable forest managers to salvage fire-killed timber with a minimum of loss from progressive deterioration. This may best be done when rate and degree of deterioration can be predicted accurately for each tree species in any particular location.

Because wood staining and wood decaying fungi are the principal agents of deterioration, forest pathologists must take the lead in the required research.

SOME UNANSWERED QUESTIONS ON THE RELATIONSHIP OF BEETLE INFESTATIONS TO RATE OF DECAY

Ernest Wright

About a decade ago, the Douglas fir bark beetle showed an alarming increase in forests of the Pacific northwest. By 1950 entomologists warned that we were faced with a serious epidemic, which, by 1953, had reached its peak. During this short period, nearly two billion board feet of standing Douglas fir timber were killed. Forest pathologists than had to determine how long this beetle-killed timber could be left standing before decay would render it economically unprofitable to log.

During these studies, in which many of us took part, we learned that the sapwood was destroyed by decay within two years after infestation and by four years even the heartwood began to show advanced decay. The loss from decay was in direct proportion to the percentage of sapwood, hence young-growth timber had the greatest commercial loss of volume. The indications also were that losses from breakage were higher in beetle-infested timber than in adjacent green timber. Decay also generally advanced fastest in the mid-bole when beetle infestation was heaviest (7).

No sooner had pressure for data on rate of deterioration of beetle-killed Douglas fir subsided than an epidemic of Pseudohylesinus bark beetle developed in Pacific silver fir in the Cascade mountain area of northern Washington in particular. This epidemic seemed to be related to Armillaria mellea root rot, although to my knowledge, this has not been established definitely as yet.

The rate of deterioration of beetle-infested Pacific silver fir was found to be somewhat more rapid than for infested Douglas fir. Four years after infestation, the lumber yield from beetle-infested Pacific silver fir was reduced to 50 per cent of the volume, and to 70 per cent when the wood was used for pulp (2).

Naturally, many questions arose during these studies as to the rapid deterioration of beetle-infested timber and how best to reduce losses from decay. Some of these questions can be enumerated as follows:

1. What relationship do sapstains from Ceratocystes have to the rate of decay?

While this question may appear academic, it is one that has perplexed forest pathologists for decades. So far as I am aware, this question remains unanswered, except that we do know from the works of Nelson and Beal (4) and Wright (5) that sapstains aid in killing beetle-infested trees. From the studies made in

Sweden (1), the latest information shows that blue stain in dry building timber does not enhance the advance of decay. This is a very intriguing subject, and detailed studies applicable to forest conditions are needed.

2. How do beetle-infested trees become so readily infected with wood-decay fungi?

Here, again, is a very intriguing subject and one worthy of detailed investigation. Since bark beetles emerge long before decay is well advanced in infested trees, decay most likely enters by means of spores picked up from the outer bark of host trees just prior to infestation. This possibility appears logical, because of our knowledge of the frequency with which logging scars become infected with decay fungi(6), but to prove this is by no means easy. To identify the spores of wood rotters on washings from the beetles is extremely difficult. I have tried this many times, and also by culture, but almost inevitably, Ceratocystes and molds obscure the wood rot mycelia. I did, however, on one occasion, isolate Fomes annosus from a Dendroctonus pseudotsugae adult after emergence and just prior to attack. This certainly is meager evidence, but suggests that use of selective inhibitors in media possibly may aid in identifying wood-rotting fungi to determine whether they are on the bark of infested trees, or on the bodies of beetles as they emerge. Washing the bark and culturing these solutions present the same disadvantages as using the beetles.

3. What is the role of root rot in infestations of bark beetles?

This question has been a subject of controversy for years. Some entomologists believe that there is a direct connection, but most do not. Forest pathologists, I think, are mostly noncommital. There appears to be considerable evidence both for and against the importance of root rots to beetle infestation. My personal opinion is that during years when bark beetles become epidemic, root-rotted trees probably do serve as breeder trees much the same as down timber. During endemic years, this possibility appears to be less likely. The relationship of root rots to bark-beetle epidemics needs careful study based on field plots established and studied from year to year until a beetle epidemic occurs. Excavation of roots from a series of infested trees will be necessary.

4. What is the ecological succession of fungi in bark beetle-infested trees?

We already know that Ceratocystes or other staining fungi are almost always, if not universally, present in association with successful beetle infestation. From this point, however, we have

little information for standing timber. Munch (3), in 1907, suggested that blue-staining fungi consume the content of the parenchymal cells, and decay fungi thus are prevented from receiving nitrogenous nourishment as long as hyphae of the staining fungi remain alive. Bjorkman (1) also found that living blue-stain did inhibit wood-rotting fungi in green wood blocks to some extent. If this is true for standing timber, then it would appear to follow that decay starts first in unstained areas of the tree. This does not appear to be the situation, based on field observation. On the contrary, the reverse appears true. Here is a point in which forest pathologists could make a real contribution through detailed and carefully planned tests. Once this point is established, then perhaps the succession of wood rotters could be more readily determined, for such an important wood rotter as Fomes pinicola may not be able to advance through stained areas until the way is prepared by lesser known forms of fungal or bacterial invasion.

5. How can decay be retarded in beetle-infested trees?

If we had the answer to some of the preceding questions, we might be in a better position to answer this last one. Many avenues appear open for further exploration. For example, if the trees are felled immediately after infestation and the logs shaded by slash, could decay be retarded? Could antibiotics that would retard decay be aerially sprayed on recently infested trees? With the same idea, should recently infested trees be injected with antibiotics? Would girdling recently infected trees retard development of beetles and sapstain? Would topping recently infested trees slow down the rapid advance of wood decay? These seem to be joint problems for cooperative studies among entomologists, pathologists, and tree physiologists. Such a team might well provide the answers to these problems.

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DETERIORATION OF INSECT-KILLED STANDS IN ROCKY MOUNTAIN AREAS

J. L. Mielke

Briefly, my instructions from our chairman of this panel were to prepare a paper on problems related to deterioration of insect-killed timber stands in the Rocky Mountain areas and to suggest research to solve them. For convenience in this discussion, that portion of the Rocky Mountain range north of the Canadian-United States boundary will be referred to as the Canadian Rockies and the portion south of the boundary, the U.S. Rockies.

The Rocky Mountain area extends southward from the Yukon in Canada to Mexico, a distance of more than 2,000 miles. This extensive area includes a great variety of environmental and other conditions that add to the complexity of the deterioration problems for both pathologists and entomologists. Also, this area contains extensive stands, often inaccessible, of mature and overmature timber more or less susceptible to insect and fungus attack. Decay fungi are common in the old stands.

Numerous tree species are found throughout the Rocky Mountain area. Included among the main commercial conifers are western white, ponderosa and lodgepole pines; grand, subalpine, white, and balsam firs; Engelmann spruce, western larch, Douglas-fir, western hemlock and western redcedar. Some occur over very wide ranges but others are comparatively limited in their distribution. All these species have their insect enemies, and all are attacked by decay fungi.

Unless insect outbreaks cover extensive areas here and there throughout the range of a single tree species and under the diversity of elevation, climate, and other environmental conditions for the species, there will be no opportunity to study rate of deterioration in stands. Results of such studies would

be applicable in most Rocky Mountain areas. The likelihood of insects killing extensive stands of timber today is decreasing. Methods of controlling insects continue to improve; and forests now have many more access roads than they did 25 years ago. If roads do not exist, they can be built quite rapidly with modern machinery. Logging equipment has been improved. Consequently, in most areas, dead stands may not remain long enough for us to determine deterioration rate.

The possibilities of another extensive beetle outbreak such as occurred in Engelmann spruce in Colorado in 1942 seems rather remote. We were then engaged in World War II. At that time there were virtually no access roads into the dead stands and machinery and men largely unavailable. In other words, virtually nothing could be done to head off the outbreak. However, in 1952, only 10 years later, an outbreak of the same beetle in spruce occurred in northern Idaho and western Montana. In a short time, with expenditure of several million dollars, access roads were constructed and the outbreak checked. Most of the timber in that area has now been salvaged. Any future outbreak in most, if not all, the other tree species probably could be handled in a similar manner.

For these reasons I think it unwise to attempt to conduct studies in stands. The problem, as I see it, lies in the endemic losses within the stands.

INSECT LOSSES

The most destructive single group of insects throughout the Rocky Mountain region is bark beetles. The next most destructive group is the defoliators, including such species as spruce budworm, pine butterfly, lodgepole pine needle miner, the saw flies, and tussock moths.

The economic importance of the problem of deterioration and loss may be shown with a few figures. In the United States, according to the Forest Service, insects have been the greatest single cause of catastrophic forest mortality; they have killed more timber than fire or disease. Their outbreaks have destroyed more than 52 billion board feet of timber since 1900. About two-fifths of this destruction occurred in the U.S. Rockies. In Idaho and Montana, during the period 1911-35, the mountain pine beetle killed approximately 15 billion board feet of timber, of which only about 50 million board feet were salvaged. In Colorado the Engelmann spruce beetle killed approximately 5 billion board feet of spruce between 1940 and 1951. During that period about 29 million board feet were salvaged. Salvage is still in progress.

Loss and salvage figures for the Canadian Rockies possibly are similar. Figures were not readily available for inclusion here.

The U.S. Forest Service publication, "Timber Resources for America's Future," reported 5 billion board feet of timber were killed by insects in the United States in 1952. This volume was made up of two components: first, the yearly endemic loss that is distributed more or less evenly throughout the forest area and that comprises probably two-thirds of the total; and second, the epidemic losses, which are more or less concentrated, comprise the remaining one-third. These proportions no doubt vary somewhat from year to year, but are taken here to represent a fairly good average annual figure applicable either on a nation-wide or regional basis. The average annual endemic, or low level epidemic, beetle loss in stands of old-growth western white pine in northern Idaho and western Montana is reported to range from 1 to 5 percent of the stand. Whatever the endemic and epidemic proportions may be, however, it is generally agreed that the endemic losses in the U.S. Rockies are considerable and economically highly important.

Timber killed by attacks of endemic insects is often difficult or impossible to salvage, mainly because the killed trees are widely distributed. Following are a few examples. In 1959 on the Toiyabe National Forest, 8,000 second-growth ponderosa pines infested with the mountain pine beetle were found scattered over approximately 4,000 acres--an average of two trees per acre. Unless such infested trees are readily accessible for salvage, they become a total loss in a few years. Their stumpage value, while the trees are still sound, probably would not justify the cost of constructing an access road into such an area. Over the past 20 to 25 years in the U.S. Rockies, bark beetles have killed several billion board feet of Douglas-fir. Much of this has been endemic annual loss so scattered that often it has not been economically feasible to salvage it. In 1957, Black Hills beetle infestations covered a total of 56,910 acres of ponderosa pine type in Colorado, Wyoming, and the Black Hills of South Dakota. The estimated volume loss was 2.7 million board feet, or about 50 board feet per acre. Also in 1957, the Douglas-fir beetle killed more than 5 million board feet of Douglas-fir scattered over 32,860 acres in Colorado and Wyoming, or an average of about 150 board feet per acre.

RATE OF DETERIORATION

It is not the purpose of this paper to attempt a detailed discussion and literature review of the rate of deterioration of our important Rocky Mountain conifer species killed by insects. In fact, little has been published on this subject. Much of our knowledge of deterioration is based upon observation and the opinion of individual timber operators regarding what is salvable. For some tree species the rate is fairly rapid, while trees of a few other species may remain standing and sound for many years.

Season checks and woodborers are the factors that first cause the trees to become unmerchantable for saw logs.

The oldest paper I found on deterioration from blue stain and rot in a western conifer killed by insects was that by Von Schrenk and published in 1903. This involved ponderosa pine in the Black Hills killed by bark beetles. Within a few years after the trees died, he found the sapwood largely lost, and decay of the heartwood had started. This seems to apply generally throughout the range of ponderosa pine in the Rocky Mountain regions. Douglas-fir is reported to last somewhat longer, and some lodgepole pine may remain standing and sound for 20 years or more. Engelmann spruce in southern Utah, killed by a bark beetle invasion of the stands that started between 1916 and 1918, is still being salvaged. Present indications are that the beetle-killed Engelmann spruce in Colorado may not remain standing and sound quite as long as the Utah spruce. In southern Idaho, on the Payette National Forest, this same species of spruce killed by beetles deteriorates considerably from decay in 4 or 5 years, but the elevation of these stands is very much lower than that of the Utah and Colorado stands. I do not believe that the deterioration rate of our insect-killed stands is the basic problem.

SALVAGE OF DEAD TREES

The economic importance of dead timber in the Rocky Mountains has long been recognized. U.S. Forest Service Circular 113, entitled "Use of Dead Timber in the National Forests," was published in 1907. While the study was centered largely in Colorado and dealt mainly with fire-killed timber, insect-killed timber was also considered. Two results of the study were stressed: (1) that sound dead timber is valuable, and (2) that although widely used in some localities, it was regarded as worthless in others. Today dead timber is utilized to a much greater extent than it was 30 or 40 years ago. Prejudice at one time against certain tree species, so-called "weed trees," has greatly decreased; there are many more access roads into timber stands than formerly; and modern logging equipment greatly facilitates salvage. When necessary, access roads can now be quickly built.

There is no question that extensive and serious insect outbreaks cause heavy losses, and many problems remain to be solved. However, it seems evident that these losses will in time be greatly reduced by various means including provision of more access roads, better or improved insect control methods designed to prevent outbreaks; added mill capacity to utilize more of the dead timber; increased demands for its use in lumber, pulp, and other products. Furthermore, the epidemic outbreaks and resultant kill of timber often destroy entire stands, and thus the dead trees are largely concentrated and much more accessible to salvage than scattered individuals killed by endemic insects.

Metcalf, who accumulated information on the importance and causes of timber mortality in eastern Montana forests, expects mortality losses to decrease as utilization increases and as more intensive management becomes economically feasible. Demands for dead timber come not only from the large operator, but also from thousands of persons who each utilize relatively small quantities of it for various purposes. As an example of the latter, during Fiscal Year 1960 in Region 4 of the U.S. Forest Service, 6,400 persons obtained more than 10½ million board feet of dead timber by means of "free use" permits. Salvage is only one part of this large and complex problem.

ENDEMIC LOSSES--THE REAL PROBLEM

It was previously mentioned that the yearly endemic insect losses, which are fairly evenly distributed throughout the forest, comprise about two-thirds of the total loss. It is my belief that these losses, if not reduced, will further increase in importance; considerable evidence appears to indicate this trend. Much needs yet to be learned about the causes of these losses, and particularly the chain of events that may lead to them. One rather commonly sees in print statements about existence of interrelationships between insects and fungi, but very little on the nature of these interrelationships has been published. It probably would take a very long time to work them out. Perhaps it would be simpler if we tackled the problems of endemic losses by first learning more about their primary causes. Then we may be better equipped to reduce or prevent the losses.

I have rather commonly heard foresters remark, "There's another bug Tree!" as they pointed to a distant dead tree conspicuous by its red foliage. For pathologists it has not been that simple to determine the cause of death of a tree. Except for a tree obviously killed by lightning, for example, we usually have had to make a closeup examination, aided often by a grub hoe, shovel, and axe. Even then we have not always been sure of the cause or causes of death. Bark beetles often attack trees weakened from various causes among which are fungus attack, drought, cold, logging injury, and fire damage. Windfalls are another favorite breeding medium for many species of bark beetles. Many outbreaks, such as the Engelmann spruce beetle attack in Colorado, can be traced to this cause. It seems well established that root rots predispose trees to windthrow. I have often had a recently fallen tree pointed out to me as simply a windthrow. Close examination of many such trees revealed few undecayed roots. It took little wind, if any, to topple a tree whose root system was so generally decayed.

How important are root rots in our forests, and what is known about them? The U.S. Forest Service publication, "Timber Resources for

America's Future," mentions only one such rot, the Douglas-fir root rot Poria weirii. Considerable is known today about this pathogen, possibly more than is known about any of our other common root rot fungi, because it has been so destructive in recent years. I can think of no reason why some of our other root rot fungi may not behave similarly some day, i.e., become epidemic. We do not know yet what causes a native organism of this kind to flare up after years of quiescence.

It has long been recognized that a root rot problem exists in the Rocky Mountain area. In 1918, Hubert published a paper dealing with fungi as contributory causes to windfall of conifers in Idaho and Montana. He found root rots in a high percentage of down trees, and pointed out that it was generally recognized then that windfalls are breeding places for insects. To my knowledge, however, no intensive studies have yet been conducted in the Rocky Mountain area on this subject, particularly for the purpose of determining the importance of root rots to windfall and the extent to which the down trees contribute to beetle losses.

Etheridge, who studied decay of subalpine spruce on the Rocky Mountain Forest Reserve in Alberta, reported that the root and butt rots were responsible for nearly half of the total number of rot infections encountered. He also noted that the real importance of these basal rots lies not in the actual volume of decayed wood, but in the structural weakening they cause; it often results in appreciable windthrow of infected trees.

Nordin's paper on heart rots in relation to the management of spruce in Alberta, reported that 62 percent of the total infections recorded for boreal spruce and 48 percent for subalpine spruce were root rots.

Gratkowski studied windthrow in old-growth Douglas-fir in the Pacific Northwest and found that root rots were by far the most important factor predisposing such trees to windthrow. These findings probably can be applied to Douglas-fir in the Rockies. According to Redmond, besides causing a high volume of cull in living balsam firs, butt rots through weakening roots and butts contribute greatly to a large amount of windfall. A thorough search of the literature for similar information was not attempted. The examples cited here should suffice for our purposes. My own findings, which are based on observations made on root rots and windfall of conifers in the U.S. Rockies, are in accord with the published information. One striking example in my experience was the abundant evidence of root rot in windthrown trees present in the Engelmann spruce stands in Colorado destroyed by bark beetles during the outbreak that started in 1942.

FUNGI CAUSING ROOT AND BUTT ROTS

The available literature indicates that many species of root rot fungi are present in Rocky Mountain conifers. Also, some of these rots are caused by species of fungi that have not yet been identified. Others have not yet been studied or identified. One example of the latter is a very common and destructive root rot organism present in subalpine fir in Region 4 and probably elsewhere.

We know from observations and a limited number of studies, most of which are preliminary, that Polyporus circinatus is widespread in Engelmann spruce in the U.S. Rockies. It also attacks other conifers here, including subalpine fir, ponderosa pine, and lodgepole pine. I doubt if all of its hosts are yet known. Back about 1944 I found the root and butt rot fungus Polyporus balsameus in Engelmann spruce in Colorado and subalpine fir in Utah. Prior to that time, the pathogen was not known on either of these hosts or west of Minnesota. We know practically nothing about its distribution and prevalence here simply because it has never been studied.

Armillaria mellea and Fomes annosus are widespread in the U.S. Rockies on several different conifer hosts. In this area since 1942, I have encountered these two fungi on numerous occasions. Many more species could be added to these few rot organisms mentioned above. To list them all here is not essential to this discussion.

SOME PROBLEMS IN CUTOVER STANDS

Evidence indicates that the same or similar mortality and loss problems that occur in undisturbed or virgin stands are also present in cutover stands. For comparative purposes here, the results of Metcalf's studies are presented to illustrate conditions in undisturbed stands. In commercial forests of eastern Montana he found causes for only 45 percent of the mortality losses encountered. Nineteen percent of this loss was attributed to insects, 11 percent to wind, and 15 percent was assigned to various other causes. The remaining 55 percent of the total loss was listed as "unclassified." In other words, Metcalf was unable to determine what caused the death of more than half of the trees examined. This loss situation is very similar in cutover stands, as will be shown here.

Watt, who studied causes of mortality in second-growth western white pine, attributed 45 percent of the total board foot volume lost to wind and snow, 19 percent to insects, and 19 percent to unknown causes. In cutover stands of ponderosa pine in Arizona, Pearson found that mortality was an important factor in yield. He reported that wind and insects were responsible for a high percentage

of the losses. The causes of a significant percentage of the total loss were listed as "unclassified." I spent some time with Pearson at Fort Valley and found not only Fomes annosus but also two other species of root rot fungi present in the stands. He had not recognized any of these. On certain cutover experimental plots in Idaho, over a period of 17 years, mortality in the residual trees was so heavy as to result in a minus increment in volume production. About 85 percent of the mortality had been assigned to bark beetles. However, I found abundant evidence indicating that Fomes annosus was the primary cause of practically all this mortality and loss. I also found that bark beetles had produced broods in some of the diseased trees and that they had emerged therefrom and successfully attacked nearby healthy trees.

On the Cache National Forest in northern Utah, mortality in cutover spruce stands has been very heavy and has been caused mainly by windthrow of trees whose root systems were largely destroyed by rot fungi. Mortality in subalpine fir in the same stands, and also in adjacent uncut stands, is considerably greater than in the spruce. Root rots are very destructive in subalpine fir, and evidence of bark beetles is common. The interrelationships between the two, if any, have never been investigated.

Hubert and Ehrlich, working independently, probably have devoted more time and effort to studying root rots of conifers in the U.S. Rockies than any other researchers. Their work was centered in the western white pine type. They have made significant contributions to our knowledge of these organisms, but also pointed out that much specific information about them was still lacking.

Will present-day logging methods complicate and add to the general problem?

Selective cutting often results in much damage to roots and the lower bole through scarring of the residual trees, and it is well known that scars provide avenues of entrance for decay-producing fungi. A few studies have been made in such stands. Two examples from the Rocky Mountain area are cited here for illustrative purposes. (1) Parker and Johnson studied decay associated with logging injury to spruce and balsam fir in the Prince George Region of British Columbia. Root and butt scars were found to be very common. Fifteen years after logging, in both the spruce and fir, essentially all root and ground-contact scars were infected with decay fungi. (2) Olson investigated underground damage from logging in the western white pine type. Modern methods of logging had caused much damage to the roots of residual trees. Olson mentioned that it is not known what effect this root damage will have on these trees,

but pointed out that hardly anything worse could be done to invite infection from root diseases.

We are now leaving many cutover stands full of injured and diseased trees. In such a condition, how much of a future cut can we expect from them? I believe it possible that both our problems and losses can increase in cutover stands in the future.

CONTROL

What are some of the possibilities of reducing the losses discussed here? As I see it, salvage before deterioration of insect-killed timber is at the ultimate end of the problem. Can anything be accomplished by starting at the other end, i.e., with the root diseases? Evidently, we have much yet to learn about the many root rot fungi present in our stands. Armillaria mellea, we know, is widespread and probably is present to some degree in almost all the conifer species in the Rocky Mountain area. It is especially prevalent in the white pine type. The relative aggressiveness and virulence of this fungus have not been determined, and much other specific information regarding it is still lacking. I doubt if it is doing any good in these stands. Fomes annosus is another root rot organism with a known wide range in the West. It is becoming of increasing importance, particularly in plantations and managed stands in the United States. The two fungi live not only as parasites but also as saprophytes. Following cutting in a stand, the stumps, root systems, and perhaps other dead material provide added host material for the fungi. Little is known about Polyporus circinatus other than it has been found on several hosts and that it is a common root pathogen of Engelmann spruce in some stands. In addition to these three fungi, many other species have been reported on Rocky Mountain conifers. It would be a tremendous and very long-time task to study all of these organisms intensively and to determine the relation of root infection of each, if any, to beetle attack of infected trees. Under the great diversity of elevation, climate, and other environmental conditions to be found over the Rocky Mountain area, such a study staggers my imagination.

Years ago the word "sanitation" appeared rather commonly in forest pathological literature. For some time now, however, forest sanitation as a means of disease control and loss reduction seems largely to have been forgotten. I believe that we should again consider its possibilities. Fire was advocated as a sanitation method long ago. Recommendations were that infected and infectious cull material and slash be destroyed by burning. I have found nothing in the literature indicating that this method was proved effective or even thoroughly tested. Should we seriously consider it again? One occasionally hears remarks today regarding the possibilities of using fire as a

management tool. Not long ago I asked a well-known silviculturist if we were creating problems for curselves by putting out fires. Without hesitation he replied, "I know that we are!"

Chemicals may be a new tool that will simplify our task and provide the answers to many of our problems. The British have obtained good protection against Fomes annosus by treating stumps with coal-tar creosote immediately after felling a tree. The creosote prevents invasion by F. annosus but permits colonization of the stumps by other fungi antagonistic to it. The results of preliminary tests with antibiotics on Armillaria mellea and Fomes annosus are encouraging. These materials, therefore, probably should be thoroughly tested.

The entomologists may solve the related insect problem by using new tools in insect control. Atomic research may pave the way. Radioisotopes have been used to control the screwworm fly. Exposing the larvae of this insect to small doses of gamma rays emitted by radioisotopes resulted in adult flies that could not reproduce. The male flies resulting from treatments with gamma rays were released in natural populations. Thus sterile males competed with normal males, and since females mate but once the result was a decrease in progeny.

Continued and increased teamwork by entomologists and pathologists will be called for to reduce the tremendous losses now caused by insects and disease. To this team I would add the chemists. I believe that the methods of control suggested here should all be given a good trial. We must look to the future. If we can reduce root rot losses in managed stands, we probably will in turn reduce the insect losses. Thus, the problem of deterioration of insect-killed timber should become much less important than it is today. Healthy stands should be productive stands.

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THE DETERIORATION OF DEAD STANDING TREES KILLED BY DISEASE

R. E. Foster

My terms of reference encompass the deterioration of dead standing trees killed by pathological or physiological agents. My commitment to review previous work in these fields has been an easy one to fulfil since the literature has proven to be virtually non-existent. Apart from the contributions of Baxter and Gill (1) on Endothia-killed chestnut, of Spaulding and Bratton (2) on glaze storm damage to hardwoods in central New York, and of Thomas and Craig (3) on Douglas fir in south-central British Columbia, it appears that forest pathologists have devoted little or no attention to the classes of deterioration mentioned.

Why should this be? Has it been concluded that forest pathogens do not kill trees, or that trees do not die from summer heat, winter cold, drought, flooding, atmospheric pollution, or from sudden exposure following partial cutting? Certainly not. Any such conclusion would be inconsistent with our observations and with factual records. Why then have the two fields been virtually ignored? I suggest that several factors have been operative.

In the first place, although large numbers of trees may be killed simultaneously by these agents over extensive areas, the patterns of mortality are generally quite different from those experienced following fire or insect attack. Mortality may be restricted to individual trees randomly distributed within a forest or may be confined to groups of trees situated under particular environmental conditions. These patterns of individual tree and group killing have seldom aroused much concern on the part of the forest owner or manager, few attempts have been made to evaluate the extent of damage, and the economic basis for investigation has thus been lacking.

In the second place our intensity of forest management has not reached the stage where we can economically salvage isolated trees or small groups of trees in the forest. Studies of the deterioration of trees killed by disease are thus largely of academic interest at the present time.

In the third place it may be difficult to determine the cause of death of a tree. Trees weakened by one agent become susceptible to attack by other agents and a large and complex group of organisms may become established following death. This situation is not unique to, but appears to be more pronounced within, the classes of deterioration under review. Mortality brought about by fire or insect attack is generally recognizable as to cause for an extended period after death, even though secondary attack may follow. Symptoms of disease,

however, are often masked or confounded following secondary invation. Physiological agents, moreover, may bring about tree mortality through reason of their relative degree of deficiency or excess, and a quite different set of circumstances may be present at a later date, thus complicating diagnosis.

Finally, it is appreciated that adjacent trees may differ in their susceptibility to attack and that mortality will not necessarily occur simultaneously, even within a relatively small group of trees. This extension of mortality over a period of time complicates sampling in that special study may be required to establish the time scale for progressive deterioration.

Although the position has been taken that there may be little economic justification today for study of the deterioration of disease-killed trees, trends towards better access to and the more intensive management of certain of our forested areas suggest that studies of this nature may warrant early attention. In the interim it may be appropriate to investigate techniques and to explore problems that may expedite our future progress in this field. The following studies are suggested:

1. Definition of the specific symptoms associated with the different causes of tree mortality.

2. Additional year of death, and if possible season of death, dating techniques.

3. Study of the changes in the physical and chemical properties of wood that may result from rapid death as compared to death following a gradual decline.

4. Study of the differences that may develop in the rate or pattern of deterioration as the result of two or more agents acting together or in sequence.

5. Appraisal of the effect of season of death on the subsequent rate or pattern of deterioration.

I can hold no assurance that this list is complete. Preliminary excursions into the field will undoubtedly indicate additional problems that warrant attention.

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PANEL III. DETERIORATION OF FELLED TIMBER AND TIMBER PRODUCTS

J. W. Roff, Moderator

Fungus Succession and the Significance of Environment in the Deterioration of Logs

Keith R. Shea[1]

Growth of wood-inhabiting fungi requires suitable substratum, temperature, moisture, oxygen and light. The fungi obtain their physiological requirements such as carbohydrates, nitrogen, and minerals from the substratum. Optimum temperature lies between 25° and 30° C. for most wood-destroying fungi. In general, wood moisture contents ranging from 35 to 50 percent are most favorable for growth. Below 20 percent moisture content, wood usually is safe from attack provided it is not already invaded. Oxygen is required for respiration by which the fungi obtain their energy. Although fungi vary greatly in their sensitivity to light, it tends to retard growth and intensify the color of the mycelia.

Fungus succession in logs has not been investigated extensively. In general, the pattern of succession is as follows: Blue-staining organisms invade the logs often in conjunction with insect attacks, followed by sap rot fungi more or less restricted to the sapwood, and last the heart rot fungi. Some fungi such as Polyporus abietinus are restricted more or less to the sapwood. Others, for example Fomes pinicola, may attack both sapwood and heartwood, whereas F. officinalis commonly is confined to the heartwood.

Rates of deterioration appear to be conditioned by factors such as tree species, size of material, density of wood, and external and internal environment. Moisture content appears to be the most important individual environmental factor. It is apparent, however, that much additional research is needed to classify the significance of environment on the deterioration of logs.

1/ Abstracted from: Shea, K. R. 1960. Fungus succession and the significance of environment in the deterioration of logs. Weyerhaeuser Co., Forestry Research Note 30.

LOGGING DAMAGE AS A FACTOR IN DETERIORATION

G. W. Wallis

With sustained yield management, partial cutting is recognized as a desirable forestry practice for many of our timber species. It allows for periodic removal of net growth and mortality, and for improvement of log quality, ultimately providing a well-stocked forest. Partial cutting, however, inevitably results in injury to the residual crop. It is with these injuries that the mill manager and pathologist alike must be concerned, for as well as the direct loss in merchantable volume from the mere presence of the injuries they afford an entrance court for wood-destroying fungi.

Scarring of the trunk and roots, broken branches and tops, sunscald, weakening of residual trees as a result of adverse environmental changes, and windthrow are the most common maladies arising out of partial cutting. Numerous studies have been undertaken on coniferous and deciduous species to determine if these injuries serve as entrance points for wood decaying fungi, only a few of which need be mentioned here by way of outlining the damage which may arise.

Wagener and Davidson (19) review the topic to 1954 in their dissertation on the heart rots in living trees. In the first year following scarring, deterioration, if present, is confined mainly to the incipient stage. Deductions in lumber cut at this time arise primarily as a result of degrade because of stain rather than from a reduction in wood volume. Loss in material to be used for pulp would generally be negligible except in the ground wood process which requires a raw material free of contaminating stains. Hesterberg (10) found that defects traced to earlier logging of sugar maple resulted in a decrease of 5 percent in select and 8 percent in common grades of lumber. A continued increase in this production of low grade material, he believed, would eventually lead to a reduction in the market.

Thirty-five to 50 percent of a residual stand may be injured in poorly managed selective cuttings (Wright, Rhoads and Issac (21), Englerth and Isaac (7)). Fungi entering through scars have been responsible for the destruction of 20 - 90 percent of the volume of these stands. The variation in loss has been attributed to a number of factors. Small scars, less than 140 - 150 square inches, were less frequently infected (Wright and Isaac (20), Parker (12)) and scars in contact with the soil were more likely to be invaded by fungi than injuries further up the trunk (Parker (12), Wright and Isaac (20)). The latter authors found no consistent relationship between scar age and presence of decay. Parker, on the other hand, found that 5-year-old scars had consistently fewer fungal infections than those 15-years-old.

Decay was usually more frequent in trees with deep as compared with shallow scars. Site appeared to have little effect on the incidence of attack, trees of larger diameter, however, had a higher level of infection. Wright and Isaac (20) recorded that the level of infection of hemlock on the coast was less than that in the Interior. No consistent difference could be found by Buckland, Foster and Nordin (4) in the volume lost through decay between fast- and slow-growing trees.

Branch stubs, whether arising from falling trees or from pruning, may act as avenues of entrance for fungi. Childs and Wright (5) believed that serious damage from heart rots, following pruning in young Douglas fir, was not likely to occur. They found, however, that trees pruned in the spring were more vulnerable to fungal attack than those pruned in the fall. Sleeth (16) found fungi fruiting commonly on old pruning wounds of white pine. Broken branch stubs were observed by Boyce (3), and Thomas and Thomas (18) to be important as entrance points for heart rot in Douglas fir.

Sunscald has been shown to be more prevalent in a stand following thinning (Riley (13), Stein (17), Wright and Isaac (20)). The ability of fungi to enter a tree by way of this disorder has been recorded on a number of occasions. Broken tops, similarly, have been shown to act as effective entrance points for wood-destroying fungi. Decrease in mechanical strength because of the combined effects of scar injury and woody tissue decayed by fungi, were considered by Nordin (11) to contribute to premature breakage and windthrow resulting in losses of the volume of entire trees.

The fungi most commonly associated with damage in the Northwest varies with the host species, region, type of injury, etc. Stereum sanguinolentum, S. Chailletii, Polyporus abietinus, Fomes pinicola, F. pini, F. annosus, Polyporus schweinitzii and Armillaria mellea are among the most commonly listed as causing damage following scarring of western hemlock, spruce, balsam and Douglas fir.

The above examples indicate the volume of work which has been done on logging injuries in living trees and some of the complexities which may arise in relation to ensuing infection by decay-producing fungi. However, when we come to the topic at hand; namely, the effects of the fungi which originated in the living tree on the deterioration of material after it is on the ground we find a considerable dirth in knowledge. Workers examining scars have limited themselves to a study of organisms in the living tree, while those conducting studies of windthrown trees take up after the tree is down, seldom making more than a passing reference to fungi present prior to this time.

Roff (14) states that pieces of logging residue which contained decay prior to felling were found generally to deteriorate at a faster rate than material which was originally sound. Roff and Eades (15) recorded a more rapid deterioration of logging residue in the first three to four years in that material containing fungi prior to cutting, but after five years this tendency became masked. Several fungi that were present in the living tree were responsible for deterioration of the residue. Of these, Fomes pinicola and Poria monticola were most prominent but Stereum sanguinolentum, Polyporus abietinus, P. versicolor, Poria subacida and Armillaria mellea were also noted.

Basham (1), and Basham and Belyea (2) recorded a pronounced increase in the rate of heart rot attack after a tree was killed by insects or fire for the first four to five years. Parker (12), in a study of scar damage to spruce and balsam in the Prince George region of British Columbia, noted that Stereum sanguinolentum and S. chailletii were responsible for a major portion of the decay recorded. Engelhardt (6) in studying windthrow deterioration in the same species and general region found S. sanguinolentum of major and S. chailletii of moderate importance as deteriorating fungi. Results of the study, which commenced three years following windthrow, showed no significant increase in heart rot from the third to the tenth year.

With an increase in selective logging we can expect greater damage to residual stands than was formerly the case with unmanaged forests. Thinning can, however, only be deemed a desirable practice if the final products obtained are such that the quality and quantity exceed that which would have been manufactured had the forest been left untouched. Defects arising from injuries, could conceivably result in reducing values below an economic level, both from the progress of decay in living trees and from a more rapid advance of primary decay in down trees.

Many suggestions have been made for improving management practices so as to reduce the damage in residual crops. However, much information is still required on many aspects of the morphology and ecology of fungi shown to be capable of infecting trees weakened by logging.

Some species of fungi that are capable of entering a few tree species through logging injuries are now known. This list needs to be extended.

Knowledge on the morphology, host specificity, infection potential, rate of spread, etc. of most of these fungi is completely lacking. Time of spore production and method of dissemination in relation to infection could be a great aid to foresters in adjusting thinning schedules.

The age at which trees become susceptible, and the vigour of a host in relation to attack needs to be examined. For instance, frost cracks may have created an entrance point for fungi in western hemlock which in turn lead to heavy damage over extensive areas (Foster and Craig (8)).

Some fungi appear to be confined to the heartwood, while others develop in sapwood and heartwood, readily entering scars. Factors of the host and/or fungus leading to this phenomena are as yet unknown.

The use of chemicals as wound protectants is not receiving the attention that it should.

Little has been done to ascertain the rate of deterioration of windthrown timber which contained wood decaying fungi prior to cutting as compared with ground material.

Hepting and Chapman (9) conducted a study to determine financial losses from decay in loblolly pine. Little has been done in this regard in the Northwest.

Foresters may be in a position to recommend procedures which will reduce injuries in a stand following partial cutting but at present we can not conceive of them eliminating this malady. Pathologists, therefore, can expect to be called upon to provide more information on decay fungi entering through these wounds. If we are going to provide intelligent advice, which may affect the cutting routine as well as the amount of decayed material, then we must shortly undertake studies to gain answers to at least some of the above problems.

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INTER-RELATIONSHIPS BETWEEN FOREST PRODUCTS PATHOLOGY AND FOREST DISEASES

J. W. Roff

Generally my talk might be classified as a gripe--not aimed personally anywhere but generally everywhere at the attitudes of both the producers and users of forest products which form the whole atmosphere in which we as products pathologists live, trying to get the best use out of this complex plant material set before us by God. In products we are witness to the denouement of the results of years of forest growth. Our research may take us high in wooden towers or down among the pit props, from dusty sawmills to odorous pulp mills and on to the slime of cooling towers and into the shadows of ships' holds.

In many cases the wood material which we are asked to examine is of unknown origin and details relating to its original condition when installed are not available. Regardless of how essential such information is to the study of decay resistance, the interest of the enquirer is centered only upon its present properties, expressed usually by the question; why did it fail? Sometimes the answer is "faulty construction"--weasel words, which always involve basic factors of wood species and wood type. The type, relates particularly in our more durable species, to position in the tree and to the pathological condition of the wood. Lacking this original knowledge, research into decay resistance of timber then requires extensive field surveys and follows many devious alleys, many of them blind: starting at the tree and leading through all phases of utilization.

We cannot adopt the philosophy that wood is wood, is wood--this material is biological, subject to change in properties in response to agencies both physical and organic. The property of decay resistance is no exception. Let us therefore take a look behind the discolored panel, the stained pulp and the rotten fence post, and see why and where this condition started. Let's start with man's inhumanity to wood--one of my more sour gripes.

It is well known that both biological and chemical deterioration of wood may be reduced if delays between felling the tree and seasoning the product are avoided. We here can all appreciate that in the production of fibre or of whole wood, time is of the essence, also that for green wood, unlike whisky, the longer the storage, usually the worse the product.

In our complex logging industry, particularly in more remote areas, delays in utilization appear inevitable; both labor and highly capitalized machinery are involved and both must be kept productive--often it seems to the detriment of the product. The sequence of felling, bucking, yarding, loading and hauling is well known and each step is completed before the next begins. Once the log is out, a storage period often follows either in water or in himid dry piles at the mill until finally the log is sawn and the lumber seasoned.

Owing to the time element and also to the number of persons involved it is difficult for operators to relate the appearance of their finished lumber to details of log production. Sometimes changes are indicated but economics loom very large so that unless immediate returns can be realized from a change in an established practise it is difficult to justify--more education is required here.

One of our problems is that of brown stain in western hemlock lumber which occurs during drying. This condition is akin to that found in pine which is related to the type of log but mainly promoted as a result of delays in manufacture. While we are just starting on this study of stain in hemlock, we should come up eventually with suggestions as to its control but how much better if through better housekeeping we could avoid the stain entirely.

Aside from this particular stain we have the general line of fungal defect which may be classed as willful damage to wood. This includes the sap-stains which appear in dying trees and down logs left too long in the woods. These affect that portion which is expected to yield clear lumber--the cream of the operation. While the more obvious part of the defect is cut away, the invisible, incipient portion remains to develop later unless dried. Unless this material is to be

finished or shipped by rail it may not be kiln-dried but (except for pine lumber) often accumulates in piles after receiving a chemical preventive treatment designed to inhibit the infection which occurs after sawing. Aside from the fact that these preventives cannot penetrate nor prevent interior stain from the log, they are also intended for use over fairly short periods upon lumber drying in well ventilated piles.

Many mills, however, take the calculated risk and rely upon dips to protect lumber in bulk piles, sometimes they get by if the logs are reasonably clear but not otherwise, and if the losses are heavy the blame is usually upon the preventive--at this stage there is no apparent connection with forest conditions which are often at the root of the trouble.

In this connection, evidence of blind faith in chemistry may be found in observing the application of these closely formulated highly technical preventives. Here, complete coverage of the lumber is essential and this is assured by submerging or by drenching the boards with heavy sprays--either method entails an additional operating expense and in reducing this, certain short cuts have been evolved. In one case the lumber is passed rapidly between fog nozzles which disperse the solution very sparingly. As long as the nozzles do not become plugged and provided the boards are not bunched as they pass through, the system operates fairly well--too often however owing to lack of supervision one or more surfaces do not receive treatment.

Another practice which happily is not too common is that of treating from a watering can--one man is detailed to sprinkle each course in a load as he builds it--nothing reaches the outside of the load and the coverage varies inversely as the distance walked and as the length of time before the next smoke break--in general the treatment is haphazard.

The above types of defects are examples of what might be termed neglect in respect to utilization of felled trees in the forest or of logs at the mill. Their presence in the product does not usually cause failure but certainly degrade the appearance. Because of the many factors, human and otherwise, which contribute to the deterioration, ranging from careless handling to poor storage conditions of the product, plans for isolating the trouble are not often readily apparent. In addition it is seldom clear as to how the associated conditions may be reproduced or controlled in the laboratory.

There is a brown surface stain caused by Ascocybe grovesii which has been isolated in logs and often develops profusely upon treated lumber in bulk piles. The development appears to be associated with high moisture conditions, particularly in the film of moisture which persists upon boards in the

pile after dipping in preventives. On malt agar we find that the fungus is fairly tolerant to pentachlorophenol and is less sensitive to changes in pH than other common blue stains and moulds. On wood in the laboratory, however, we have not as yet been able to sustain this high moisture condition which is apparently necessary to produce the profuse growth seen in service and the surface development must be followed with a binocular.

I have dealt at some length with defects from the forest, fungal and otherwise which degrade the product and will now pass on to those which destroy the structure. Decay is a necessary condition in the forest which amongst other things has lead to the survival of the best in the species. In the product, however, it has lead to premature wood failure, lawsuits and substitutes such as metal frames, concrete poles and asbestos shingles.

In many cases it appears to me that when we consider the decay resistance of forest products or the pulping or other properties of forest species we are actually often concerned with the study of partially-decayed, or at least fungus-infected wood. As fungi are a part of the forest flora, it is no surprise to discover these organisms even in apparently disease-free trees. Also considering the longevity of the trees life and the variety of hazards to which it is exposed, it is to be expected that damaged trees should exist and also that they should yield infected wood at the mill. The products pathologist must then be ready to detect the decay and assess its effect in overall wood properties.

It is in our most durable species that decay resistance is most variable and this variation may be related both to physiology and also to fungal attack. In both redwood and red cedar for example, chemical extractives which occur in heartwood give the wood its well known decay resistance. In each case however, the outer heartwood in the butt contains a higher proportion of these phenolic compounds than that nearer the pith or in that further up the tree.

In general, it seems probable that the ability to produce toxic extractives increases with age of the cambial cells so that wood in the pith region is always lacking in this protection. If this is the case it may also explain the fact that fungi (usually imperfects) are nearly always found in this region although the reverse may be true. Examination of heartwood taken from part decayed cedar logs always indicates the presence of non Basidio mycetes far in advance of evident rot and it is not unlikely that these fungi prepare the way for later actual wood destruction by other organisms.

Work which we have completed with yellow cedar indicates that heartwood infected with species of Fungi Imperfecti in the laboratory is much less resistant to subsequent attack by common building rots than is clear heartwood. In this case the imperfects have not as yet been identified but their presence results in a black heartwood stain peculiar to the wood species.

One of the most obvious concomitants of fungal attack in wood is that of colour change so that one of the most striking properties of both redwood and of cedar may be associated with this defect. It is unfortunate that exposure to light soon dulls the array of colour seen in fresh sawn wood.

In western red cedar, heartwood is normally darker near the pith becoming straw coloured at the periphery. Tests indicated that straw coloured heartwood was most decay resistant provided that it originates in the peripheral region of the tree. Occasionally in fast grown trees the wood is reasonably light coloured over the entire cross section but here, wood from the inner portion is no more resistant than inner brown-coloured heartwood. In standing dead trees too, the heartwood may assume an even brown colour across the face but here again it is the position and not the colour which determines decay resistance of the wood, that nearest the periphery still being superior.

When pieces of cedar heartwood of different origin are being compared in decay resistance, material which is darkest in colour is not always the least resistant nor does the presence of decay in the log necessarily mean exceptionally low resistance although the latter is somewhat inferior in this respect to that of heartwood from sound logs. When we examined material of various colours from different logs containing advanced decay, tan coloured heartwood from logs decayed by the white rot Poria weirii was exceptionally low in decay resistance while dark black-brown wood associated with an unidentified brown rot was reasonably high in resistance.

The pattern of decay resistance in these durable woods is therefore an overlay of several factors relating to tree growth, physiological age of the cambial cells and the action of decay upon the extractives. The association of original disease in the tree with colour in the wood is most marked but owing to the variation in the occurrence of these natural preservatives, relative to radial position and height in the tree, the general effect of tree disease upon ultimate decay resistance of these woods is difficult to assess.

In a moderately decay resistant wood such as Douglas fir, we have found that the presence of incipient decay originating

in the tree, may have a marked effect upon its resistance to subsequent building rots. In one case where nearly half the floor area of a small house had collapsed after 5 years, we were able to isolate both Polyporus schweinitzii and also Fomes officinalis from a number of partially-decayed joists, along with Coniophora puteana which was the chief cause of rot.

While each of the first mentioned tree-decay organisms are known to persist in wood in service, they were not found in sound members but only in those subsequently attacked by the building rot.

In many cases the presence of incipient decay in wood does not result in a change in the appearance so that even with the best of intentions, this type of material may be included in shipments. When, as in the case of many brown rots, these develop readily in service, the problem of recognizing part decayed logs before sawing, becomes even more important.

A large number of complaints from lumber buyers also originate from "hidden rot" a condition often associated with brown pocket rot caused by Poria monticola in Sitka spruce and hemlock lumber sold in timber sizes for re-manufacture. Samples of 6 x 6's have been received which were apparently sound on all faces and at the ends---these when sawn lengthwise opened up into rot pockets up to 4 inches in depth at the center of the piece.

Unless the wood becomes dry and an abnormal number of shrinkage check develop, the defect is certainly hidden particularly to an inspector who in the case of the producer only sees green lumber.

In general, early decay caused by brown-rot producing fungi reduces the strength of wood rapidly but the white rots are less destructive. We find also that in the laboratory brown rots usually produce advanced decay in test material much sooner than do the white rots.

In discussing the significance of rot producing fungi, it has often occurred to me that little thought has been given to the length of time required for individual organisms to produce the final stage or breakdown of tissue. In the laboratory, white rots such as Fomes pini, Polyporus tomentosus and to a lesser extent, Poria weirii soon develop the visual advanced decay symptoms. Other organisms such as Fomes annosus, Stereum pini or S. sanguinolentum spread rapidly through the wood but remain in the incipient stage for periods of 6 months or more. It would be interesting to study whether or not decays in trees develop at the same relative rate.

In our work with lodgepole pine using test sticks cut from diseased trees, material infected with F. pini (incipient) was consistently lower in strength than either sound wood or that infected with either S. pini or S. sanguinolentum. In this case, however, the red to brown colorations associated with all of these organisms was sufficiently similar to make it difficult to separate in lumber, particularly when seasoned.

In pulp mills a common method of reducing costs is seen in the use of large outdoor piles for storing chips awaiting processing. Provided that the period of storage is not longer than one year and also provided that good housekeeping practices are followed and the area is kept clean, the pulp yield from this material is relatively uneffected by storage.

Studies of two piles which we made however, showed that owing to market conditions the storage period had been extended to three years and large pockets of decayed wood, surrounded by a web of mycelium, were present throughout the lower portion of the pile. While the identification of the causal organism was not verified it resembled the white rot, Polyporus versicolor. The fact that this fungus is associated with the tree and also that decay was more or less localized within the pile, suggested that the loss originated in the forest and also that rot was carried into the pile in infected chips.

Further examples of this type of relationship may be found and cited indefinitely. Enough has been said, I think, to stress the fact that with one or two possible exceptions consideration of forest disease is paramount in accessing the properties of forest products, durability, strength and suitability for the purpose intended.

Decay resistance of wood which is treated with preservatives is affected, at least at the surface and to the extent of penetration, by the presence of the treating compound and its toxicity is the controlling factor. Should the preservative become lost through leaching or other means or if untreated wood becomes exposed as a result of subsequent cutting or checking through the treated layer its effect is lost. Once again the material in question returns to its natural properties and to its association with the forest.

In products pathology our only concern is for better utilization of wood. Our first consideration is environment--how will this influence the fungal population waiting to attack the wood? Secondly, what of wood species, can we expect natural decay resistance to prevail or should further protection be provided? Combined with this aspect or sometimes even ahead of it is the question of the individual, the actual material concerned, either in research or in service--is it already infected. What is its history--first as forest material and secondly as wood or as building material.

PANEL IV. NURSERY AND TRANSPLANT DISEASE PROBLEMS

Introduction by R. V. Bega

This panel will be concerned primarily with nursery and reforestation diseases as they occur in western North America.

With the increase in reforestation demands in western North America we are beginning a phase of forestry that has for years been practiced in the East and South and, of course, in Europe. I feel very strongly that now is the time for pathologists in the West to begin a concentrated research project on regeneration diseases rather than carry on an almost extension type service-as-needed program. We should begin now to anticipate problems that could arise and begin building a basic knowledge utilizing situations and answers from other areas as well as those that are occurring now or could occur in our own area.

We have two alternatives in the approach to reforestation, natural regeneration and artificial regeneration.

Natural regeneration is, of course, not practical in the majority of our situations, especially where we are converting large brush fields or burns, large clear-cut areas or even in stand conversion. We must then at the present time concentrate on artificial regeneration where, with the exception of areas suitable to direct seeding, we must use the process of nursery production and subsequent transplant.

Our panel today is concerned with disease problems as they occur in the nursery and the effect that these diseases can and frequently do have on the storage and transplanting of seedlings. The first two papers are fairly general in that they will point up some of the problems that we now have as well as those we should anticipate, also some research approaches that we may follow. The third paper will discuss specific problems and what research approaches have been taken and others that could be taken to combat them.

FORESTRY NURSERY DISEASE PROBLEMS

R. V. Bega

This presentation, as I mentioned in the introduction, will be fairly general in an attempt to point out some of the needs in nursery disease research, followed by a discussion of the approach that we are taking in the Pacific Southwest Region.

I would like to work around five major points in discussing this artificial regeneration program:

1. Where are we now in regard to disease and disease control?
2. What do we have as a result?
3. What do we need?
4. How should we approach it?
5. What are the effects on the planting stock?

Points 1 and 2--Where are we now as regards disease and disease control, and what do we have as a result?

I believe that we can safely say that we are just out of the starting gate and disease is already leading by three lengths! As we look into the nursery program as it now is in the West, even in its as yet limited capacity, we find that contrary to the few complaints we get from nurserymen, disease is in an endemic state. The only time that we do get calls for assistance is when there is a sporadic outbreak in a given year or when in a given nursery some practice such as continuous cropping has built up the inoculum potential to the point where heavy losses are occurring. When these outbreaks occur, we then go in and try to find out, in a short time, what caused it and what stopgap measure we can use to combat it.

As a result of this type of program we have losses of seed in storage and in stratification. We have losses in the nursery due to such edaphic factors as damping-off and root and hypocotyl rot, and in some areas to aerial pathogens. We then have failures in transplanting which we find in many cases to be directly attributable to diseases picked up in the nursery. And last, but most important of all, we stand the risk of introducing new diseases into our wilderness areas, new diseases that can with time be a limiting factor in timber production in these areas. We have excellent examples of this in the past, the result of which has been extremely expensive

eradication programs. I feel very strongly that there is no need for this sort of thing to happen again.

Points 3, 4, and 5--What do we need, how should we approach it, and what is its effect on the planting stock?

In clean, vigorous plant production we cannot consider the older idea of increased seeding to compensate for last year's losses or in anticipation of current season losses. What better way is there to increase the inoculum potential in a given soil than by increasing the food supply of the organism involved. It is the responsibility of the pathologist to point out to the nurseryman the advantage that this type of culture gives to the disease producing organism.

Why should we have, or better yet, why should we tolerate losses when we already have at hand much information from other types of agriculture, information pertaining to seed selection, seed certification, soil conditioning, soil fumigation, and plant certification. Most of these techniques are easily adaptable to forestry practice. Granted we are using many of these approaches at the present time; it is on a somewhat limited scale. Soil fumigation, for example, is many times rejected because of the cost factor or because of the mycorrhizal factor. To answer the cost factor question we need only to analyze the costs involved in losses in the nursery, the subsequent losses in transport and storage, and the again, subsequent losses after transplanting. I feel certain that these costs far outweigh the cost of fumigation. As concerns the mycorrhizal factor, this is a false interpretation which I will go into later.

I am going to present a few obvious gaps in knowledge and then digress a bit from generalities and tell you what approach we are taking in California. Here, too, I might point out that I am going to talk primarily about soil-borne diseases, whether physical or biological. I am doing this with the assumption that aerial diseases will be brought out in the discussion.

In general we must first of all know what organisms are involved in each geographical area and with each crop in question. By this I mean not only the pathogenic organisms, but also, and perhaps equally important, the saprophytic and parasitic organisms. What are their relationships one to another and what factors are responsible for fluctuations in their population density?

The reasons for knowing what pathogens are involved are obvious; the reasons for studying the saprophytes and parasites are manifold. We accept the fact that we have this

group of organisms that fluctuate between saprophytism and pathogenesis. This group is particularly prevalent in the soil. We need information on their habits to take advantage of their sometimes beneficial nature and to guard against their sometimes harmful nature.

With the help of plant physiologists, biochemists, and geneticists we need to learn more about host-parasite relationships and also about host-saprophyte relations, about rhizosphere relations, and about metabolic product exchange between host and parasite or saprophyte. With the help of soil chemists and physicists we need to obtain more information on the relation of physical soil factors on host-parasite or saprophyte relationships, or simply on the growth and survival of these organisms in the soil. This is especially true as we get further into the use of soil amendments, soil chemicals (including fertilizers), and soil fumigants. We might also add continuous cropping and crop rotation to this list.

We could devote an entire conference to what we need to know; perhaps we had best stop here and present some of the approaches that we can and are taking toward solving some of these problems.

In California we are taking a "three-phase" approach to the study of regeneration diseases: (1) seed protection, (2) seedling protection, and (3) transplant protection.

1. Seed protection. Here in conjunction with our Forest Management Research Division we are studying the fungi, bacteria, and yeasts involved in seed loss. By seed loss we mean that seed which is lost in collection, in storage, and in stratification. In storage of sugar pine seed, for example, we are finding a direct correlation between organisms in and on the seed and seed viability, with different organisms playing a major role at different storage temperatures. We are now beginning a study of the use of chemicals in seed storage and in stratification media.

2. Seedling protection. In this instance, I am talking about seedling protection in the nursery. The best protection here is by eradication of certain organisms and of cutting down the inoculum potential of others.

Our greenhouse and field studies are now narrowed to the point of testing soil fumigants against different plant species and different organisms. We have several good leads already, especially with methyl bromide and chloropicrin and with combinations of these two. As I mentioned before, one of the objections to soil fumigation was the fear of eliminating beneficial organisms from the soil, especially mycorrhizal

fungi. I would like to point out here that we are not aiming at soil sterilization, but merely at selective fumigation. We can now show that with proper manipulation of the fumigant we can actually stimulate mycorrhizal development.

Our laboratory studies are concerned with isolation and identification of organisms in treated and nontreated soil and of organisms on plants grown in these soils. We are also running tests to determine the role of some 30 clones of Fusarium oxysporum and five clones of F. solani isolated from diseased and apparently healthy nursery stock. We are interested in their role as both primary invaders and secondary invaders as well as their role as normal soil flora. The prevalence of Fusarium spp. in our isolations is so great that we now consider them innocent until proved guilty. We have in addition a large backlog of other soil organisms building up in our collection of isolates that we will eventually run tests on, but our primary emphasis now is Fusarium because of its prevalence and its host specificity.

We are also studying in the field and plan laboratory and greenhouse studies on the cyclic fluctuation throughout the year of pathogenic and nonpathogenic soil organisms. To briefly mention a few, I'll list Sclerotium bataticola, Rhizoctonia solani, Pyrenochaeta terrestris, Pythium sp., Phytophthora sp., Fusarium sp., Phoma sp., and Cylindrocarpon sp.

Other field studies that we will be carrying on in conjunction with the Forest Management Research Division are such cultural practices as effects of crop rotation, cover cropping, fallowing, and soil amendments.

3. Our third phase of research is aimed at the transplant. I won't say much here as this is the subject of the paper following mine. I will point out, however, that we are pressing Federal, State, and private nurserymen to be conscious of disease and to want to grow disease-free, certified stock.

I might add in closing that along research lines we are following our nursery stock into the field on a large scale basis and taking data on mortality and the reasons for it.

Nursery Diseases and Transplant Losses

Richard S. Smith

Introduction

How great are our transplant losses in the field? They vary considerably from one species to the next. Under favorable conditions the transplant mortality in ponderosa pine is low whereas in sugar pine it is high. Survival of over 70% in a sugar pine plantation is unusual. The mortality in sugar pine transplants has been consistently so high that many field foresters feel this to be a natural condition.

What are the causes of these losses? As pathologists we should make some attempt to determine the causes. Most frequently the losses are ascribed to drought, yet in the plantations visited this last summer the soil moisture was usually found to be adequate around dead and dying plants, even as late as September. What part do diseases play in these losses? Is it a coincidence that sugar pine which suffers severely from root rots in the nursery beds also has a high rate of mortality in the field after transplanting?

The importance of nurseries as a source of pathogens which may be spread with the transplants into the field is well recognized in general agriculture. Because of this many agricultural growers have taken definite steps to remedy the problem by growing their stock under disease-free conditions. At the demands of the growers many commercial agricultural and ornamental nurseries are now producing certified disease-free transplant stock.

Are Root Pathogens Spread with Transplants

Forest nursery soils are known to harbor a variety of root pathogens, as evidenced by the disease problems existing in these nurseries. Are root pathogens carried into the field on the transplants and once in the field are they causing a portion of the present mortality? In order for a root pathogen originating in the nursery to cause mortality in the field it must first successfully complete several steps. The pathogen must become closely associated with the future transplants; it must survive the period of storage; it must be able to survive and establish itself under field conditions; and it must be capable of pathogenesis under field conditions.

Are root pathogens closely associated with the future transplants in our forest nurseries? Isolations from the roots of 1-0 and 2-0 sugar pine from one forest nursery yielded a number

of possible root pathogens including Rhizoctonia solani, Pythium sp., Sclerotium bataticola, Fusarium solani, Fusarium oxysporum, Pyrenocheata terrestris, and Phytophthora cinnamomi. These isolations were made late in the year after mortality had ceased. Furthermore the stock which was isolated from was healthy appearing and would have been transplanted into the field the next year.

Are these pathogens able to survive the conditions under which seedlings are stored between pulling and shipping? We have little information on this aspect but in general the cool conditions of storage can not be counted on to eliminate a pathogen although these conditions may temporarily arrest disease development.

Are these pathogens able to establish themselves in the field under field conditions? This is a question we must ask of each root pathogen associated with nursery stock. The introduction of a root pathogen into the field either in or on the host's roots is one of the best if not the best of methods of insuring the successful establishment of the pathogen in the field. Isolations made this year from a number of dead and dying sugar pine in two widely separated plantations revealed that most of the same pathogenic root inhabitants present in the source nursery were also present in the field on the transplant stock.

Are these nursery root pathogens capable of pathogenesis under field conditions? Have the seedlings passed the age of susceptibility or is the biological or physical environment in the field unfavorable to pathogenesis? Are there some fungi which are non-pathogenic or only slightly so while in the nursery but which may become aggressive pathogens under field conditions? Again this is an area which is largely a gap in our knowledge and is in need of further study.

The Introduction of New Pathogens into the Field

Within the last few years good nursery sites have become scarce in many areas of western North America. Because of this some new forest nurseries have been and others are being located on old agricultural land. In these old agricultural lands the forest seedlings are exposed to a variety of new root pathogens which may or may not be pathogenic to the forest species. The fact that many of these root pathogens of agricultural plants are not normally associated with forest seedlings would lead one to believe that they are not normal inhabitants of forest soils. Furthermore, many of these agricultural pahtogens are not indigenous to North America and therefore are not likely to be present in our wild lands. The possibility of introducing these new pathogens into the forest lands on nursery stock exists. The questions are: can they become established in forest soils, are they capable of pathogenesis in the field,

and what effect may they have on the future productivity of the lands concerned?

At one nursery late in 1959 Phytophthora cinnamomi was isolated from dying 2-0 sugar pine. Since then its pathogenicity on sugar pine has been confirmed. Within the last few years at these work conferences there has been considerable thought and discussion given to the threat posed by the possible introduction of this pathogen into forest areas or nursery stock.

In another area of the same nursery which is to be planted for the first time this next spring an examination of a few remaining fruit trees revealed the presence of the crown gall organism, Agrobacterium tumefaciens. Crown gall is considered mainly as a disease of Angiosperms. But in the late 1930's and early 1940's C. O. Smith by means of artificial inoculations found A. tumefaciens to be pathogenic on Sequoia sempervirens, Sequoia gigantea, Abies concolor, Libocedrus decurrens, and other western conifers. To what extent the isolate obtained from this nursery is pathogenic to conifers has yet to be determined.

In the same area Armillaria mellea was also found. But different isolates of this fungus exhibit such differences in host specificity and in virulence to a given host that pathogenicity tests must be run before any conclusions can be made.

Possible Control Measures

How may the introduction of root pathogens into the field by way of nursery stock be prevented? There are two main approaches to the problem: (1) to grow the seedlings in the absence of root pathogens, and (2) to rid the seedlings of root pathogens by means of an eradicant or therapeutant.

The first approach should involve two separate but necessary steps: (1) seed disinfection, and (2) soil sterilization. Historically seed has been disinfected either by means of a chemical treatment such as a fungicide, or by means of a physical treatment such as a hot water dip. Presently the chemical methods of seed disinfection show the most promise. In the case of forest tree seed which usually undergoes a period of stratification, the seed may be chemically treated either before, during, or after stratification. Soil sterilization also may be accomplished by chemical or physical treatments. Soil sterilization in the field is usually accomplished by chemical treatments such as the application of soil drenches or soil fumigants. The soil fumigants, methyl bromide and chloropicrin seem to be especially promising.

The second method of combating the problem, that of ridding seedling roots of associated pathogens again may be accomplished by chemical or physical treatments. Physical treatments

such as heat therapy has been frequently explored by the agricultural plant pathologist with varying degrees of success. In some conifers which suffer when the roots are immersed for long periods, as they would be in a hot water dip, there is hope that a stream air mixture being developed by Baker at UCLA may prove effective. The continuous development of new fungicides, especially with low phytotoxicity increases the possibility that chemotherapeutic root dips may well be a future solution to the problem.

Objections to Existing Control Measures

This last year during soil fumigation trials one of the most frequent questions asked was, "What will this fumigant do to the mycorrhizal fungi? Will they be killed and if so how will the seedlings react?" Samples obtained this October from fumigation plots showed that with the dosages used, that mycorrhizae were present and well developed on the roots of sugar pine seedlings grown in the fumigated soil.

Another objection which is often voiced is that the sterilized soil is an excellent medium through which fast growing pathogenic fungi may quickly spread due to the lack of competitive microbes. The advocates of soil fumigation counter with the argument that the dosages they use to eliminate the pathogen are not high enough to sterilize the soil but instead leave many microbes which can compete with invading pathogens.

An objection which is not often heard but which may become important in the future concerns the accumulation of phytotoxic principles in the soil due to repeated fumigation. There is some evidence in strawberry culture that yearly applications of methyl bromide to the soil results in corresponding yearly increases in the bromine content of the strawberry foliage. This would indicate that there is an increase in the bromine content in the soil each year due to the addition of methyl bromide. Thus the possibility of the accumulation of phytotoxic principles in the soil should be kept in mind when recommendations for periodic fumigation are made.

DAMPING-OFF - A CONCEPT, A PATHOGEN, A HOST, AND A DISEASE

H. S. Whitney

In keeping with the general theme of this conference, of asking questions rather than presenting answers, it is my intention to quickly describe damping-off and then to indicate by way of questions, a few major gaps in our knowledge of this disease.

Damping-off is the name of a common disease of germinated seeds and of young seedlings which is caused by filamentous fungi. Most plant species are affected (8,10). This disease has been known ever since plants have been grown in dense seed beds for planting out. The fungi causing damping-off are more or less non-specialized soil-inhabiting facultative parasites. Rhizoctonia, Pythium and Fusarium are the most consistently associated genera. However, other genera such as Phytophthora, Diplodia, Cylindrocladium, Pestalotia, Botrytis, Sclerotium and others have also been shown to be pathogenic (4, 8).

Three types of damping-off of coniferous seedlings have been distinguished. These are: pre-emergence, post-emergence, and late or standing damping-off. Emergence in each case refers to emergence of seedlings from soil.

Pre-emergence damping-off consists in attack, injury, and killing of germinated seeds and seedlings before they emerge from the soil. There are no observable symptoms of this disease as it occurs in nature.

Symptoms of post-emergence damping-off begin with a loss of turgor and a discoloration of the hypocotyl at the ground level. Elongation ceases and seedlings may not unhook if in the loop stage when attacked.

Hypocotyl necrosis and death of the seedling follow quickly, usually within 48 hours. The seedlings topple over and signs of the pathogen become evident. Recovery from post-emergence damping-off is not generally observed.

Late or standing damping-off is similar to post-emergence damping-off but is characterized by failure of diseased plants to topple over. This results in seedlings which are "standing dead."

Environmental conditions for optimum development of the disease may be nearly the same as those for the optimum development of the seedlings, depending on the plants and pathogens involved.

Damping-off frequently occurs and is most conspicuous in nursery beds as an epidemic but it also occurs endemically.

Sampling procedure and proof of pathogenesis probably present the principle difficulties in measuring damping-off losses in forest tree nursery beds. Because of these and other difficulties the economic significance of damping-off losses, particularly losses due to endemic damping-off, is very difficult to determine. It seems safe to say however that at no other time in the history of a forest stand can so little do so much to so many.

Present control measures for damping-off are based largely on protection from infection and on eradication of the pathogen. Control procedures differ from one nursery to the next and within the same nursery from one year to the next. This together with the fact that today's control measures are a result of extensive empirical experiments, bears witness to there being large gaps in our understanding of this disease.

The first question I would like to ask concerns the concept of types of damping-off. Are there really three types of damping-off? What are their similarities, their differences, their interrelationships? Is there any valid basis for distinguishing among these types of damping-off? Are the differences in kind or in degree?

Considering pre-emergence damping-off first: How much "no show" is due to the action of pathogenic filamentous fungi? Positive evidence for pre-emergence damping-off can be obtained under controlled conditions but the exact role of fungi in pre-emergence damping-off in the nursery is not easily ascertained. This is probably because there are many factors such as viability, temperature, moisture, insects, small mammals and birds, as well as numerous cultural practices which influence seedling emergence. Until all factors affecting germination and development of seedlings are better understood the nature and importance of pre-emergence damping-off will remain a matter of speculation.

Nevertheless laboratory experiments and field observations leave little doubt that pre-emergence damping-off does occur.

Post-emergence damping-off is characterized by attack on the hypocotyl at or just below, the ground level. The cortical tissues collapse and as the disease progresses, seedlings fall over. Attack by damping-off fungi may also begin well below the soil line on the hypocotyl or roots of emerged seedlings. Such infections, especially those below ground, are similar to pre-emergence damping-off infections except possibly in age of cells attacked.

The only real difference between pre- and post-emergence damping-off is in whether or not the seedlings have emerged from the soil at the time of attack.

Late damping-off results from attack by filamentous fungi on older seedlings which have developed a vascular system sufficiently strong to support them after loss of turgor which occurs during pathogenesis. Injury may be primarily to the roots or to the stem at or below the ground level.

Although some seedlings may develop a supporting vascular cylinder and still be in perfect health, we must consider that other seedlings would not. Older seedlings may be predisposed to disease by one or more physical and or biological factors and the resulting disease is called late damping-off. From observations of disease development in forest nursery beds and from reports of studies of factors affecting disease development in seedlings of other crops, it seems reasonable to suggest that fungus infection of older seedlings in an earlier stage of development could act as a predisposing factor. That is to say that pre-and post-emergence damping-off could predispose seedlings to late damping-off. In this sense late damping-off is thought of as a continuation of post-emergence damping-off. Further, it is hypothesized that the stage of development of the host at the time of infection and the rate and extent of disease development are prime factors in determining whether pre-, post- or late damping-off results; and also that all three types are different manifestations of the same disease.

Some questions that might be asked at this point are: How many seedlings in a first year planting have never been attacked by damping-off fungi? How can one distinguish among root or hypocotyl rot and late or incipient post-emergence damping-off? What are some factors determining the fate of attacked seedlings? It is suggested that to classify or divide soil-borne diseases of seedlings into narrow types represents a gap, perhaps an ever widening gap, in our thinking about such diseases.

I would like next to mention some special problems referring principally to damping-off of coniferous seedlings caused by Rhizoctonia.

Consider first the fungus. One of the most frequently asked and least frequently answered questions concerning Rhizoctonia is, how does this fungus survive in soil in the absence of its host? There is evidence for its survival as a saprophyte in soil (2). But there is also evidence that it is a poor competitor under certain conditions with other soil saprophytes (13). There are reports of fungus parasites and antagonists to Rhizoctonia (3). Rhizoctonia may form sclerotia which can survive in soil for several years (9, 14). This fungus has been reported as a non-pathogenic component of the rhizosphere

of certain plants (17). Increased microbial activity in soil has been reported to suppress the pathogenic activity and saprophytic colonizing ability of Rhizoctonia (5). No comprehensive synthesis has been made however of all the relevant information and the exact mode of saprophytic survival and behavior of Rhizoctonia in soil is still anyone's guess.

One major difficulty in studying the behavior of this fungus in soil is that it is difficult to isolate. As is well known, Rhizoctonia does not show up in ordinary soil dilution plates. It can and has been isolated by plating out mycelial fragments, sclerotia, bits of decaying organic matter, and by using specific organic baits. These methods are tedious and highly selective, and a simple method of measuring the total amount of Rhizoctonia in soil is still lacking.

Some gaps in our knowledge of damping-off resulting from a lack of information on the survival and behavior of this fungus in soil are:

What is the inoculum potential in a given seed bed?

What is the infective unit of Rhizoctonia in soil?

What is the half-life and generation time for a given population of Rhizoctonia in soil?

At what stage in the life cycle of this organism can we employ control measures to best advantage?

What are the possibilities of biological control?

Another important phenomenon relating to the pathogen that is important in so far as disease is concerned is that of genetic variability. It seems that Rhizoctonia has an almost unlimited capacity for variation. This is achieved primarily by segregation and recombination of genes in the basidium, by anastomosis and nuclear migration within clones and by natural mutation. We should have a thorough understanding of the formation, occurrence and distribution of the perfect stage in nature. We also need to know more about mutations and mutation rates in this fungus. The extensive capacity for variation in Rhizoctonia suggests that we should expect changes in the fungus and in diseases which it causes.

I will now consider briefly the host plant. It has been shown that for seedlings in general, including conifers, that the faster the host develops the less the disease develops (6, 11, 12, 15, 16). It is commonly observed that coniferous seedlings become progressively more resistant to damping-off as they age. Seedlings a month or more emerged are not usually susceptible.

From such observations it might be concluded that seedlings should be aged as rapidly as possible, obviously however, something other than time itself must be involved.

It has been shown that lignification in cell walls is important in arresting the development of Pythium damping-off in cucumber seedlings (12). Also the development of cuticle as it affects permeability and penetration has been cited as important in determining susceptibility of radish seedlings attacked by Rhizoctonia (7). Similar changes in composition and or structure in cell walls of coniferous seedlings may be important in the resistance of coniferous seedlings to damping-off. If three day old seedlings could be induced to have cell walls like those of thirty day old seedlings perhaps damping-off losses would not be as severe.

Some questions I would like to raise which concern the host and pathogen together are: what is the first step in pathogenesis? Where does it occur on the host, and why? What are some factors governing the meeting of the pathogen and host?

When Rhizoctonia attacks seedlings of several host plants, including white spruce, the fungus characteristically forms infection cushions. Infection cushions are formed by multiple branching of hyphal tips. Evidence has been obtained that for radish seedlings, there is a chemical substance which diffuses from the hypocotyl and stimulates the development of these infection cushions (7). It has been hypothesized that this stimulus can not escape from the seedling after the cuticle is well developed.

Rhizoctonia attacking white spruce seedlings on water agar has been observed to contact and grow beyond the seedlings for 15 to 20 mm. with no development of infection cushions at first. The first signs of infection cushions are seen after two to three days. This development is observed to be localized on the hypocotyl although other regions of the seedling may have also been overgrown by the fungus. Infection cushion development then spreads towards the shoot and root apices, and the seedlings are usually dead within 36 hours. Why is there an initial delay in the infection process? Why does the pathogen develop beyond the seedling at first? Why is the initial infection restricted to the hypocotyl? How can these observations be interpreted in terms of diffusion of a stimulus and cuticularization?

Further research into the details of these and other host pathogen relationships of a physiological nature should be carried out using homogenetic seedlings and a homokaryotic pathogen, studied separately and together in controlled physical and biological conditions.

Finally a word or two on control of damping-off. Present control, which is based largely on prevention and eradication, is achieved by a combination of four different semi-successful methods; soil pasteurization, seed treatment, post-emergence treatment (sprays and dusts) and improved cultural practices. It seems that there is no universal control recommendation which will be effective in all nurseries at all times, and best results have been obtained by adapting these four approaches to local conditions. It is again emphasized that the unpredictable and largely unexplicable results obtained in efforts to control damping-off illustrate that we have a great deal to learn about diseases in nursery beds.

Future programs for the control of damping-off should be an integral part of a general program of producing pathogen-free shipping stock.

I would like to conclude by drawing to your attention a statement made by Dr. Kenneth F. Baker (1). This statement concerns diseases in seed beds of horticultural and vegetable crops, but I believe it is equally applicable to diseases in forest tree nursery beds.

"Many explanations are offerred for the failure of a nursery crop, and these often confuse rather than clarify. Thus it is said that flats of seedlings have been watered too much or too little, the seedlings were planted too deep or too shallow, the weather was too hot, the plants were too soft or too hard, or came from a poor lot of seed, or were grown in the wrong soil mix. In most cases investigation has revealed that pathogens had caused the disease and that the condition blamed had merely aggravated the trouble."

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15. _____. 1943. Influence of temperature, moisture, and soil reaction on the damping-off of red pine seedlings by Pythium and Rhizoctonia. J. Agr. Res. 67: 273-293.

16. _____. 1943. Seasonal development in the nursery of damping-off of red pine seedlings caused by Pythium and Rhizoctonia. J. Agr. Res. 67: 417-432.

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ACTIVE PROJECTS - NEW

(Project leaders' affiliations and addresses are given in the Membership List, Appendix VII.)

C. Cone, Seed, and Seedling Diseases

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

Objective: To determine the pathogens responsible for damping-off and root-rot losses in natural redwood reproduction and to investigate disease development.

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

Objective: To develop techniques for the selective isolation of certain fungi from soil or roots to improve methods for studying fungus populations in soil and roots.

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

Objective: To investigate the physiological interrelationships of host and pathogen in the development of damping-off.

F. Stem Diseases - Malformations, Witches' Brooms, Dwarf-mistletoes, etc.

60-F-1 Studies of dwarf mistletoe in British Columbia (R. E. Foster)

Objectives: To determine the chromosome characteristics of North American members of the genus Arceuthobium, to examine host range, to determine naturally occurring biological agents, and to develop quantitative and qualitative measures of dwarf mistletoe infection and damage.

Progress: This project was initiated in September 1960 and objectives and methodology are currently under development. In general, however, the initial approach will be two-fold: (1) to attempt to gain reliable criteria of separation of the

different species and formae, and (2) to attempt to define recognizable categories of dwarf mistletoe infection that can be correlated with measureable amounts of damage.

60-F-2 Translocation relationships of natural substances and toxicants between conifers and dwarf mistletoes (O. A. Leonard, R. J. Hull, and C. G. Greenham)

Objectives: (1) To study the translocation of natural materials between conifers and mistletoes and the tissues involved in transport. (2) To study the translocation of toxicants between conifers and mistletoes. (This is a NSF project)

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).

Objectives: (1) To demonstrate the amount of losses resulting from growth reduction in different age classes, under a variety of site conditions and intensities of infection; and (2) to demonstrate the extent of mortality that results in different age classes, under a variety of site conditions and intensities of infection.

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

Objective: To determine quantitatively the effect of dwarfmistletoe on productivity of ponderosa pine stands.

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

Objectives: To determine what proportion of the seeds produced alight on the trees, the redistribution of seeds following landing, the proportion that germinate and cause infection, and factors affecting survival of young infection.

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

Objectives: To determine the effects of dwarfmistletoe on volume, diameter, and height growth and basal area in lodgepole pine stands under 150 years old.

G. Stem Diseases - Stains and Decays

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).

Objectives: (1) To determine the extent of decay in stands of spruce, balsam and lodgepole pine in the Wet Belt of the Kamloops Forest District; (2) To determine the external indicators of decay, and to assess their value as aids to cruising; (3) To identify the fungi of major importance.

Results to date: Seven hundred spruce and balsam were analysed in detail in a mature forest in the Hendrix Creek area. Heart rots caused chiefly by Echinodontium tinctorium, Fomes pini, and evidently Stereum sanguinolentum, were found to be of major significance. Analysis of the data gathered, and identification of fungi is in progress.

H. Stem Diseases - Rusts and Cankers

60-H-1 Studies of forest tree rusts in Alberta (J. E. Nighswander).

Objectives: (1) Long term: To study aspects of the genetics of the more important forest tree rusts of Alberta, with the final aim of elucidating some of the major factors influencing their epidemiology. (2) Short term: To make a comparative study of the nuclear phenomena of peridarmium harknessii and P. stalactiforme.

60-H-2 Induction of resistance to Cronartium ribicola in Pinus monticola seedlings by seed treatment with chemical mutagens (James W. Hanover).

Objectives: To determine the effects of ethylene methane sulfonate, a chemical mutagen, on western white pine seed. In particular, to study its effects on the disease resistance factors in white pine.

60-H-3 Comparative biochemistry of resistant and non-resistant Pinus monticola to infection by Cronartium ribicola (James W. Hanover).

Objectives: It is highly desirable that a rapid means be found for detecting resistance to blister

rust in very young pine seedlings. To determine the basis for resistance to a disease which seems to be physiological in nature, two types of information are required. First, the biochemistry of the host must be completely understood, and second, the nutritional requirements of the parasite must be elaborated. This project will provide the first body of information by studying the biochemistry of needle and bark tissues of resistant and non-resistant western white pines.

J. Defects and Decays of Forest Products, Dead Timber, Slash, Etc.

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

Objectives: To study the brown mold Ascocybe grovesii in relation to its growth requirements and reaction to preservatives using both malt agar and wood media.

General remarks: Chemical compounds for use as preservatives on wood products against fungal deterioration are applied generally throughout the trade. While these are largely toxic to a variety of organisms, situations arise under which specific fungi appear tolerant to applications normally considered to be inhibitory. Ascocybe grovesii which occurs in different tree species is also commonly reported on green lumber treated with commercial sap stain and mold preventatives containing sodium pentachcorophenate.

K. Miscellaneous Studies

60-K-1 Tests of the efficacy of systemic fungicides on four native rusts and one needle cast fungus (James L. Mielke)

Objectives: To test various antibiotics such as phytoactin, and Acti-dione and its derivatives to determine their usefulness in direct control of: (1) Peridermium fillamentosum; (2) Cronartium comandrae; (3) C. stalactiforme on ponderosa and lodgepole pines; (4) Melampsorella caryophylacearum on subalpine fir; and (5) Elytroderma deformans on ponderosa pine.

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

Objectives: To examine the translocation of systemic fungicides in Douglas fir, to conduct field tests of control of Rhabdocline needle cast and to undertake bio-assays of fungi responsible for disease in Douglas fir.

General remarks: This project is being undertaken by the Forest Biology Laboratory, Victoria, in cooperation with Iowa State College. It constitutes the initial phase of a more comprehensive project by the Forest Biology Laboratory to review the experience in forest disease control and to undertake and coordinate studies in the silvicultural, chemical, and biological control of British Columbia tree diseases.

Progress: Field trials were undertaken on 360 trees in a Rhabdocline area in the Interior region of B. C. Basal stem and foliar applications of nine systemic fungicides or derivatives are being tested in concentrations ranging from 100-800 p.p.m. Samples are being obtained at periodic intervals to determine translocation and persistence.

ACTIVE PROJECTS - CHANGES

F. Stem Diseases - Malformations, Witches' Brooms, Dwarfmistletoes, Etc.

55-F-2 Chemical control of dwarfmistletoe of conifers in California. Change of project leader from R. V. Bega to C. R. Quick.

APPENDIX II

TERMINATED PROJECTS

58-B-1 Drought damage to western white pine and associated species (C. D. Leaphart).

An unusually dry summer in 1958, characterized by abnormally high temperatures, damaged western white pine, western larch, grand fir, and lodgepole pine in a portion of northeastern Washington and northern Idaho. Damage ranged from foliage wilting to death of trees. Affected trees were from 3 to 25 feet high.

55-D-1 Root stain disease of eastern white pine (C. D. Leaphart).

The dying of eastern white pine in two plantations in northwestern Montana is caused by a root stain fungus, Verticicladiella sp. Death results from girdling action of the fungus at or near the root collar. It appears that this disease, which radiates out from a common center, may cause complete destruction of the two plantations. No native tree species growing in the diseased centers have been killed.

53-H-3 White pine blister rust (W. A. Porter)

A final report on this project is listed in Appendix IV of the current Proceedings. The original objectives of this project have been concluded and terminated; further activities, designed to evaluate selected trees, have been incorporated within the terms of reference of 57-A-3, Exotic Plantation Studies. Existing plantations of selected material will be maintained by the Provincial Forest Service and their condition reported on from time to time by the Forest Service and the Forest Biology Laboratory.

53-J-2 Deterioration of wind-damaged spruce and balsam forests (N. T. Engelhardt)

A preliminary report on this project was listed as No. 10 in Appendix IV of the Proceedings of the Sixth Conference. A final report has been accepted for publication and will appear as No. XXIII in the series Studies in Forest Pathology, Cdn. Dept. Agric.

53-G-2 Decay of alpine fir in the Prince George Forest District (N. T. Engelhardt)

The death of the project leader has necessitated that this project be terminated without formal reporting.

56-I-1 Dieback of sapling Douglas fir in coastal British Columbia (W. A. Porter).

The final report on this project was listed as No. 46 in Appendix IV of the Proceedings of the Seventh Conference.

57-G-8 Scar-damaged spruce-balsam forests (A. K. Parker)

A preliminary report on this project was listed as No. 43 in Appendix IV of the Proceedings of the Sixth

Conference. A final report is listed in Appendix IV of the current Proceedings.

APPENDIX III

NEW OR MODIFIED TECHNIQUES

1. A Method of Preparing Dried Cultures for the Herbarium (R. J. Bourchier)

This method has been used for several years by Dr. S. J. Hughes of the Mycology Unit of the Plant Research Institute, Canada Department of Agriculture, Ottawa. It has been very useful at the Calgary Laboratory and may be so to other members of the Conference.

The procedure is as follows:

Prepare a drying bed or surface by stretching "Parafilm" over a sheet of glass or masonite; this can be any size to accommodate one or more cultures at one time. Place a few drops of Mayer's adhesive on the "Parafilm" and spread it till it is slightly larger than the area of a Petri plate. Cut the agar around the colony with a scalpel in such a way as to produce a disk of agar with edges bevelled out and down. The diameter of the lower surface of the disk will thus be greater than the diameter of the upper surface. Place this disk bearing the fungous colony on the adhesive and allow to dry for several days. After drying, cut the "Parafilm" around the agar with a scalpel and peel away from the glass or masonite. This produces a flexible herbarium specimen ready for filing. Paper labels can be placed between agar and "Parafilm" prior to drying to provide a permanent means of identifying the specimen.

2. Large-scale seedbed inoculation with Cronartium ribicola (R. T. Bingham)

Up to 3 adjoining, 4-feet-wide nursery beds 100' long can be inoculated under a single 32' x 100', 6-mil-thick, polyethylene tarp (cost $60.00, reusable), outdoors if shaded with canvas. Once 95-100% relative humidity is obtained inside this shaded inoculation chamber, evaporation is practically nil. In one 30' x 20' pilot trial chamber no additional watering or mist nozzle operation was required for one week thereafter.

APPENDIX IV

PUBLICATIONS

This list includes publications issued since the 1959 meeting or those overlooked in the last Proceedings. In addition, 11 older publications dealing with Inland Empire work on the resistance of western white pine to blister rust are listed. These were not included previously in Work Conference Proceedings Publication Lists.

1. Andrews, S. R. and J. P. Daniels. 1960. A survey of dwarfmistletoes in Arizona and New Mexico. Rocky Mtn. For. and Range Expt. Sta. Station Paper 49, 17 pp.

2. _____ and W. E. Eslyn. 1960. Sooty-bark canker of aspen in New Mexico. Plant Disease Reporter 44: 373.

3. Anonymous. 1960. Northern Idaho Forest Genetics Center. U.S. Forest Serv., Intermountain Forest and Range Expt. Sta. Brochure, 4 pp.

4. Bega, Robert V. 1960. The effect of environment on germination of sporidia in Cronartium ribicola. Phytopathology 50(1): 61-69.

5. _____ and Richard S. Smith. 1960. Diseases threaten forest nurseries. Pac. S. W. For. and Range Expt. Sta. Misc. Paper No. 52.

6. _____ and Richard S. Smith. 1960. Thermal death range of sclerotia of Macrophomina phaseoli. Phytopathology 50(9): 628.

7. Bingham, R. T. 1959. The intra-species approach in breeding for disease resistance in trees. Abstract in Proc. IX Internatl. Bot. Cong. Vol. 2, pages 32-33.

8. _____. (Editor) 1960. An annotated directory to Canadian and foreign workers in forest genetics and related fields. Jour. For. 58(8): 602-618.

9. _____ and A. E. Squillace. 1955. Self-compatibility and effects of self-fertility in western white pine. For. Sci. 1(2): 121-129.

10. Bingham, R. T. and A. E. Squillace. 1957. Phenology and other features of the flowering of pines, with special reference to Pinus monticola Dougl. Intermountain Forest and Range Expt. Sta. Research Paper 53: 26 pp.

11. _____, A. E. Squillace, and J. W. Duffield. 1953. Breeding blister-rust-resistant western white pine. Jour. For. 51(3): 163-168.

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APPENDIX V

MINUTES OF THE BUSINESS MEETING

The Business Meeting was called to order by Chairman Parker on December 2 at 1:00 p.m., with approximately 30 members in attendance.

The first item of business was the minutes of the previous meeting. It was motioned by G. W. Wallis that the minutes as printed in the 1959 Proceedings be approved. This was seconded and unanimously carried.

The Secretary-Treasurer then submitted the following report:

	Credits	Debits
Balance from 1959 conference	$ 81.84	$ ---
Interest (1960)	1.74	---
Banquet	264.00	281.72
Mrs. Buckland's trip to Centralia	---	20.99
Deficit in Buckland Memorial Fund	---	8.81
Totals	$347.58	$311.52
Balance on hand	36.06	

Motioned by K. R. Shea, seconded by R. V. Bega that the financial statement be approved. Carried.

In Memorium

Memoriums to three members of the Work Conference who passed away since the last meeting were expressed for Dr. C. W. Waters and Prof. A. W. Slipp by C. Gardener Shaw, and for Dr. H. N. Hansen by Robert Bega. Chairman Parker then called for one minute of silence in honor of these deceased members.

Interim Program Chairman's Report

Chairman Parker called on R. G. McMinn, Interim Program Chairman, who presented his report. The following points were raised as possible guidelines in future programming:

1. The well filled discussion periods at the Centralia meeting should reassure program chairmen that a 50-50 balance between formal presentation and discussion is feasible now that an atmosphere of informality prevails within the group.

However, panel chairmen should seriously consider their responsibility in maintaining discussion and keeping it appropriate to the subject.

2. Where possible, consideration be given to prior consultation among panel members so that an integrated coverage of the topic may be presented. It would probably help the panel chairman lead a better discussion if he had also read the papers to be presented before the meeting.

3. When possible, consideration might be given to circulating the text of papers one or two weeks ahead of the meeting so that at the time of the meeting a topic could consist of the presentation of visual material and discussion. In this way a maximum amount of time could be spent in discussion with members already primed with questions.

4. When possible, consideration be given to an arena-style layout of the meeting room so that the maximum number of people may face each other or at least not just stare at the back of others' heads. If possible, squeaky chairs should be dispensed with.

The following topics were suggested for future programs, presented approximately in order of interest expressed:

1. Root and rootlet diseases; methods of investigation; significance of rootlet mortality.

2. Dwarfmistletoes.

3. Fundamental basis of plant science - anatomy and physiology - as they pertain to parasitism and disease; development of diagnostic criteria for assessing health and vigor.

4. Diseases of unknown origin - declines (pole blight, X disease of Pinus ponderosa); abiotic diseases.

5. Control methods and possibilities - chemical, biological, mechanical (including fire).

6. Climatic factor in disease causality.

7. Fungus insect successions and relations.

8. Virus diseases: this topic should only be discussed in a suitable location where agricultural virologists or others are available to provide the bulk of factual information.

9. Project reviews - updating and discussion.

10. Disease and growth impact

11. Twig and branch diebacks

12. Armillaria

13. Antagonism

There was general agreement that the business meeting should be held earlier in the conference and not scheduled as the last item on the agenda, as has been our custom.

Chairman Parker expressed appreciation on behalf of the conference, to Bob McMinn for a very useful report.

Dwarfmistletoe Committee Report

K. R. Shea, as Chairman of the Dwarfmistletoe Research Committee, discussed the report (Appendix VI) handed out during registration for the conference. It was agreed that the annual reports should be continued, and that they should be handed out at the start of the conference. K. R. Shea volunteered to continue as chairman of this committee.

J. L. Mielke suggested that the summary of dwarfmistletoe chemical control tests prepared for the 1955 Proceedings should be brought up to date.

Special reports on dwarfmistletoe research were given by Bob Scharpf on germination and infection for several California species; J. R. Parmeter on infection tests with the mistletoes on red and white firs; and by John Rediske and O. A. Leonard on their work on translocation between dwarfmistletoe and its host.

Meeting Sum-up

Chairman Parker then called on C. P. Wessela, who presented an excellent sum-up of the topics and ideas expressed at the conference.

New Business

Motioned by G. W. Wallis and seconded by C. G. Shaw, that Mrs. Buckland's trip to Centralia be financed out of the conference treasury. Carried.

Moved by R. E. Foster and seconded by J. W. Kimmey, that the statement of policy concerning conference organization and activities printed in the Proceedings of the Fifth Conference, pages 126-129 inclusive, be amended to incorporate a new

section (Section IX) to read as follows:

IX. Policy Statement Revisions.

Subsequent amendments to this Statement will require a not less than two-thirds affirmative vote, as determined by ballots cast by eligible members, such vote to be determined through letter ballot distributed by the Chairman or officer designated by him. Eligible members will be determined as the number of persons, excluding guests, whose names appear in the Membership List of the Proceedings issued immediately prior to the date of issue of the letter ballot.

Carried unanimously.

Moved by R. E. Foster, seconded by P. C. Lightle, that the statement of policy printed in the Proceedings of the Fifth Conference, and as subsequently amended, be further amended by the following nine motions:

1. That a classification of "honorary member" be established and that this designation be assigned at the pleasure of the conference to members who, following long and valued contributions to the field of forest pathology and to the proceedings of the work conference, retire from active continuous employment in forest pathology. Carried.

2. That attendance be restricted to honorary members, members, and invited guests. Carried.

3. That invitation of guests be left to the individual members, but that members inviting guests should use considerable discretion, keeping in mind the objectives of the conference. Members should inform Council whenever possible that they wish to invite a guest so that a formal invitation may be extended, and so that Council will have an indication of the size of the attendance to be expected. Carried. (This was a substitute proposal motioned by G. W. Wallis and seconded by R. V. Bega in lieu of a motion submitted and subsequently withdrawn by R. E. Foster.)

4. That invitations to guests be specific in regard to their duration. Carried.

5. That the presence of guests at business or other meetings be at the pleasure of Council. Carried.

6. That the regional boundaries of the Western Region be defined as that region in North America lying west of the

one-hundredth Meridian south of the 49° parallel, and west of the Alberta-Saskatchewan boundary north of this parallel. Carried.

7. That membership be restricted to those persons who, in the opinion of Council,
   (1) are resident within the Region and qualified for membership,
   (2) may not reside within the Region but who are directly concerned with the administration of activities in the Region which fall within the scope of the Work Conference, or
   (3) may not reside within the Region but who carry on activities which fall within the scope of the Work Conference and who are and have been active participants in the Western Work Conference as determined through regular attendance and contribution.

   Carried.

8. That Council revise the membership list annually, adding or deleting names as required, to ensure that it consists only of active, qualified members. Carried.

9. That unless otherwise approved by Council, the distribution of the Proceedings be restricted to members and honorary members. Carried.

Since such motions involve changes in policy concerning conference organization and activities as printed in the Proceedings of the Fifth Conference, approval of at least two-thirds of the members by letter ballot is necessary for their enactment (second item under New Business in these minutes). However, it is the opinion of Council that many of these proposals should be considered further, and at our next annual meeting before they are voted upon. Several of these motions met with lively discussion from the group, and it seemed apparent that many members were not sufficiently aware of the concepts of the motions, or of the interpretations that are likely to be made if they are passed.

The Council will, therefore, submit a mail ballot to determine whether the membership agrees to postponement of action on the listed motions until the next meeting. R. E. Foster has agreed to head up a committee to look into this area further and to present a report at our 1961 meeting.

Motioned by R. V. Bega, seconded by K. R. Shea, that a notation regarding each member's fields of special interest be added after their name in the Proceedings. Carried.

It was then motioned by F. G. Hawksworth, seconded by C. G. Shaw, that the conference elect a historian, who would maintain

a file of correspondence relating to the Work Conference, and also a file of Proceedings. Carried.

Since such an appointment involves a change in policy concerning conference organization, approval of at least two-thirds of the members by letter ballot is necessary for its enactment. This ballot will be sent out in time so that the results will be available for the 1961 meeting.

Ninth Conference

Chairman Parker read a letter from G. P. Thomas inviting the conference to hold its next meeting in Banff, Alberta or vicinity, not later than October 15, 1961.

Motioned by G. W. Wallis, seconded by P. C. Lightle that the 1961 meeting be held in the fall at Banff. Carried unanimously.

R. E. Foster suggested that the conference consider a meeting in Mexico. The Council plans to correspond with our Mexican colleagues in hopes that it may have some definite proposals in this regard to present at our 1961 meeting.

Election of Officers

Chairman. Motioned by R. E. Foster, seconded by K. R. Shea, that F. G. Hawksworth be nominated as Chairman of the 1961 Work Conference. A. C. Molnar motioned that nominations be closed and F. G. Hawksworth was elected by acclamation.

Secretary-Treasurer. Motioned by G. W. Wallis, seconded by D. R. Miller, that J. R. Parmeter be nominated as Secretary-Treasurer for the 1961 Work Conference. R. V. Bega motioned that nominations be closed, and J. R. Parmeter was elected by acclamation.

Motioned by R. E. Foster, seconded by P. C. Lightle, that the Conference extend a vote of thanks to the Weyerhaeuser Company for the many courtesies extended during our meeting that contributed greatly to the success of the Conference. Carried unanimously.

The business session was adjourned at 4:00 p.m.

APPENDIX VI

COMMITTEE REPORT ON STATUS AND NEEDS OF RESEARCH ON DWARFMISTLETOE

J. E. Bier, F. G. Hawksworth, J. R. Parmeter, and K. R. Shea, (Chairman).

Highlights of 1960 Research

1. Intensification and Spread
   a. A biological evaluation of dwarfmistletoe in the Whitetail Control Unit of the Mescalero-Apache Reservation was completed by the Rocky Mountain Station in 1960. Results of the evaluation are being used by the Bureau of Indian Affairs in planning the first re-cleaning operation of the unit, which is scheduled to start early in 1961. Initial direct control measures were applied between 1953 and 1955.

   The evaluation was made according to the following procedures. The net control area was subdivided according to treatment years, and a dot overlay (16 dots per section) was superimposed on a large map of the control unit. Enough dots were then selected at random for 1/10-acre plot centers to give a 0.125 percent sample of the net area covered during each year of control. Dwarfmistletoe-infected (prunable and not prunable) and healthy stems were tallied on each plot by size classes. Since plots were also classified according to the level of initial control (presumed to be indicative of degree infected at that time) and present infection, it was possible to outline three major problem areas on the control unit map.

   Results of the evaluation follow:

   (1) Direct control was applied originally to only 47 percent of the net area; it was moderate or severe on only 27 percent.

   (2) Dwarfmistletoe has developed on 79 percent of the plots in stands where initial control was severe, 33 percent where it was moderate, 19 percent where it was light to negligible, and in 8 percent of the plots where control was not applied at all.

   (3) About 160,000 pines have dwarfmistletoe. This number is 35 percent of the estimated total treated originally. At least 25 percent of

the infected trees can be freed of dwarfmistletoe by pruning as compared with an estimated 4 percent initially. (Andrews, RM)

b. A plot study was installed in western Montana to measure the development of dwarfmistletoe in a 60-year-old western larch stand following thinning. The study consists of 8 plots and 4 treatments with one replication of each treatment. Spread and intensification of dwarfmistletoe under various thinning intensities and under various degrees of infection in individual host trees will be measured. Growth of trees with different degrees of infection (free, light, medium, heavy) will be compared within thinning treatments. (Graham, INT)

c. Results of dwarfmistletoe surveys in the Kaniksu and Nezperce National Forests were published. Survey results emphasize the magnitude of the dwarfmistletoe problem in these two forests. (Graham INT)

d. The presence of dwarfmistletoe by host and locality is being recorded on the Station's forest survey plots in northern Idaho and on the Region's timber management inventory plots in Montana. (Graham, INT)

e. A post-control appraisal survey was made in 33 lodgepole pine clearcut blocks on two national forests in eastern Montana to determine the effectiveness of the control operation undertaken in the fall of 1959. Results show that clearcut blocks can be sanitized to a tolerable level but that many infected trees will be missed during adverse weather conditions, and that more intensive training in recognition of dwarfmistletoe by sanitation crews is needed. (Graham, INT)

f. Field work was completed on a cooperative study between the Rocky Mountain and Intermountain Stations to determine (1) the distance of spread and degree of infection in lodgepole pine reproduction from infected border stands and from individual or groups of infected overstory trees and (2) the minimum age at which lodgepole pine reproduction will become infected. Data and samples were taken on 44 study areas in the Rocky Mountain Station territory and on 35 study areas in the Intermountain Stetion territory. Results have not been completely analyzed. (Hawksworth, RM and Graham, INT)

g. A dwarfmistletoe observation plot, established in 1920 on the Plumas National Forest and discontinued in 1933,

was relocated and re-examined to determine the changes in status of the parasite for the 40 year period. Results are not yet fully compiled. In the trees still present in 1933, net increase in counted infections between 1920 and 1933 was 635 but 37 percent of the trees showed a decrease in infections present. There appears to be no close correlation between intensity of infection and tree mortality over the total 40 year interval. (Wagener, PSW)

h. On either red or white fir, approximately 85% of the mistletoe seeds intercepted by the trees were deposited originally on needles and 15% on branch wood. Following the first rain storm, about 70% of the seeds on red fir had become favorably placed on branch wood, whereas only about 35% of the seeds on white fir had become so placed. Differences in the numbers of needles per unit length of branch and differences in the usual angle of needle growth appeared to account for the differences in placement of seeds. (Scharpf, PSW and Parmeter, Univ. of Cal., Berkeley).

2. Biological and Chemical Control

a. Septogloeum gillii was found on A. americanum in one locality on the Arapaho National Forest, Colorado. This is the first time the disease has been noticed in the Central Rockies. (Hawksworth, RM)

b. In the fall of 1959 formulations of a number of plant hormone herbicides (2, 4-DA; 2,4-DP; 2,4,5-TA; MCPA; MCPP; etc.) were diluted with kerosene to 2,000 to 5,000 ppm. concentration and sprayed on dwarfmistletoe cankers on Jeffrey pine saplings and poles. Many cankers, including some large trunk cankers on 6- to 10-inch trees still appeared dead in September 1960. The same materials were applied as basal stem sprays to DM infested Jeffrey pines. Some trees were damaged; a few were killed. A very few treatments still hold some promise of killing dwarfmistletoe without seriously damaging the host. (Quick, PSW)

c. In 1960, 100 tests (893 trees) were made with a wide assortment of herbicidal chemicals. Most materials were applied in stove oil carrier; a few in kerosene, and a few in diesel oil. Two other carriers--one part agricultural spray oil in four parts water and one part polyglycol in four parts water--were sometimes used. Each material was sprayed directly on dwarfmistletoe plants and cankers and as a basal stem spray on infested trees. Basal stem treatments commonly included both smooth-barked (young) trees and rough-barked (older) trees. Treatments were distributed

among several forest species as follows: 22 tests (230 trees) on Jeffrey pine, east side of Lassen National Forest; 23 tests (210 trees) on sugar pine and white fir, Chowchilla Mtn., Sierra N. F.; and 12 tests (91 trees) on red fir, 22 tests (202 trees) on white fir, 15 tests (112 trees) on ponderosa pine, 6 tests (48 trees) on Jeffrey pine, all in the vicinity of Pinecrest, Stanislaus N. F.

Some of the trees showed chemical damage by the first of September. Herbicidal damage from basal-stem sprays with petroleum oil carriers tends to concentrate in the upper parts of crowns of red fir and white fir but appears and progresses rather evenly over the crowns of damaged Jeffrey pine and ponderosa pine. No preliminary checks have yet been made of sugar pines treated in 1960. (Quick, PSW)

d. In August 1953 some greenish, slug-like larvae were observed eating dwarfmistletoe on heavily infested sugar pine saplings southeast of Meadow Valley, Plumas N.F. The larvae were taken to Berkeley and reared to maturity on the dwarfmistletoe. The imago was identified as the Great Purple Hairstreak (Atlides halesus Cramer, Lycaenidae, Lepidoptera). (Quick, PSW)

3. Life History, Taxonomy, and Morphology

a. Studies were begun which should eventually provide data for life tables for A. americanum and A. vaginatum. Information is being recorded on what proportion of the seeds are intercepted by the trees, on redistribution of the seeds, germination, and infection. Some preliminary results of these studies were: (1) In a heavily infected ponderosa pine stand, 20 percent of the A. vaginatum seeds produced were intercepted by the trees; (2) For both A. vaginatum and A. americanum, 94 percent of the seeds that hit the trees were intercepted by the needles, and 6 percent by the twigs; (3) The intensity of the first rain following seed discharge appears to be critical in determining the amount of movement of seeds from needles to twigs. (Hawksworth, RM)

b. Mr. William Schacht, a forestry student at Duke University, plans to write his Master's thesis on the morphology of the endophytic system of A. vaginatum. Specimens for the study were collected this summer in Colorado. The main emphasis of this work will be on the character of the endophytic system of young infections. This study will complement the one by Wallace Eslyn on older infections (See below). (Hawksworth, RM)

c. In a laboratory study of 36 artificially infected branches of ponderosa pine, sinkers of southwestern dwarfmistletoe (Arceuthobium vaginatum) were found as much as 7.2 cm. beyond any evidence of swelling and a maximum of 8.1 cm. beyond the youngest shoots.. Age of host tissue when inoculated, sex of the dwarf-mistletoe plant, and direction of growth had had no significant effect on the rate of lateral extension. The annual rate of extension of the endophytic system averaged 1.0 cm. in each direction. (Eslyn, RM)

d. Scotch pine planted along edge of an old homestead clearing in Northeast Washington was found heavily infected with the larch dwarfmistletoe and lightly infected with the lodgepole pine dwarfmistletoe. (Graham and Leaphart, INT)

e. A stand of limber pine infected with the cyanocarpum form of western dwarfmistletoe was found near Red Lodge in eastern Montana. Heavy and extensive damage by this dwarfmistletoe also occurs in stands of limber pine in the Craters of The Moon national monument in southern Idaho. (Graham & Leaphart, INT)

f. Numerous localized branch cankers on lodgepole pine in western Wyoming were found to be infected with both dwarfmistletoe and comandra blister rust. It appears that this association between dwarfmistletoe and comandra rust occurred frequently enough to be more than accidental. (Kimmey, INT)

Biology, morphology, taxonomy, etc.:

g. The viability of seeds of Arceuthobium campylopodum on red and white firs was determined by observing seed germination in the field. Approximately 80% of the seeds on red fir and 70% of the seeds on white fir had germinated by June of the year following dissemination. Few seeds germinated after June and seeds which did not germinate by December of the same year were found to be non-viable. (Scharpf, PSW, and Parmeter, Univ. Cal. Berkeley)

h. Seeds collected for testing retained viability longer when stored at 36°F than seeds held at 5°F, 56°F. and room temperature. Approximately 80% of the seeds from dwarf-mistletoe on fir remained viable after 100 days at 36°F. Dry storage prevented moulding of the seeds. (Scharpf, PSW, Parmeter, Univ. Cal. Berkeley)

i. Laboratory tests showed that seeds germinate readily at about 56°F. Light, though not required, significantly

reduced the time necessary for germination. Growing radicles of seeds in the light show a more rapid rate of elongation than radicles of seeds in the dark, other conditions being equal. (Scharpf, PSW and Parmeter Univ. Cal. Berkeley)

4. Host-parasite Relationships.

a. A. americanum was found on an Engelmann spruce in Colorado. Although only one spruce in a mixed spruce-lodgepole pine stand was infected, this tree had over 100 brooms and appeared to be more susceptible than the adjacent lodgepole pines. (Hawksworth, RM)

b. A. americanum was found in three pure ponderosa pine stands in Colorado. The parasite appeared to be just as damaging as in lodgepole pine. A. americanum is common on ponderosa pine in mixed ponderosa-lodgepole stands, but it had not been noted previously in pure ponderosa pine. (Hawksworth, RM)

c. An investigation of intermediate fungus species which prepared conditions suitable for Basidiomycetes in cankers associated with dwarfmistletoe on western hemlock revealed twelve different species of fungi--nine ascomycetes and three Fungi Imperfecti. Two were undescribed species, Durandiella tsugeae and Helotium columbianum, and one, Mytilidion decipiens Karst., had not been found previously in North America. Tests of seven species to determine parasitism revealed only one of the Fungi Imperfecti to show slight parasitism. (Baranyay, Calgary)

d. In a study of canker distribution on young white firs, no cankers were found in the upper 7 branch whorls of any of the trees examined, and cankers were rare in the next 3 whorls. Thus, most cankers were found on branch whorls older than 10 years. On these older branches, cankers were common on growth segments as young as 3 years, however. (Scharpf, PSW, and Parmeter, Univ. Cal., Berkeley)

e. Observation of germinated seeds of dwarfmistletoe on red and white firs in the field indicates that in most cases the radicle of a seed penetrates the host branch at the acute angle made by the branch and a needle. Approximately 85% of the germinated seeds on red fir and 75% of the germinated seeds on white fir appeared to penetrate at this point on the branch. Killing of the needle commonly occurs in red fir as the radicle wedges itself firmly between the needle and the branch.

The significance of this needle killing to infection by dwarfmistletoe is not yet known. (Scharpf, PSW, and Parmeter, Univ. Cal., Berkeley)

f. Penetration of red fir by dwarfmistletoe occurs in the summer and fall of the year following seed deposition. Symptoms of infection do not occur until the second year following seed dissemination. A noticeable swelling of the branch at the point of penetration indicates infection. Continued observation will be made to determine the presence of latent infections. (Scharpf, PSW and Parmeter, Univ. Cal., Berkeley)

g. Freshly collected dwarfmistletoe seed from Douglas-fir, Pacific silver fir, western larch, and western hemlock hosts do not remain viable when stored under conditions comparable to those used for tree seed storage. Molds attacked seeds stratified in moist sand. Moisture and the gelatenous sheath surrounding the seed apparently are significant in mold development. Naturally expelled seed collected from Douglas-fir hosts and held for three months in the laboratory germinated readily. When placed on Douglas-fir seedlings in the greenhouse, radicles developed and were observed to make contact with host tissues. (Shaw, WSU)

h. Tests are underway to determine the susceptibility of the coastal form of Douglas-fir to dwarfmistletoe from the mountain form. Other inoculation trials using the mountain form of Douglas-fir and western larch are being made to determine the susceptibility of host tissues of varied ages. (Shaw, WSU)

i. Studies of host-parasite relations using radio isotopes have shown that dwarfmistletoe of lodgepole pine conducts phytosynthesis and the photosynthates are rapidly translocated throughout the host. Conversely, tagged photosynthates from the host moved readily through the xylem or phloem into the dwarfmistletoe plant. The principal effect of dwarfmistletoe on the host appears to be a "girdle" effect which results in an accumulation of host phytosynthates at the infection site. (Rediske and Shea, Weyerhaeuser)

Needed Research and Studies not yet Reported on

1. Intensification and spread:

a. A problem analysis on dwarfmistletoe entitled "Research and survey needs in a program to control the dwarfmistletoes of Douglas-fir, lodgepole pine, and western

larch in the Intermountain and northern Rocky Mountain regions" was completed. This analysis gives the major problems, information needed to solve the problems, and suggests some 30 individual studies needed to provide the necessary information. (Leaphart, INT)

b. To be initiated in 1961 is a project on losses resulting from growth reduction and mortality in lodgepole pine stands infected by dwarfmistletoe. Objectives are: (a) to demonstrate the amount of losses resulting from growth reduction in different age classes under a variety of site conditions and intensities of infection. (b) to demonstrate the extent of mortality that results in different age classes, under a variety of site conditions and intensities of infection. (Baranyay, Calgary)

c. We have established several permanent plots, and will establish additional ones, for experimental study of productivity differences between sanitized and unsanitized ponderosa stands and rate of intensification of the pest under a fairly wide range of conditions. (Childs, PNW)

d. Measuring mistletoe impact in managed ponderosa stands was initiated so that an appraisal can be made of the economics of control. This information is needed now, and we cannot wait for experimental results. Consequently, we have put at least half of the effort of the Division this year into a field study of mistletoe effects on volume increment in stands of various ages up to about 150 years. We have also started an effort to get intensification data by means of stand reconstruction. These 2 jobs will be continued at least through next year. (Childs, PNW)

e. Investigations were initiated on the effects of dwarfmistletoe on growth of Douglas-fir near Klamath Falls, Oregon. To date, stem analyses of 32 mature trees suggest that light infections have no marked effect on increment whereas moderate and severe infections reduced increment corresponding to the degree of infection. Additional analyses are planned. (Shea, Weyerhaeuser)

2. Biological and Chemical Control.

a. Preliminary greenhouse trials have indicated several promising new phytocides for the control of dwarfmistletoe on Douglas-fir and pine. Continued experimentation is planned. (Rediske, Shea, Weyerhaeuser)

3. Life History, Taxonomy, and Morphology:

a. Transpiration rates of A. campylopodum Engelm. on all important forest species perhaps should be studied. The variation of transpiration rates, if any, with advancement of season and with stage of growth would be of interest. Most forest trees in California grow very rapidly for a few weeks in June and July, then start into growth dormancy. Dwarfmistletoe appears to be in active growth stages farther into the summer and fall than the conifer hosts. Susceptibility to hormone type herbicides commonly is more or less proportional to growth activity. When would the conifers be relatively most dormant and the dwarfmistletoe relatively most active? (Quick, PSW)

4. Host-parasite relations:

a. Studies on host specialization of dwarfmistletoes have been started in California. Particular attention is being given to the forms of A. campylopodum. Cross inoculations have been made in the greenhouse and host specialization is being checked in the field. (Scharpf, PSW and Parmeter, Univ. Cal. Berkeley)

b. The effect of bark thickness to infection is being studied in the field and in the laboratory. (Scharpf, PSW and Parmeter, Univ. Cal., Berkeley)

c. Studies on the fungi associated with dwarfmistletoe infections should be extended. Plans have been made to study decay fungi associated with bole cankers on red and white fir. (Scharpf, PSW and Parmeter, Univ. Cal., Berkeley)

d. Dwarfmistletoe (Arceuthobium campylopodum) on white fir (Abies concolor) was chosen for studying the translocation relationships between these two plants. The location of the study was near Strawberry in Caliveras County, California, at an elevation of about 5500 feet. Only the female plants of the dwarfmistletoe have been employed in the studies thus far. Experiments have been conducted as briefly described: The following C-14 compounds have been used in these studies; $BaCO_3$, 2, 4-D (2,4-dichlorophenoxyacetic acid) (3-amino-1, 2,4-triazole) or amitrol, and atrazine)2-chloro-4-ethylamino-6-isopropylamino-s-triazine). The total activity used was about 63 microcuries per treatment with the $BaCO_3$ and 0.5 micro-curies with the 2,4-D, amitrol, and atrazine. The $BaCO_3$ was used to serve as a source of CO_2 which could be used by the fir

and the mistletoe for photosynthesis. The translocation of the photosynthate could then be studied by autoradiography, counting, and chromatography. The plan is to conduct similar studies in the fall, winter, spring, and early summer. In addition, the mistletoe is being manipulated with the hope that it can become a more effective "sink". The three herbicides have been applied to the leaves of the fir and the stems of the mistletoe. In addition, applications were made to the bark and the wood of the fir. (Leonard and Hull, Univ. Calif., Davis)

IN MEMORIUM

HANS N. HANSEN

1891 - 1960

Professor Emeritus Hans N. Hansen passed away in Lafayette, California, on April 26, 1960. He was born in Varde, Denmark, November 8, 1891, came to the United States at the age of 17, and became a naturalized citizen in 1918. He attended the University of California, obtaining his B.S. in 1924, M.S. in 1925 and the Ph.D. in Plant Pathology in 1928, when he joined the faculty of the University. He retired in 1959.

Dr. Hansen's principal field of research was the fungi which cause plant disease, with specialties in the diseases of figs, forest trees, shade trees, and ornamentals, and in the variation phenomena in fungi. His research in the genetics of variation in fungi and the effect of variation on taxonomy led to world-wide recognition in this field and to a simplified scheme for the classification of species, especially of the genus Fusarium.

As a teacher he was highly respected and sought after. He guided and inspired many undergraduate and graduate students who are now dispersed to various parts of the world. He also organized and offered the course in Forest Pathology at the University. He will be deeply missed by his many friends, colleagues, and students.--R. V. Bega.

ALBERT W. SLIPP

1906 - 1959

Albert W. Slipp died in his home at Moscow, Idaho on December 10, 1959, shortly after the conclusion of the 6th W.I.F.D.W.C. Although he had been in ill health for many years, his death was still unexpected, so well did he bear up under his illness.

Professor Slipp was born August 10, 1906 in North Sydney, Nova Scotia, Canada. After graduating from the University of New Brunswick in 1930 with a B.S. in Forestry, he took graduate work at Harvard, Emory and the University of Idaho, where he received a Master of Forestry degree in 1939. Initially he devoted his attention to full-time research, becoming a recognized authority on white pine blister rust. He was also

interested in fungus ecology, and published on the distribution of the Boletes in the Inland Empire. In 1945 he added teaching responsibilities to his research program.

Professor Slipp was a member of numerous scientific organizations, including Sigma Xi, American Phytopathological Society, the Society of American Foresters, the Northwest Scientific Association and the Idaho Academy of Science.

His cheerfulness and perseverance will long be remembered by all who knew him.--C. G. Shaw

CHARLES W. WATERS

1897 - 1960

Dr. Charles W. Waters died unexpectedly on January 21, 1960 from exposure to fumes emanating from a faulty furnace. He is survived by his widow, a son, and two grandchildren.

Dr. Waters was born in Oketo, Kansas on December 7, 1897. He attended successively Berea College (B. L., 1917; B.S. 1919), Ohio State University (M.A., 1921) and the University of Michigan (Ph.D., 1927). After teaching for brief periods at Syracuse University, Miami University, and the University of Michigan, in 1926 he joined the staff of Montana State University as assistant professor of Botany and Forest Pathology. His knowledge of the fungus diseases of trees was comprehensive and he was deeply concerned with the economic drain which these diseases made on our valuable forest stands. For more than 30 years Professor Waters has been known not only for his own investigations on rust diseases, mycorrhizae, ponderosa pine needle blight, mistletoe infestations, and other forest tree diseases, but also for his excellence as a teacher. His enthusiasm, friendliness, and encouragement resulted in many of his students choosing Forestry or Forest Pathology as their career.

Dr. Waters was a member of the American Phytopathological Society, Society of American Foresters, the Northwest Scientific Association, and the Montana Academy of Science.

His untimely passing is cause for sorrow among his colleagues, friends and former students.--C. G. Shaw