AN ABSTRACT OF THE THESIS OF

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Title: COMPARISON OF PHELLINUS WEIRII ROOT ROT DAMAGE IN A 60-YEAR-OLD DOUGLAS-FIR (PSEUDOTSUGA MENZIESII) STAND WITH THE DAMAGE IN THE PRECEDING OLD-GROWTH STAND

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Laminated root rot of Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) caused by the fungus Phellinus weirii (Murr.)Gilbertson was studied in two successive stands in the Oregon Coast Range. Damage due to the disease in a 60-year-old second-growth stand was compared with incidence in the preceding 300-year-old stand on the same site. Information for the comparison was gathered by mapping all current stand trees (living and dead) and the remains of previous stand trees on four one hectare plots. Damage in each stand generation was assessed in terms of the reduction in stand density and basal area by P. weirii caused mortality, the extent of infection among living trees, and the plot area covered by the disease. Current stand damage estimates were based on the amounts and distribution of healthy, infected and dead trees. Live infected trees were diagnosed by the presence of either surface mycelium or internal decay as evidenced by root collar excavation and increment boring of all trees around disease centers. Previous stand damage estimates were based on

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

The comparisons of damage revealed that laminated root rot was more destructive in the previous stand at the time of its harvest than in the current second-growth stand. The reduction in stand density by P. weirii caused mortality was greater in the previous stand (75.5%) than in the current stand (41.7%). In the previous stand 60.9% of the trees living at harvest were infected while 35.1% of the living trees were infected in the current stand. The reduction of Expected Basal Area (of a healthy stand on the site) by P. weirii caused mortality was also greater in the previous stand (56.6%) than in the current stand (22.5%). The proportion of the Actual Basal Area infected was also higher in the previous stand (64.8%) than in the current stand (41.1%).

Phellinus weirii was present on 82.5% of the previous stand plot area compared to 51.2% of the current stand plot area. The difference is largely attributable to the larger Area of Concentrated Mortality in the previous stand (68.4% vs. 36.3%). Differences between plots in the Total Area of Infection in the current stand were best related to differences in the type of inoculum from the previous stand and area affected by these inoculum sources (Area of Potential Inoculum). Sixty-five percent of the currently diseased area lay beyond the Area of Potential Inoculum from the previous stand, indicating significant tree to tree spread in the current stand.

Although damage levels were higher in the previous stand (at its harvest) than those currently found, the average annual rate of damage increase was much higher in the current stand than the previous stand.

Projections of current damage rates to a comparable age predict much higher losses due to the disease in the current stand than were found in the previous stand.

Comparison of *Phellinus weirii* Root Rot Damage in a 60-year-old Douglas-fir (*Pseudotsuga menziesii*) Stand with the Damage in the Preceding Old-growth Stand

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Comparison of Phellinus weirii Root Rot Damage in a 60-year-old Douglas-fir (Pseudotsuga menziesii) Stand with the Damage in the Preceding Old-Growth Stand.

I. INTRODUCTION

Laminated root rot, caused by the fungus Phellinus weirii (Murr.) Gilbertson, is one of the most damaging diseases of Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) in the Pacific Northwest. This endemic pathogen causes an estimated annual loss of 32 million cubic feet of timber in the Douglas-fir region of Oregon and Washington (Childs and Shea 1967). The long-term survival of the pathogen as a saprophyte in stumps after the harvest of an infected Douglas-fir stand, and its subsequent infection of Douglas-fir regeneration on the site have led to speculation on the potential for increasing damage under intensive management (Shea 1958; Childs 1963, 1970). Surveys of infected stands consistently show a steadily increasing incidence of mortality due to P. weirii through time and predict high losses at rotation age (Johnson et al. 1972; Childs 1970). To date, all of the studies concerned with P. weirii damage in second-growth stands have dealt with estimation of rates of damage within the time frame of the current stand only. Comparisons of damage in stands of different ages have been made for stands on similar sites (Childs 1970). However, none of these attempted to directly relate the amount and distribution of damage in the current stand to the type and extent of infection in the preceding old-growth stand. Such a documantation of the history of P. weirii on the site is essential for accurate prediction of losses in future forest generations.

The prime objective of this study was to directly compare the damage caused by Phellinus weirii root rot in a 60 year-old second-growth Douglas-fir stand with the reconstructed P. weirii damage in the previous stand on the same site. Comparisons of damage were made from information gathered by mapping all current stand trees and previous stand residuals on four separate one hectare plots. Damage due to the disease in each of the two stands was assessed in terms of the reduction of density and volume by mortality, extent of infection in standing trees, and area covered by the disease. This thesis has been divided into several sections, each with its own description of the background, methods, and results as well as a discussion of the particular study. A comprehensive summary of the most pertinent results of these studies and a discussion of the ramifications of the results to forest management are presented in the final section.

II. LITERATURE REVIEW

The causal fungus of laminated root rot was originally described by Murrill (1914) as Fomitoporia weirii Murrill from a collection made in 1912 by James Weir in Idaho on western redcedar (Thuja plicata Donn.). In the same article Murrill recombined the species as Poria weirii Murrill to conform with Saccardo's nomenclature. The first report of P. weirii on Douglas-fir was by Mounce et al.(1940) from a collection made in 1929 on Vancouver Island, B.C. in an 18 year-old stand. They concluded that despite some differences, the root rot organism from Douglas-fir was indeed Poria weirii as described by Murrill or a form of it. On the basis of microscopic characteristics, Gilbertson (1974) placed the fungus in the genus Phellinus in the family Hymenochaetaceae.

A. Diagnosis of laminated root rot

Since the appearance of P. weirii fruiting structures in nature and in culture is sporadic, one must rely on the structure of the vegetative mycelium, the characteristic decay of wood, and the manifestations of the disease in the forest for proper diagnosis.

1. Cultural diagnosis. The appearance of P. weirii vegetative mycelium in culture is very distinctive (Mounce et al.1940; Nobles 1948, 1965). The hyphae of the advancing zone in culture are hyaline, thin-walled, septate with branching usually occurring below a septum. In the aerial mycelium the hyphae are yellow to brown in KOH with thick walls and often end in brown setal hyphae which are 4.5 - 6.0 μ in diameter and up to 350 μ long (Mounce et al. 1940).

P. weirii in culture can be confused with Phellinus ferrugineo-fuscus (P. Karst)Bourd. However, the two species can be separated by different growth rates and colony morphologies (Nobles 1948, 1965). In nature, fruiting structures of the two species are easily separated by the positioning of setal hyphae relative to the hymenium in the basidiocarps (Gilbertson 1974).

2. Field diagnosis. The disease caused by P. weirii can be readily recognized in the field due to the characteristic decay it causes in Douglas-fir wood (Mounce et al. 1940; Childs 1970; Partridge and Miller 1974; Hadfield and Johnson 1977). The incipient stage of decay appears as a brown stain which is often crescent shaped in cross section of the stem and appears as streaks or bands in longitudinal sections. This discoloration can occur up to 4 to 6 feet ahead of the advanced stage (Mounce et al. 1940). Although the stain is usually in the heartwood, it can be found in the sapwood. In advanced decay the wood is yellowish, laminated, and pitted. The oval pits are about 0.5 mm wide and 1 mm long and are filled with white fibers. The wood separates along the annual rings and tufts of reddish brown hyphae are found between the layers. This mycelium contains hyaline as well as brown setal hyphae which can be readily seen with a 10x hand lens. The mycelium often forms a cinnamon-brown crust over the exposed ends of decayed roots. In the final stages of decay the wood degenerates to a stringy mass. Eventually even this disintegrates leaving a hollow area in the butt of the tree (Childs 1970).

External symptoms of laminated root rot such as reduced terminal growth and discoloration of foliage do not become evident until a

large portion of the roots are diseased. These same symptoms can be caused by a number of other factors and are not by themselves diagnostic (Bega 1978). Wallis and Reynolds (1965) found that the percentage of roots killed before symptom expression was quite variable. Buckland et al.(1954) described trees with two types of host response to infection: those that died rapidly upon infection, and those that tolerated infection but were decayed internally.

Some of the other common decay fungi of Douglas-fir that can be confused with P. weirii are: Armillariella mellea (Vahl. ex Fr.) Karst., Fomes annosus (Fr.)Karst., and Phellinus pini (Thore ex Fr.)Pilat. All of these cause a white rot but do not produce any setal hyphae in the decayed wood (Partridge and Miller 1974). Infection by A. mellea can be diagnosed by the presence of exudation at the root collar and necrotic lesions with white mycelial fans under the bark of roots and the root collar. Rhizomorphs can be found in the soil around roots as well as beneath the bark. A. mellea also forms mushroom fruiting bodies. Advanced decay is yellowish or white with a spongy texture (Morrison 1976). Infection of Douglas-fir by F. annosus rarely causes direct mortality. It causes a red-brown incipient decay and a yellow, pitted stringy advanced decay. Perennial leathery fruiting bodies are formed on roots and stems of windthrown trees and stumps (Wallis and Ginns 1976). P. pini causes a reddish-purple to olive incipient decay. The advanced decay is reddish with small pits containing white fibers. Cross sections of decayed wood may reveal a ring-like distribution of pits (Partridge and Miller 1974). The abundance of perennial fruiting bodies and lack of setal hyphae in decayed wood readily distinguish this fungus

from P. weirii (Bega 1978).

B. Disease development

1. Initial infection of young stands. The initial sources of P. weirii inoculum for a young stand of Douglas-fir are infected stumps and other residuals from the previous stand. Since the fungus is capable of surviving as a saprophyte after the death of the host, it can reinfect new regeneration on the site (Buckland et al. 1954; Wallis and Reynolds 1965). The survival of the fungus in old stumps is facilitated by resin impregnation of the wood which helps protect it from invasion by antagonistic microorganisms (Buckland et al. 1954). P. weirii also protects itself by the formation of zone lines. Nelson (1973, 1975) found that survival of P. weirii under unfavorable conditions was directly related to zone line formation. These zone lines consisted of darkly pigmented thick-walled hyphae and completely enveloped the fungus.

Although the fungus has been found to survive up to 50 years after the death of the tree (Wallis and Reynolds 1965; Hansen 1979c), there has been no conclusive proof that the fungus is still infectious at that age. Hansen (1976) found that 94% of the stumps that showed indications of past P. weirii decay still had viable P. weirii 20 years after harvest. The fungus was found only in roots with intact bark. Living ectotrophic mycelium was found on 33% of the stumps. However, the fungus was in a "seemingly precarious state" with the mycelium retreating behind zone lines. Further excavations in stands harvested 30 and 50 years earlier showed a drastic reduction

in the survival of P. weirii(Hansen 1979c). In stands with a current Douglas-fir overstory, P. weirii was alive in 59% of the 30 year-old stumps and in 26% of the 50 year-old stumps. There was no evidence of P. weirii spread into sound wood in a stump after tree harvest. The appearance of surface mycelium decreased as the stump age increased. In the older stumps, viable P. weirii was found in larger roots and closer to the stump indicating a progressive retreat.

In their excavations in 15 to 25 year-old Douglas-fir stands, Wallis and Reynolds (1965) found that "infection arose when the living roots came into contact with viable mycelia persisting in the roots of trees of the previous rotation". The trees of the previous stand had been cut at least 50 years previously and had been over 400 years old. Many of the larger roots still contained viable P. weirii. Buckland et al.(1954) found P. weirii infecting a 40 year-old Douglas-fir through a pressure point on a root that had grown through the infected root of a 380 year-old Douglas-fir felled 50 years previously. Neither of these studies examined the timing of the infection of second-growth trees by P. weirii in the stumps. Infection could have occurred at an early stage of stand development or it could have been a recurring phenomenon.

On the basis of numerous observations in second-growth stands, Childs (1970) stated that "the great majority of infection centers in present-day stands are attributable to vegetative persistance of the fungus in centers that originated in previous stands". He also postulated that differences in the amount and distribution of disease in the current stands may be "correlated with past differences in infection

or type". The type of infection found in old-growth stands varied considerably. Childs (1970) found that although characteristic P. weirii root rot centers can be found in old-growth Douglas-fir, often the disease manifested itself rather differently. On one clearcut area, for example, there were no rot-thrown trees and no obviously visible indicators of disease were found in the contiguous uncut stand. However, the stump surfaces indicated that 27% of the living Douglas-fir trees were infected. Of the standing snags, 33% had signs of P. weirii decay. A higher percentage of western hemlock than normal for comparable sites caused Childs to speculate that P. weirii may have caused more mortality of Douglas-fir when the stand was young.

Stumps of previously infected trees were found to cause infection of succeeding stands by several other root pathogens. Initial infection of Ponderosa pine (Pinus ponderosa Laws.) stands by Armillariella mellea was found to arise from infected stumps of harvested old-growth trees (Shaw 1974). Fomes annosus infected stumps can lead to infection of second-growth western hemlock (Driver and Wood 1968). Whitney (1972) found that white spruce was infected by Inonotus tomentosus (Fr.) Gilbertson from stumps of previously killed trees.

2. Spread within stand. Wallis and Reynolds (1965) found that after initial infection from previous stand residuals, "P. weirii spread distally and proximally from the point of infection". Spread of P. weirii through a stand of Douglas-fir is facilitated by ectotrophic mycelial growth on the bark of infected roots. This mycelium is characterized by a dense, fanlike white advancing margin. Behind

this margin is a white mat covered by a brown crust. Wallis and Reynolds (1965) found that occasionally this mat extended up to 8 cm into the surrounding soil. Roots in the duff layer were seldom covered with this mycelium. The ectotrophic mycelium grew in advance of the internal decay. P. weirii was able to invade the wood directly through uninjured bark of roots at least 6 cm in diameter. The hyphae invaded the wood and formed the characteristic brown stain. Wallis and Reynolds (1965) concluded that: "intratree root contacts, observed on all trees excavated in this study, provide extensive pathways for the growth of the mycelium from root to root and appear to facilitate a rapid deterioration of the root systems and to hasten the death of trees". They also found more than 70 instances of P. weirii mycelium spreading from diseased to adjacent healthy trees through contact of the healthy roots with the fungus on infected roots. Buckland et al. (1954) found in numerous excavations of root systems that "the principal mode of transmission is through root fusion which forms a direct passage for the fungus". In a 35 year-old stand they found a series of 15 Douglas-fir trees fused and diseased. Spread of disease by root contacts and surface mycelial growth of the pathogen has also been reported for Fomes annosus in pines (Risbeth 1951) and Inonotus tomentosus on white spruce (Whitney 1962).

Root to root spread of P. weirii results in clearly defined disease foci or "pockets" in the stand. These openings can be easily diagnosed as P. weirii root rot centers by the numerous windthrown trees with "root balls" and P. weirii decay. The downed trees in the centers are typically in several stages of deterioration and are oriented in

several directions which distinguishes them from bark beetle attacks or blowdowns during storms (Hadfield and Johnson 1977). Root rot centers caused by Armillariella mellea are different in that many of the recently killed trees will be standing due to the rapid tree mortality induced by this fungus and delayed decay of the wood (Roth et al. 1977).

3. Growth through soil. Wallis (1976a) found that growth of P. weirii through unsterilized soil from an inoculum source was limited to a few centimeters. At a distance of 5 cm only 20% of the roots were infected from an inoculum source. None of the roots at 10 cm from the inoculum source were infected. Woody material not already colonized by other fungi was effective in bridging diseased and healthy roots.

4. Airborne spores as inoculum. Long distance spread of P. weirii by basidiospores has been postulated (Shea 1958; Childs 1963), but it has never been conclusively demonstrated. P. weirii has no known asexual conidial stage. A few instances of above ground infection of wounds by P. weirii have been found on western hemlock (Wright and Issac 1950) indicating possible spore infection. Nelson (1971) was unable to infect freshly cut stumps of Douglas-fir with P. weirii basidiospores, whereas vegetative inoculum did colonize 75% of the stumps. In a follow-up of his earlier work, Nelson (1976) looked at the conditions necessary for colonization of Douglas-fir wood by P. weirii basidiospores. Colonization of previously frozen and scalded wood disks was best at temperatures of 15° to 20° C after inoculation with an aqueous suspension of 400 or more basidiospores/ml. To date, there have been no reports of colonization of wounds or freshly cut stumps of Douglas-fir by P.weirii. Fomes annosus on the

other hand, readily infects both wounds and stumps via airborne spores (Hunt and Krueger 1962).

C. Douglas-fir root system morphology

Since local spread of Phellinus weirii is facilitated by root contacts, one of the primary factors affecting it must necessarily be the morphology of the host root system. To date, there have been relatively few comprehensive studies of the morphology of Douglas-fir root systems with a corresponding analysis of the factors affecting their development. McMinn (1963) excavated the root systems of 28 Douglas-firs ranging in age from 10 to 15 years. Young trees were found to have an aggressive taproot which suggests a potentially deep rooted species. Rooting depth was limited by soil depth. The size of the root system was related to crown rather than bole size. The radial symmetry was distorted by such factors as: slope, proximity to other trees, occurrence of old roots, and soil disturbance. The main laterals of dominants tapered rapidly due to frequent branching and did not extend a great distance from the root stock. The laterals descended into the lower soil horizons forming a buttressed configuration. Intermingling of roots of adjacent trees was evident even in densely rooted central portions of the root systems. McMinn (1963) also found that roots of the current stand growing along decaying roots of the previous stand extended farther than roots growing in mineral soil, hence increasing the possibility of infection by root rots from the residuals. Intertree root grafts were infrequent and occurred mainly between "closely adjacent" trees. Smith (1964) found

that average lateral root spread for Douglas-fir was best estimated by average crown width. The ratios of root spread to crown width averaged 1.1 for open- and 0.9 for forest-grown trees . Eis (1974) studied the morphology of western hemlock, western red cedar, and Douglas-fir root systems. He found that there was no general relationship of root spread to topography or crown configuration. Douglas-fir had consistently larger root spread and root diameter with smaller taper than the other species. Dominants of all three species had larger, more symmetrical root systems and more laterals than codominants and intermediates.

III. SITE DESCRIPTION

A. Stand Characteristics

The site selected for this study is located in the Chintimini-Woods Creek area on Mary's Peak west of Corvallis, Oregon (T12S R7W WM Sections 10 and 16). The stand is typical of Coast Range Douglas-fir forests. The mean annual precipitation ranges from 200-300 cm. and comes in the form of rains in the fall and winter with a dry period in the summer (Corliss and Dyrness 1965). The soil is an Inceptisol of the Slickrock series (Knezevich 1975). This deep, well-drained, gravelly loam formed in colluvium weathered from Tyee formation sandstone. A typical soil profile consists of the following horizons: A1 0-10 cm. - very dark brown gravelly loam; B1 10 -28 cm. - very dark greyish brown gravelly clay loam; B2 28-61 cm. - dark grayish brown gravelly clay loam; C 61-130+ cm. - weathered standstone and shale (E.E. Nelson PNW Forest and Range Exp. Sta. U.S.F.S. unpublished data). The vegetation on the site is characteristic of seral stages of the mesic associations within the Tsuga heterophylla climax zone (Franklin and Dyrness 1973). The principal component of the overstory is Douglas-fir with some western hemlock mixed in. Other overstory species are: bigleaf maple (Acer macrophyllum Pursh), red alder (Alnus rubra Bong.), western red cedar, and noble fir (Abies procera Rehd.). The understory consists of varying amounts of the following species: vine maple (Acer circinatum Pursh), salal Gaultheria shallon Pursh), Oregongrape (Berberis nervosa Pursh), swordfern (Polystichum munitum (Kaulf.)Presl), bracken fern (Pteridium aquilinum (L.)Kuhn), and

other species. The overstory is fairly even-aged and averages about 60 years old. The stand falls within Douglas-fir Site Class II and Site Index 165 (100 year base) (L.F. Roth, Oregon State Univ. personal communication). The previous stand overstory was primarily Douglas-fir with some western hemlock. The stand was approximately 300 years old and was railroad logged between 1910 and 1920, and had burned prior to regeneration of the current stand by natural seeding (E.M. Hansen, Oregon State Univ. personal communication).

B. Disease Description

1. Disease diagnosis. The causal agent of the root disease on the study site was diagnosed as being Phellinus weirii by utilizing a number of field and cultural characteristics. Field diagnosis was accomplished by noting the clumped distribution of windthrown trees in varying stages of decay (Hadfield and Johnson 1977; Wallis 1976b). Characteristic P. weirii decay with setal hyphae was found in the lower bole and roots of dead trees (Partridge and Miller 1974). Fruiting bodies were collected from infected and killed trees during the course of the study. These were sectioned (some with a freezing microtome) and examined under a compound microscope. All of the conks collected from suspected P. weirii mortality conformed to descriptions of that species (Mounce et al. 1940; Gilbertson 1974). P. weirii can be distinguished from other members of the Hymenochaetaceae by the shape and positioning of setal hyphae in the trama of the basidiocarp (Gilbertson 1974). During the course of the study numerous fungal isolations were attempted from trees suspected to be

infected with P. weirii. Wood chips from decayed areas were plated out on two percent malt agar with two mg/liter benomyl. The plates were incubated in the dark for up to two months and were periodically examined microscopically for the presence of P. weirii hyphae (Nobles 1948, 1965). Any new colonies which appeared were subcultured and also examined. All isolations from suspected P. weirii mortality yielded P. weirii in culture.

2. Disease in the stand. The area selected for this study has been the site of several other completed and on-going studies concerned with laminated root rot. Nelson (1970) studied the effects of nitrogen fertilizer on survival of Phellinus weirii in cubes buried in the area. Hansen (1979c) looked at survival of P. weirii in old-growth stumps 50 years after harvest. Data are being collected on a ten acre damage plot established by T.W. Childs 25 years ago (E.E. Nelson, PNW Forest and Range Exp. Sta. personal communication). A survey of the 200 hectare area (E.M. Hansen, Oregon State Univ. personal communication) revealed that up to 18% of the area was out of Douglas-fir production due to laminated root rot mortality. Due to its proximity to Corvallis, Oregon, the area has been used extensively for field exercises by forest pathology classes under the direction of L.F. Roth and E.M. Hansen of Oregon State University.

The activity of P. weirii root rot in the area resulted in the formation of some sizeable openings in the otherwise closed canopy (Figure 1). These openings supported very lush growth of shrub species such as vine maple, salal, and Oregongrape. Regeneration by Douglas-fir

Figure 1. View across Phellinus weirii infection center in study area.

was seldom successful due to competition for light with these shrubs and reinfection by P. weirii. Old-growth stumps were often found in close proximity to P. weirri mortality suggesting possible inoculum sources for the current stand. The presence of extensive root rot mortality in the current stand and the frequent occurrence of previous stand residuals indicated that the area would be suitable for a study comparing damage in two succeeding stands.

C. Plot Description

The four separate plots mapped during this study were approximately one hectare each. Plot locations were not randomly selected. The following criteria were used in plot selection:

1) Phellinus weirii root rot damage present in the current stand;
2) previous stand residuals present (stumps and snags) and consisting primarily of Douglas-fir;
3) signs of P. weirii decay in previous stand residuals.

The four plots were located within 1.5 kilometers of each other to minimize differences in site influence on disease development (Figure 2). A travel log and accurate plot locations are presented in Appendix 1.

The plots were named as follows: Woods Creek (WC) 2, WC3, WC4, and WC5. Although the plots were generally similar in site characteristics, there were some differences related to aspect and elevation. Plot WC2 was on a south slope at about 440 m. above sea level. The average diameter at breast height (DBH at 1.3 m) was 25-26 cm. The south aspect resulted in a drier type community with an understory of salal, vine

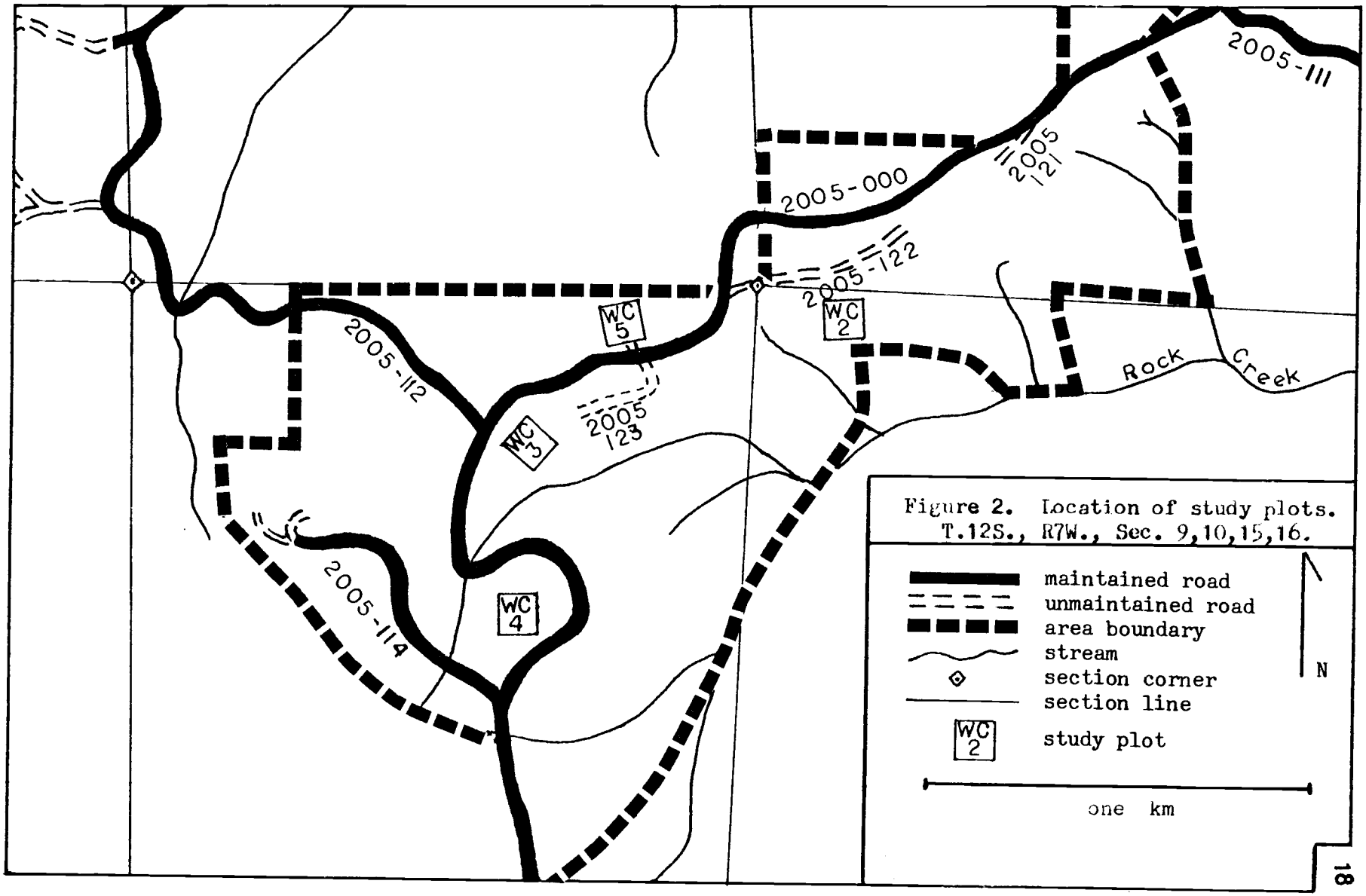


Figure 2. Location of study plots. T.12S., R7W., Sec. 9,10,15,16.

maple, and oceanspray (Holodiscus discolor (Pursh)Maxim. The stand was rather dense with about 1450 trees/ha. Plot WC3 was also on a south slope but was at an elevation of 530 m. The average DBH was 36-38 cm and the density was about 870 trees/ha. The understory consisted of salal and swordfern with some vine maple and oceanspray in the larger openings in the canopy. Plot WC4 was the highest in elevation at 610 m. The average DBH was 31-33 cm and density was about 940 trees/ha. The plot was fairly level but was at the base of a slope with poor drainage of cold air in the winter resulting in heavier frost than the other plots. The predominantly Douglas-fir overstory contained some western hemlock and noble fir. The understory was mainly salal and swordfern. This plot was located in one corner of the ten acre damage plot being studied by the Forest Service. Plot WC5 was on a slight northern slope at about 500 m. The average DBH was 34-35 cm with a density of 860 trees/ ha. The understory was of salal and swordfern with some vine maple in the canopy openings.

IV. DETERMINATION OF PHELLINUS WEIRII INFECTION IN LIVE, STANDING DOUGLAS-FIR

A. Background

Phellinus weirii is a relatively slow growing fungus which usually does not cause the rapid death of host trees past the sapling stage. At times it does rapidly colonize the host cambial region and can lead to subsequent mortality of standing trees (Buckland et al. 1954). However, Wallis and Reynolds (1965) found that in 25-45 year-old Douglas-fir death by girdling was relatively infrequent and was usually limited to smaller trees. More commonly infected trees remained standing until a large portion of the supporting lateral roots were decayed. The expression of crown symptoms was found to be related to the proportion of roots killed, but there was considerable variation among individual trees (Wallis and Reynolds 1965).

Childs (1970) found that 60% of the trees within 30 feet of P. weirii kills were also infected. Therefore, to accurately estimate the distribution of P. weirii in a stand one must certainly consider standing, infected trees. Damage evaluations based solely on dead trees and those with crown symptoms would grossly underestimate the extent of infection, since a great number of infected trees show no external evidence of disease. A preliminary study was deemed necessary to find an efficient way of determining the extent of this "hidden" infection without resorting to destructive sampling. A sample of living trees on the perimeter of P. weirii openings were examined for the presence of signs or symptoms of P. weirii root rot.

Crown symptoms of P. weirii root rot have been described by various workers (Mounce et al. 1940; Buckland et al. 1954; Childs 1970; Wallis 1976b; Hadfield and Johnson 1978). Determination of internal decay by P. weirii has most often been done by examining stumps of freshly cut trees for the presence of P. weirii stain or advanced decay (Buckland et al. 1954; Childs 1970; Wallis and Reynolds 1965). Childs (1970) examined increment cores from living trees to check for infection as indicated by discoloration or decay. He added that: "a few infected trees were undoubtedly diagnosed as uninfected". Increment core sampling for laboratory isolations indicated a higher incidence of Fomitopsis annosa infection on Pinus species in North Carolina than did other signs of the fungus (Cordell and Stambaugh 1966). Dimitri (1968), working with F. annosa infection of spruce in Germany, found that increment core sampling detected only 46% of the diseased trees shown by disk sampling.

Other methods for determining internal decay in trees include the use of X-rays (Eslyn 1959), sonics (Miller et al. 1965), and a pulsed-current resistance meter (Shigo and Shigo 1974). Of these, the resistance meter seemed to hold the most potential for practical application in the present study. Tattar and Blanchard (1976) have reviewed the basic principles behind this method. The electrical resistance of wood decreases as it is progressively discolored and decayed by microorganisms. This is in part due to increases in the concentrations of mobile cations (potassium, calcium, manganese, and magnesium) in the decayed tissues. A compact field meter called a "Shigometer" (Northeast Electronics, Concord, N.H.) has been used to

detect decay and discoloration in living trees (Shigo and Shigo 1974; McGinnes and Shigo 1975; Shigo and Berry 1975; Piirto and Wilcox 1978) as well as utility poles (Shigo and Shigo 1974; Shigo et al. 1977; Shortle et al. 1978). However, even the most accurate methods for determining internal decay in trees will not effectively indicate decay in trees with recent infection where the column of decay has not reached the sampling point. Ectotrophic mycelial growth of P. weirii on the roots of Douglas-fir has been found far in advance of the main column of decay in the wood (Wallis and Reynolds 1965). Johnson et al. (1972) used the presence of surface mycelial growth on upper sections of roots and on root collars as an indication of root rot infection in young plantations of Douglas-fir.

B. Materials and Methods

Twenty sample trees around Phellinus weirii infection centers were examined for signs or symptoms of P. weirii infection. Diagnosis of infection included: visual examination of crowns for stress symptoms, root collar excavation and examination for surface mycelium or decayed roots, and detection of internal decay by use of a Shigometer and an increment borer. These different methods for diagnosis of infection were evaluated on the basis of accuracy and operational efficiency. Ten trees (including two controls located more than 20 meters from the rearest P. weirii kill) on each of two plots (WC4 and WC5) were examined in the following manner:

1. Crown symptoms evaluation. The crowns of study trees were examined (with the aid of binoculars) for stress symptoms

related to root rot (i.e. reduced terminal growth, chlorotic foliage, heavy cone crop, and general tufted appearance of the crown).

2. Root collar examination. The root collars of study trees were exposed by excavating the soil out to a 1.0 meter radius around the base of the stem and down to 0.5 meters below the ground line. All of the roots and the lower stem were carefully examined for P. weirii surface mycelium (or remnants of it) and for obviously decayed roots or root stubs.

3. Internal decay detection. The presence of internal P. weirii decay was checked in four places on each of the trees (two on roots and two on the lower bole). The following methods were used to determine internal decay at each sampling point:

a. Shigometer readings. At each sampling site a hole 2.4 mm in diameter and 15 cm deep was drilled using a portable power drill. The probe of the Shigometer was inserted up to 12 cm into the wood. The presence of stain and advanced decay was noted by interpreting the readings according to Shigo and Shigo (1974) and Shigo et al.(1977). Incipient decay or discoloration were indicated by a 50% decrease in the electrical resistance and advanced decay by a 75% decrease in the resistance. Readings were recorded at depth intervals of 1.5 cm.

b. Core inspection. Increment cores were extracted at the same locations as the previous test. Each core was examined for evidence of P. weirii as stain or decay

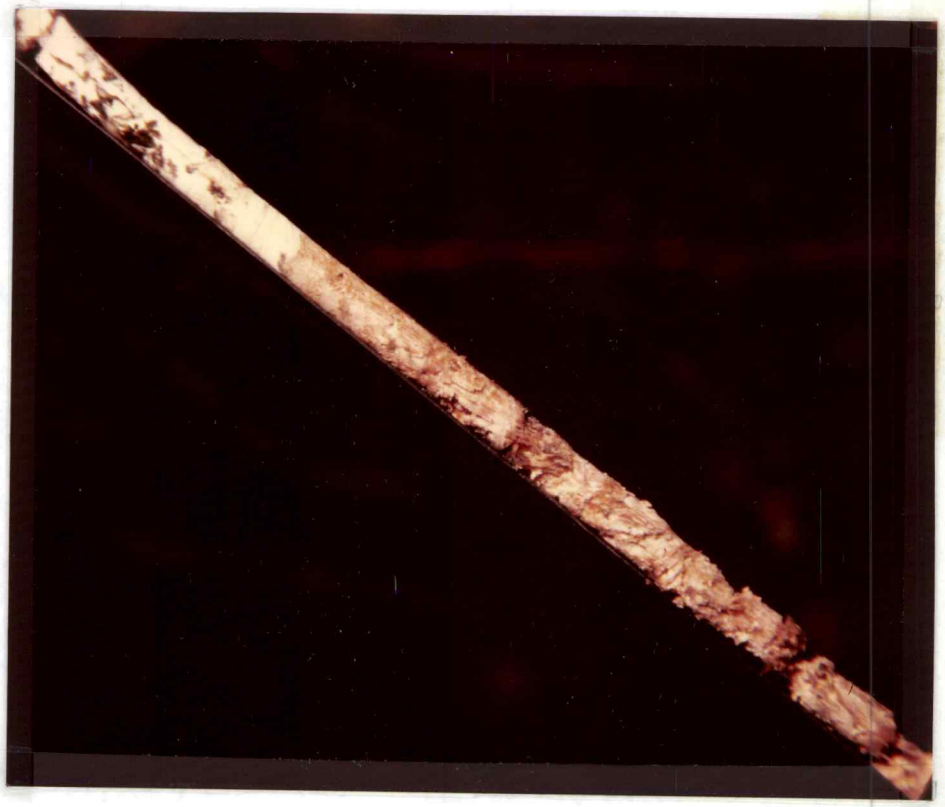


Figure 3. A core extracted by increment borer exhibiting stain and decay caused by Phellinus weirii.

(Figure 3). The exact location of the stain or decay was noted by recording the depth at which they occurred.

c. Core culturing. The increment cores were placed in plastic straws and transfered to the lab where they were sectioned in 5 cm segments, flamed, and plated out on 2% malt agar with 2 ppm benomyl added to suppress contaminants. Cores were examined at weekly intervals for presence of P. weirii mycelium.

C. Results and Discussion

The correlation between external signs or symptoms of P. weirii root rot and the presence of internal decay is presented in Table I. The best agreement between external and internal indicators was found in trees with stress crown symptoms. This was as expected since these trees are the most heavily infected. All trees with crown symptoms also exhibited external signs such as surface mycelium or decayed roots at the root collar. These trees, consequently, are easily diagnosed as infected by P. weirii. Detection of internal decay in trees with decayed roots at the root collar and no other signs or symptoms was more variable since internal infection was not uniformly distributed throughout the lower bole. This indicates that the entire root collar of suspected trees should be examined for decayed roots and surface mycelium, and that sampling for internal decay should be conducted at more than one point. Most of the trees with active surface mycelium on the roots at the root collar did not show any indications of internal decay. Therefore, if the root

Table I. Correlation between external signs or symptoms of *Phellinus weirii* root rot and the presence of internal decay.

External evidence	No. of trees	No. of trees with internal decay shown by:			No. of trees without decay[a]
		Shigometer	Core inspect.	Core culture	
Crown symptoms	4	4	4	3	0
Surface mycelium only	5	1	1	1	4
Decayed roots only	2	1	2	1	0
None	9	6[b]	2	2	3

[a]None of the three internal decay indicators showing decay.

[b]In four instances the Shigometer was the only indicator showing internal decay.

collars of suspected trees are not examined, many infected trees will be missed. Total excavation of the root systems would undoubtedly uncover even more infected trees, but would certainly be much more time consuming. When surface mycelium was found at the root collar, it extended up to the top 15 cm of mineral soil or higher. Deeper excavation (down to 0.5 m) did not reveal more infected trees. Some trees with no external signs or symptoms were found to have internal P. weirii decay. Hence, root collar excavation alone is insufficient for diagnosis of infected trees.

The correlations of the three internal decay indicators tested are presented in Tables II, III, and IV. The culturing of decay samples is the most certain indicator of P. weirii infection. It does , however, present several problems. In two instances samples of advanced decay were not transfered to the laboratory because of their deteriorated state. These samples were easily diagnosed as P. weirii decay in the field by noting the presence of setal hyphae and a pitted, laminated pattern of decay. Other similar samples which were cultured failed to yield P. weirii mycelium due to heavy contamination by bacteria and imperfect fungi. Visual and cultural identification of P. weirii decay from increment cores agreed in 85% of the samples (Table II). All of the cores which yielded P. weirii mycelium had visible stain or decay (Figure 3). However, 19% of the cores which had stain or decay failed to yield P. weirii in culture (Table II). Most instances of disagreement between visual examination and culturing were attributed to contamination problems in culturing and not to misidentification

Table II. Visual inspection of increment cores for diagnosis of *Phellinus weirii* decay as compared to culturing of the cores on malt agar.

Isolation of *P. weirii*	No. samples	Visual inspection of cores	
		Agree[a]	Disagree[a]
Positive	14	14 (100)[b]	0 (0)
Negative	64	52 (81)	12 (19)
Total	78	66 (85)	12 (15)

[a] With core culturing results.

[b] Percent of core culturing results.

Table III. Use of Shigometer for diagnosis of *Phellinus weirii* decay as compared to culturing of increment cores on malt agar.

Isolation of *P. weirii*	No. samples	Shigometer results	
		Agree [a]	Disagree [a]
Positive	14	11 (79) [b]	3 (21)
Negative	64	52 (81)	12 (19)
Total	78	63 (81)	15 (19)

[a] With core culturing results.

[b] Percent of core culturing results.

Table IV. Use of Shigometer for detecting incipient and advanced decay caused by Phellinus weirii as compared to visual inspection of increment cores.

Increment core results	No. observations	Shigometer results	
		Agree [a]	Disagree [a]
Sound wood	55	46 (84)	9 (16)
Incipient decay	23	13 (57)	10 (43)
Advanced decay	11	10 (91)	1 (9)
Total	89 [b]	69 (78)	20 (22)

[a] Within 2.5 cm with increment core results.

[b] Nine of the cores had observations of incipient and advanced decay.

of P. weirii stain or decay since other wood decay fungi were not isolated. In all of the instances of disagreement between visual inspection and culturing of cores (Table II) infection of the tree was verified by decay at other sampling points or by the presence of external signs or symptoms. Visual examination of increment cores was therefore an accurate way of determining P. weirii infection in standing trees surrounding disease centers.

The Shigometer was also found to be effective in indicating internal infection but was less so than visual examination of increment cores (Table III). The Shigometer failed to indicate the presence of decay at 21% of the sampling points which yielded P. weirii in culture. The overall performance of the Shigometer as compared to visual inspection of the core was good (Table IV). The agreement for samples with advanced decay was excellent (91%). Correct indication of sound wood was also good (84%), however, in 43% of the samples the Shigometer failed to indicate the presence of stained wood. This was a major drawback to using this instrument for diagnosing infection by P. weirii in standing trees. Some of the other problems encountered with the use of this instrument were similar to those discussed by Piirto and Wilcox (1978). Problems with the flexible probe and connections to the meter caused erratic readings. Rechargeable power drills lacked sufficient power for a full day of use on trees, hence, two drills had to be carried at all times. The length of time required for drilling the holes and obtaining repeatable readings from the Shigometer exceeded the time required for extracting and examining an increment core.

D. Conclusions

The results of this preliminary study helped to outline the procedures used for diagnosing infection by P. weirii in live standing trees described in the Materials and Methods of Chapter V. Since crown symptoms were not by themselves diagnostic of laminated root rot, a more thorough examination of the trees was deemed necessary. The most accurate and practical method for detection of internal decay was visual examination of increment cores for stain or decay characteristic of P. weirii. More than one core sample per tree was necessary due to variation in the internal decay column of an infected tree. Root collar excavation revealed infected trees by the presence of surface mycelium or decayed roots. Some of these trees showed no internal decay at the sampling points, hence, increment boring alone would not show many infected trees. Although total root excavation would uncover all infected trees, excavation down to 15 cm was more practical and revealed many infected trees. Diagnosis of infection in standing trees is best achieved by a combination of internal decay detection (with an increment borer at two or more points near the root collar) and root collar excavation (down to 15 cm) and examination for surface mycelium or decayed roots. The presence of either internal decay or external signs are diagnostic of P. weirii infection in standing trees.

V. COMPARISON OF PHELLINUS WEIRII DAMAGE IN TWO SUCCEEDING DOUGLAS-FIR STANDS.

A. Background

The main objective of this study was to investigate the carry-over of disease from residuals of an old-growth stand to the succeeding second-growth stand. The basis for comparison was the amount of damage due to Phellinus weirii found in each of the two stands. Damage assessments for root diseases of forest trees have been made in several different ways. The most easily obtained estimate is the proportion of trees affected by the disease. This method has been used for assessment of P. weirii damage in young Douglas-fir plantations (Johnson et al. 1972), 35 to 60 year-old second-growth stands (Bier and Buckland 1947; Molnar et al. 1961), and old-growth stands (Childs 1970). Estimation of the amount of volume in trees killed by P. weirii has also been used for damage assessment (Bier and Buckland 1947; Buckland et al.1954; Molnar et al. 1961; Childs 1970). However, as pointed out by Foster and Johnson (1963), these methods do not accurately estimate the importance of the disease to a stand. Their study of root rots and climatic disorders in young Douglas-fir plantations led them to conclude that damage "must be assessed in terms of the extent to which it reduces the density or modifies the pattern of the residual population". Several different sampling methods were used to determine the distribution of damage due to climatic disorders and root rots.

Although total numbers of damaged trees did not vary, trees affected by climatic disorders were found to be randomly dispersed, whereas trees affected by root rot were aggregated in centers. Later examinations of the study areas (Johnson et al. 1972) revealed an increase in the damage due to root rots and led to predictions of high losses by time of final harvest. Calculations were also made to estimate the size of openings at age 40 that would result from continued spread of the root rots.

Hansen (1978) found that an estimated 11% of the area of some mature Douglas-fir stands was unproductive due to P. weirii infection. He concluded that " an estimate of the area out of production because of infection is more realistic (than recording damage by the number of afflicted trees) in old stands but is meaningless in young stands before continuous infection centers have formed". Bloomberg (Pacific Forest Research Centre, Victoria, B.C. unpublished survey instructions) has devised a survey for estimating the amount of area out of production due to root rot diseases of forest trees. Williams and Leaphart (1978) used aerial photos to estimate area affected by root rots in northern Idaho. They claimed a 92% accuracy level for interpretation of disease centers from large scale color infrared photographs. Aerial photos were also used to determine spread of P. weirii in a high elevation mixed conifer stand (Nelson and Hartman 1975). However, this technique was not effective for locating disease centers in the dense Oregon Coast Range forests (Johnson and Wear 1975).

In order to judge the relative merit of the different damage assessment methods, damage due to P. weirii in the two stands surveyed

was assessed in terms of the reduction of density and volume by mortality, and the extent of infection in standing trees. Comparisons of damage were made on the basis of 100% surveys and maps of four plots approximately one hectare each. Current stand estimates were based on amounts and distribution of healthy, infected, and dead trees. Previous stand estimates were based on amounts and distribution of residuals (stumps, snags, and old down trees).

B. Materials and Methods

1. Mapping of plots. The study plots were mapped in the summer of 1977. The mapping was done using a staff compass and optical rangefinders. The first compass point was located near one of the plot corners and was referenced to a permanent point (see Appendix 1). Map points included all identifiable current stand P. weirii mortality (i.e. standing dead trees, snags, and windfalls), all current stand trees greater than 15 cm DBH (diameter at 1.3 m), and all identifiable previous stand residuals (i.e. stumps, snags, and windfalls). The distance, slope, and azimuth from the compass point to each of the map points within approximately 25-30 m (depending on visibiltiy) were measured and recorded. The distance measurements were made using optical rangefinders manufactured by Ranging Inc. (East Rochester, NY 14445). A Ranging 120 was used for all points closer than 15 m and a Ranging 620 for all points at 15 m or farther. The instruments were periodically calibrated with a tape measure to insure accuracy. The information recorded for each map point included: bearing, distance, and slope to compass point, species, and DBH. For current stand mortality the following was also recorded: cause of death, estimated

year of death, and condition at death (killed standing or windthrown). Previous stand residuals were classified as stumps, snags, or windfalls. Past P. weirii infection was determined as described later in this section. The presence of signs or symptoms of P. weirii infection in current stand trees was also recorded. When all visible trees and residuals around the initial compass point were mapped, a new compass point was selected and referenced to the previous one by recording distance, slope, and azimuth. This procedure was repeated until the total plot was mapped.

2. Computer plotting of maps. All of the recorded information for each map was keypunched onto computer cards. Maps of each plot were generated by computer. The plotting was done through the Oregon State Open Shop Operating System (OS3) on a Control Data 3300 computer. The program used was a modified version of a COMPLOT plotting program used by E.M. Hansen (Oregon State University, Corvallis, Oregon personal communication). COMPLOT is a set of subroutines designed for easy programming of graphic applications by expanding operator instructions to include details needed by the plotting device (Fuhrer 1977). The program operates by converting distance and azimuth measurements to x- and y-coordinates and thereby plots the location of each map point and compass point. Different symbols were assigned to each of the map point categories. The actual plotting was done by a Gerber 1022 flatbed off-line plotter. A listing of the program used for this study is located in Appendix 2. Three different maps were generated for each of the plots: a whole-plot map of all map points from the current and previous

stands, a split-plot map of only the previous stand map points, and a split-plot map of only the current stand map points. The scale used for all of these maps was 1:200.

3. Examination of current stand trees for evidence of P. weirii infection. Mortality in the current stand caused by P. weirii was identified on the basis of fungus and decay characteristics. Living trees on the perimeter of disease centers were examined for any signs or symptoms of P. weirii infection. The crowns were examined for any obvious stress symptoms. The root collars were exposed by excavating the soil out to 0.5 m around the base of the trees and down to 15 cm below the ground line. The roots and lower bole were then carefully examined for evidence of P. weirii surface mycelium or decayed roots. If no signs were found, two of the roots on each of the trees were bored with an increment borer and the cores examined for P. weirii stain or advanced decay. If the tree had obvious crown symptoms, then the lower bole was bored prior to excavation. The presence of either internal decay, surface mycelium or decayed roots was considered positive evidence of P. weirii infection. This procedure was repeated for all trees on the perimeter of disease centers and all adjacent trees until healthy trees were encountered.

4. Examination of previous stand residuals for evidence of past P. weirii infection. Previous stand residuals were examined for signs of past or present P. weirii activity. The following categories were developed to classify the previous stand residuals by certainty of infection:

positive P. weirii infection -

1) live P. weirii mycelium characterized by setal hyphae,
2) characteristic pitted, laminated decay with old setal hyphae and dark brown zone lines,
3) characteristic pitted, laminated decay without setal hyphae but with dark brown zone lines,

questionable P. weirii infection -

4) characteristic pitted, laminated decay without setal hyphae or dark brown zone lines,
5) characteristic pitted, laminated decay colonized by brown rotting fungi,
6) characteristic pitted, laminated decay colonized by other white rotting fungi,
7) no visible signs of P. weirii but bole and roots hollowed out (very old snags),
8) no visible signs of P. weirii but windthrown with root ball (very old windfall),

no P. weirii infection -

9) no visible above- or below-ground signs of past P. weirii decay.

This system is an adaptation of Hansen's diagnosis of past infection by P. weirii in old-growth Douglas-fir stumps (Hansen 1979c).

If no positive signs of infection were found in the above-ground portions of the residuals, at least three roots were excavated out to one meter from the main bole and down to 0.5 meters below ground line, and the roots were examined for the above indicators. Figure 4 shows typical positive P. weirii decay in old stumps.

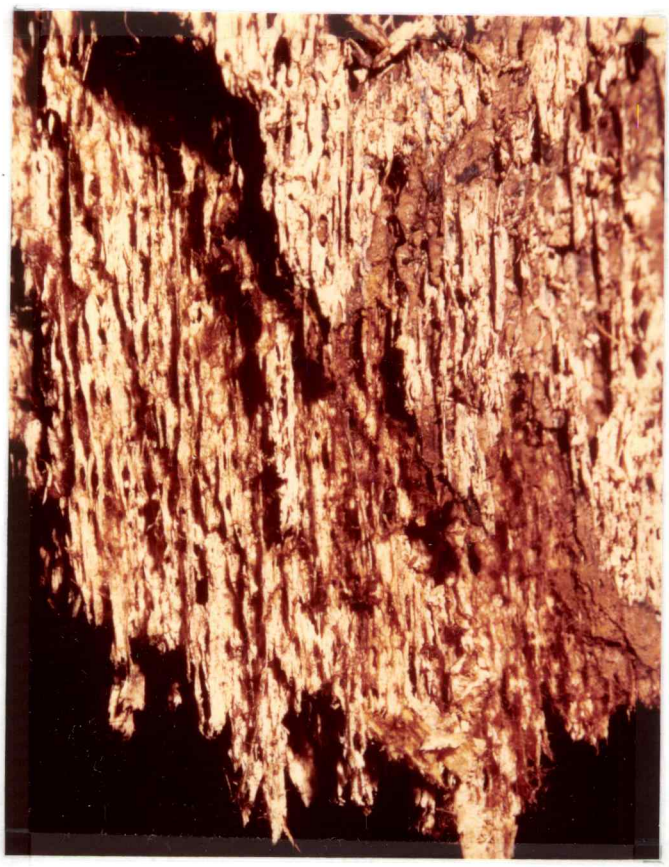


Figure 4. Example of "positive" Phellinus weirii decay in previous stand stump.

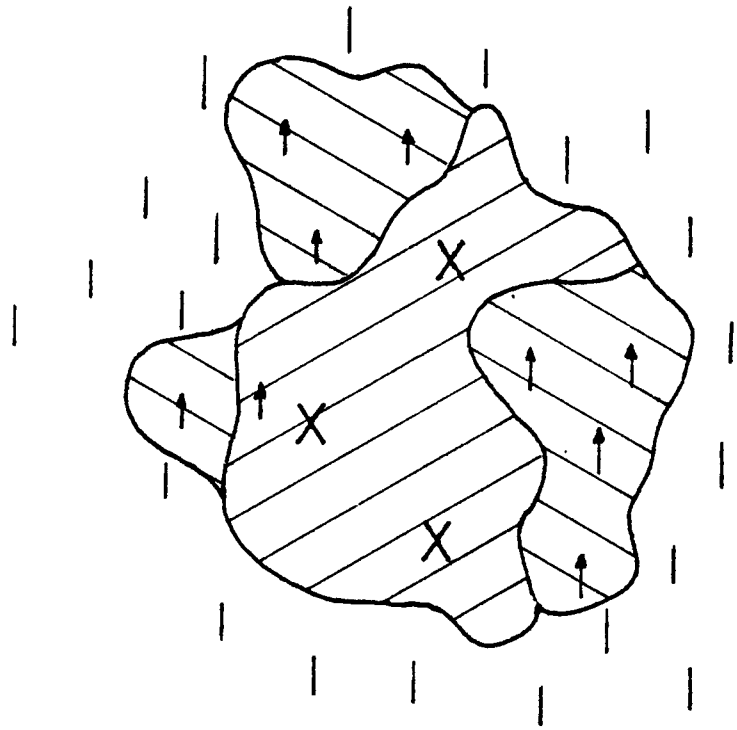
5. Calculation of Expected Stand Density and Average Tree Area. Stand density approximations were made from the maps of the study plots. In order to determine the average stand density for a healthy stand on the site, density plots were located outside areas of disease-created openings. For the current stand, the numbers of trees on ten 100 m^2 circular plots were averaged for each study plot, and for the previous stand, the number of residuals on five 200 m^2 circular plots were averaged for each study plot. From these figures the average area occupied by each tree was calculated for each study plot. This average tree area represents an approximation of the average crown width. The ratio of average crown width to average lateral root spread for Douglas-fir has been found to be nearly one (Smith 1964), hence, the radius of the circular crown area represents an approximation of the average lateral root length.

6. Delineation of Area of Concentrated Mortality. The amount of damage caused by P. weirii root rot in the two succeeding stands was estimated in terms of the proportion of the total area infested with P. weirii. No directly applicable system of accurately delineating diseased area was found in the root rot literature, so guidelines and definitions were developed for the current and previous stands. All area measurements were made on 1:200 scale maps using a polar compensating planimeter. Reduced maps of all the disease areas delineated for each of the four plots are found in Appendix 3.

The first measurement of diseased area was an attempt to determine the amount of area in P. weirii-caused openings in the two stands. The "Area of Concentrated Mortality" corresponds to the area out of

production of Douglas-fir and other susceptible species due to P. weirii mortality. Included in this area are also those living, infected trees that fall within the average tree area of a P. weirii killed tree. The approximate boundaries of this area for the current stand were first drawn on the maps in the field by delimiting the non-stocked area surrounding P. weirii mortality. Non-stocked area included all of the open area from a P. weirii kill out to the crown of the next standing tree. The average tree area was used as an average crown area. All standing infected trees within the average tree area of a P. weirii kill were included in the non-stocked area. If a healthy tree was within the tree area of a P. weirii kill the boundary was drawn mid-way between the two. These same guidelines were used to reconstruct the Area of Concentrated Mortality in the previous stand at the time of its harvest. Stumps were equivalent to standing trees and snags and windthrown trees were equivalent to mortality. Residuals with questionable indicators of past P. weirii infection were included if they were adjacent to positive ones. An example of the delineation of the Area of Concentrated Mortality in the current stand is presented in Figure 5.

7. Delineation of Area of Standing Infection. The "Area of Standing Infection" consists of the combined tree areas of live, standing trees (outside the Area of Concentrated Mortality) that are infected with P. weirii. Infection was determined as described earlier by crown examination, root collar examination, and increment boring. If a healthy tree was found within the tree area of an



Key:

| Healthy tree

↑ P.weirii infected tree

X P.weirii killed tree

Area of Concentrated Mortality

Area of Standing Infection

⟷ Average rooting lenght of trees.

Figure 5. Delineation of diseased areas in the current stand.

infected tree the boundary was drawn mid-way between the two. This area was also reconstructed on the previous stand maps by combining the tree areas of P. weirii infected stumps (outside the Area of Concentrated Mortality). Figure 5 also shows an example of the delineation of the Area of Standing Infection for the current stand.

8. Delineation of Area of Potential Inoculum. In order to get a better idea of the actual distribution and amounts of inoculum sources from the previous stand, a third area was also outlined for the previous stand on each plot. The "Area of Potential Inoculum" represents the combined tree areas of all potential sources of P. weirii inoculum left over after the harvest of the previous stand. These inoculum sources include all previous stand residuals (stumps, snags, and old down trees) that contained "positive" signs of P. weirii decay. In addition to these, all residuals with "questionable" P. weirii decay that were adjacent to positive ones were also included.

C. RESULTS

1. Expected Stand Densities and Average Tree Areas for the two succeeding stands. The Expected Stand Densities for the current stand ranged from 860 trees/ha (plot WC5) to 1450 trees/ha (WC2) (Table V). The previous stand Expected Stand Densities were lower ranging from 300 trees/ ha (WC3) to 470 trees/ha (WC5) (Table V). The Average Tree Areas varied in accordance with the Expected Stand Densities. Average Lateral Root Length was larger for the previous stand trees

Table V. Expected Stand Density (trees/ha), Average Tree Area (m^2/ ha), and Average Lateral Root Length (m) for the current and previous stands on each study plot.

Current Stand:

Plot	Expected Stand Density[a]	Av. Tree Area[c]	Av. Lateral Root Length[d]
WC2	1450	6.9	1.5
WC3	873	11.5	1.9
WC4	940	10.6	1.8
WC5	860	11.6	1.9
Average	1031	10.2	1.8

Previous Stand:

Plot	Expected Stand Density[b]	Av. Tree Area[c]	Av. Lateral Root Length
WC2	370	27.0	2.9
WC3	300	33.3	3.3
WC4	360	27.8	3.0
WC5	470	21.3	2.6
Average	375	27.4	3.0

[a] based on ten 100 m^2 plots in the healthy areas of each study plot.
[b] based on five 200 m^2 plots in the healthy areas of each study plot.
[c] computed as follows: 10,000 m^2 / trees per ha = av. area per tree.
[d] computed as follows: av. root length = $\sqrt{}$(av. area per tree/π) .

than current stand trees (Table V).

2. Comparison of damage by number of trees affected. The first estimator of P. weirii root rot damage to the two succeeding stands was the proportion of trees infected or killed by the pathogen. A comparison of the percentage of trees affected by P. weirii in the current and previous stands indicates that the proportions of trees infected and killed by the pathogen were considerably higher in the previous stand than in the existing 60 year-old stand (Table VI). Half of the total current stand trees (alive and dead) were healthy, but only one fifth of the previous stand trees were healthy at harvest. In both stands one third of the total trees were standing and infected. Mortality due to P. weirii was higher in the previous stand (35.7%) than in the current stand (17.3%) (Table VI). A more meaningful comparison is based on the reduction of the Expected Stand Density by the disease (Table VII). This corrected estimate of loss due to P. weirii mortality compensates for those trees killed in the earlier stages of stand establishment that have since decayed beyond recognition. The reduction of stand density by P. weirii was lower in the current stand (41.7%) than in the previous stand (75.5%) (Table VII). Of the remaining standing trees, 35.1% and 60.9% were found to be infected in the current and previous stands respectively (Table VIII).

3. Comparison of damage by amount of basal area affected. The second estimator of P. weirii damage was the proportion of the total basal area affected by the disease. Basal areas were calculated for the following groupings of susceptible trees: healthy trees, infected trees, P. weirii mortality, and other mortality. The amounts and

Table VI. Comparison of proportions of total susceptible trees (live and dead) affected by laminated root rot in the current and previous stands.

Plot	Total susceptible[a] trees/ha	Standing healthy trees/ha	Standing infected[b] trees/ha	*P. weirii* killed trees/ha	Dead by other causes trees/ha
			Current Stand		
WC2	1318	696(52.8)[c]	322(24.5)	223(16.9)	77(5.8)
WC3	590	281(47.6)	171(29.0)	80(13.6)	58(9.8)
WC4	565	312(55.2)	145(25.7)	86(15.2)	22(3.9)
WC5	655	271(41.4)	204(31.1)	151(23.1)	29(4.4)
Average	782	390(49.8)	211(26.9)	135(17.3)	47(6.0)
			Previous Stand		
WC2	157	20(12.7)	27(17.2)	97(61.8)	13(8.3)
WC3	144	26(18.1)	52(36.1)	48(33.3)	18(12.5)
WC4	185	54(29.2)	64(34.6)	42(22.7)	25(13.5)
WC5	254	43(16.9)	82(32.3)	76(29.9)	53(20.9)
Average	185	36(19.4)	56(30.3)	66(35.7)	27(14.6)

[a]Susceptible to *P. weirii* (Douglas-fir, western hemlock, noble fir).

[b]Infected by *P. weirii*.

[c]Percent of total susceptible trees.

Table VII. Reduction of stand density by Phellinus weirii mortality.

Plot	Expected Stand Density trees/ha[a]	Actual Stand Density trees/ha[b]	P. weirii Mortality[c] trees/ha
----------Current Stand----------			
WC2	1450	1018	432(29.8)[d]
WC3	873	452	421(48.2)
WC4	940	457	483(51.4)
WC5	860	476	384(44.7)
Average	1031	601	430(41.7)
----------Previous Stand----------			
WC2	370	47	323(87.3)
WC3	300	78	222(74.0)
WC4	360	118	242(67.2)
WC5	470	125	345(73.4)
Average	375	92	283(75.5)

[a] Extrapolated from healthy areas of plot.

[b] Actual number of live trees per hectare.

[c] Difference between Expected Stand Density and Actual Stand Density.

[d] Percent of Expected Stand Density.

Table VIII. Proportion of Actual Stand Density infected by *Phellinus weirii*.

Plot	Actual Stand Density trees/ha[a]	Infected by *P. weirii* trees/ha[b]
	Current Stand	
WC2	1018	322(31.7)[c]
WC3	452	171(37.8)
WC4	457	145(31.7)
WC5	476	205(43.1)
Average	601	211(35.1)
	Previous Stand	
WC2	47	27(57.4)
WC3	78	52(66.7)
WC4	118	64(54.2)
WC5	125	82(65.6)
Average	92	56(60.9)

[a] Actual number of live trees per hectare.

[b] Live trees infected by *P. weirii*.

[c] Percent of Actual Stand Density.

relative proportions of these groupings are listed in Table IX. The amount of damage to the two stands estimated by basal area comparisons (Table IX) is for the most part equivalent to the damage estimated by number of trees affected (Table VI). However, the basal area of trees killed by P. weirii is low (10.9% for the current stand and 35.3% for the previous stand) compared to the numbers of trees killed (17.3% for the current stand and 35.7% for the previous stand). To compensate for trees killed in the early stages of stand development an Expected Basal Area was calculated for both stands on each plot by extrapolating basal areas found on healthy areas of the plot to the entire plot area. Loss to P. weirii mortality was calculated by subtracting the total basal area of standing trees from the total expected basal area for a healthy stand on the site. This corrected loss in basal area was also much higher in the previous stand (56.6%) than in the current stand (22.5%) (Table X). Of the remaining basal areas, 37.7% and 64.8% were infected in the current and previous stands respectively (Table XI).

4. Comparison of damage on the basis of amount of area affected. The third estimator of P. weirii damage to the two stands was the area infested with the fungus. Area estimates generally agreed with the other two damage estimators by indicating a higher amount of damage in the previous stand than in the current stand. Area of Concentrated Mortality occupied 68.4% of the previous stand and 36.3% of the current stand (Table XII). Differences in the current stand Area of Concentrated Mortality among the four plots were not related to differences in previous

Table IX. Basal Area (B.A. m^2/ha) lost to *P. weirii* as related to total B.A. of susceptible trees (live and dead).

Plot	Total B.A.	B.A. of healthy trees	B.A. of standing infected trees	B.A. of *P. weirii* killed trees	B.A. of other mortality
			Current Stand		
WC2	66.69	38.62 (57.9)[a]	19.16 (28.7)	5.92 (8.9)	2.99 (4.5)
WC3	62.00	32.83 (52.9)	21.10 (34.0)	6.11 (9.9)	1.96 (3.2)
WC4	47.02	27.72 (58.9)	14.10 (30.0)	4.54 (9.7)	0.66 (1.4)
WC5	58.81	27.03 (46.0)	21.94 (37.3)	8.85 (15.0)	0.99 (1.7)
Average	58.63	31.55 (53.8)	19.08 (32.5)	6.36 (10.9)	1.65 (2.8)
			Previous Stand		
WC2	121.72	20.23 (16.6)	28.25 (23.2)	67.74 (55.7)	5.50 (4.5)
WC3	120.00	20.46 (17.0)	53.15 (44.3)	38.82 (32.4)	7.57 (6.3)
WC4	100.68	32.04 (31.8)	43.79 (43.5)	16.56 (16.5)	8.29 (8.2)
WC5	94.20	15.59 (16.5)	37.26 (39.6)	31.01 (32.9)	10.34 (11.0)
Average	109.15	22.08 (20.2)	40.61 (37.2)	38.53 (35.3)	7.93 (7.3)

[a]Percent of Total B.A. of susceptible trees (Douglas-fir, western hemlock, noble fir).

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

Plot	Expected B.A.[a]	Actual B.A.[b]	*P. weirii* mortality B.A.[c]
Current Stand			
WC2	67.15	57.78	9.37(14.0)[d]
WC3	69.55	53.93	15.62(22.5)
WC4	55.55	41.82	13.73(24.7)
WC5	68.95	48.97	19.98(29.0)
Average	65.30	50.63	14.67(22.5)
Previous Stand			
WC2	183.93	48.48	135.45(73.6)
WC3	181.01	73.60	107.41(59.3)
WC4	92.07	75.83	16.24(17.6)
WC5	120.85	52.85	68.00(56.3)
Average	144.47	62.69	81.78(56.6)

[a] Extrapolated from healthy areas of plot.

[b] Actual B.A. of live trees.

[c] Difference between Expected B.A. and Actual B.A.

[d] Percent of Expected B.A.

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

Plot	Actual B.A.[a]	Infected by P. weirii B.A.[b]
	Current Stand	
WC2	57.78	19.16(33.2)[c]
WC3	53.93	21.10(39.1)
WC4	41.82	14.10(33.7)
WC5	48.97	21.94(44.8)
Average	50.62	19.08(37.7)
	Previous Stand	
WC2	48.48	28.25(58.3)
WC3	73.60	53.15(72.2)
WC4	75.83	43.79(57.7)
WC5	52.85	37.26(70.5)
Average	62.69	40.61(64.8)

[a]Actual B.A. of live trees.

[b]B.A. of live trees infected by P. weirii.

[c]Percent of Actual B.A.

amounts. Plot WC2, for example, had the highest percentage for the previous stand (84.2%) but the lowest for the current stand (26.9%) (Table XII). Direct comparisons of previous and current stand values of Area of Concentrated Mortality (Table XII) did not indicate any trends or patterns. Hence, the size of disease openings in the previous stand did not accurately predict the size of disease openings in the succeeding stand.

Table XII. Comparison of Area of Concentrated Mortality (A.C.M.) in the current and previous stands.

Plot	Total Plot Area in m^2	A.C.M. in Current Stand (m^2/ha)	A.C.M. in Previous Stand (m^2/ha)
WC2	13631.8	2689.4 (26.9)[a]	8417.8 (84.2)
WC3	13615.6	3717.4 (37.2)	7046.3 (70.5)
WC4	12818.3	3840.7 (38.4)	4931.0 (49.3)
WC5	11032.7	4280.9 (42.8)	6961.1 (69.6)
Average		3632.1 (36.3)	6839.1 (68.4)

[a]Percent of Total Plot Area.

As mentioned earlier, the Area of Concentrated Mortality did contain some live infected trees. Most of these trees were found to be heavily infected with either crown symptoms or totally decayed roots at the root collar. These trees tended to be in the interior

of the Area of Concentrated Mortality rather than on the perimeter. However, this inclusion of some live trees prevented the designation of this area as an "area out of production" of susceptible species. The designation of this area as the Area of Concentrated Mortality was based on the finding that an average of 74% of the susceptible current stand trees within this area had been killed by the fungus. In the previous stand, 88.7% of the residuals within the Area of Concentrated Mortality had been killed by P. weirii prior to harvest. No healthy trees were included in this area.

The Area of Standing Infection was very similar for the two stands on all four plots. The Area of Standing Infection covered 14.1% of the previous stand and 15.2% of the current stand (Table XIII). The average percentage for the previous stand was affected by a low figure (4.8%) for plot WC2. The map of the previous stand disease areas for this plot (Appendix 3) revealed that most of the plot was covered by a large opening. Infected stumps were found on the perimeter of this center but they were beyond the plot boundaries.

The Total Area of Infection was obtained by adding the Area of Standing Infection to the Area of Concentrated Mortality. The average Total Area of Infection was higher for the previous stand (82.5%) than for the current stand (51.6%) (Table XIV). Differences among the four plots in the total amount of area infested with P. weirii did not correlate with differences in the current stand amounts. Hence, the total amount of diseased area in the previous stand did not accurately predict the total amount of diseased area in the current stand.

Table XIII. Comparison of Area of Standing Infection (A.S.I.) in the current and previous stands.

Plot	A.S.I. in Current Stand (m^2/ha)	A.S.I. in Previous Stand (m^2/ha)
WC2	1557.2 (15.6)[a]	479.4 (4.8)[a]
WC3	1567.4 (15.6)	1826.3 (18.2)
WC4	1170.6 (11.7)	1588.2 (15.9)
WC5	1797.4 (18.0)	1753.4 (17.5)
Average	1523.2 (15.2)	1411.8 (14.1)

[a]Percent of Total Plot Area.

Table XIV. Comparison of Total Area of Infection (T.A.I.) in the current and previous stands.

Plot	T.A.I. in Current Stand (m^2/ha)	T.A.I. in Previous Stand (m^2/ha)
	4246.6 (42.5)[a]	8897.2 (89.0)[a]
WC3	5284.8 (52.8)	8872.6 (88.7)
WC4	5011.3 (50.1)	6519.2 (65.2)
WC5	6078.3 (60.8)	8714.5 (87.1)
Average	5155.3 (51.6)	8250.9 (82.5)

[a]Percent of Total Plot Area.

The amount of area covered by P. weirii inoculum sources from the previous stand was represented by the Area of Potential Inoculum. An average of 26.6% of the area was covered by these inoculum sources (Table XV). The highest value was for plot WC5 (28.9%) and the lowest for WC4 (24.3%). The relative proportions of the different categories of previous stand residuals included in the Area of Potential Inoculum are listed in Table XVI. Stumps with positive P. weirii decay were the most prevalent (37.3%). Plot WC2, however, had more positive snags (47.0%) than stumps (19.1%). This was the plot with the lowest Total Area of Infection in the current stand (Table XIV), which suggests that stumps were more effective than snags as inoculum sources. The relative effectiveness of the various previous stand inoculum sources was studied and is reported in Chapter VI.

Table XV. Area of Potential Inoculum (A.P.I.) from the previous stand.

Plot	A_2P.I. (m^2/ha)
WC2	2574.0 (25.7)[a]
WC3	2749.7 (27.5)
WC4	2433.2 (24.3)
WC5	2888.1 (28.9)
Average	2661.3 (26.6)

[a]Percent of Total Plot Area.

Table XVI. Frequency of residual categories within the Area of Potential Inoculum.

Plot	Total residuals within A.P.I.	Stumps		Snags		Down trees	
		pos.[a]	quest.[b]	pos.	quest.	pos.	quest.
WC2	162	31 (19.1)[c]	4 (2.5)[c]	76 (47.0)	31 (19.1)	13 (8.0)	7 (4.3)
WC3	132	53 (40.1)	17 (12.9)	27 (20.5)	26 (19.7)	7 (5.3)	2 (1.5)
WC4	131	57 (43.5)	22 (16.8)	15 (11.5)	21 (16.0)	5 (3.8)	11 (8.4)
WC5	172	80 (46.6)	9 (5.2)	35 (20.3)	23 (13.4)	14 (8.1)	11 (6.4)

[a]Residuals which had "positive" evidence of past P. weirii infection; see page 38.

[b]Residuals which had "questionable" evidence of past P. weirii infection; see page 38.

[c]Percent of total residuals within A.P.I.

D. Discussion.

Root collar excavations of trees on the perimeter of *P. weirii* infection centers revealed the presence of surface mycelium of the pathogen on many of the roots. The appearance of this ectotrophic mycelial growth varied considerably depending on the activity of the fungus. Trees close to recent *P. weirii* mortality contained little or no active surface mycelium. Careful examination of the bark disclosed remnants of surface mycelial activity such as small isolated pockets of live mycelium covered by a crust-like layer or just remnants of the crust layer with numerous brown setal hyphae located in fissures in the bark. Increment boring of these trees invariably revealed considerable internal decay characteristic of *P. weirii*. Many of these trees showed no obvious crown symptoms. Trees further removed from *P. weirii* kills contained more recent surface mycelial growth. The fungus on roots of these trees was either active or just recently desiccated. Trees with active surface mycelial growth on the lateral roots or at the root collar seldom had internal decay at those points.

These observations tend to support the hypothesis for spread of *P. weirii* within a stand put forth by previous researchers (Buckland *et al.* 1954; Wallis and Reynolds 1965). Their studies suggest that the primary mode of spread for *P. weirii* after initial infection is by surface mycelial growth on roots of living infected trees and subsequent spread onto roots of adjacent healthy trees. Wallis and Reynolds (1962) found that surface mycelial growth occured well in advance of the decay column in the heartwood of the roots.

Comparisons of damage due to P. weirii to the two stands revealed that the disease was more destructive in the previous stand (at time of its harvest) than in the current 60 year-old stand. Damage estimations based on the number of trees or amount of basal area affected by the disease tended to underestimate the extent of damage. Childs (1970) discussed the problems associated with these methods of disease assessment:

> "Percentage of trees killed is only roughly indicative of infection severity, since remains of small killed trees soon disintegrate or are hidden by the litter; this percentage is also distorted by irregularities in stand density and normal decrease in numbers of trees as stands become older. Basal area percentage underestimates impact of the disease, since basal areas of trees killed several years ago do not adequately represent , in proprotion to present basal areas of surviving trees, their potential contribution to yield."

To circumvent these problems, calculations were made to determine the reduction of Expected Stand Density and Expected Basal Area by P. weirii mortality. These estimates represent the total amount of loss due to P. weirii mortality from stand establishment to the present for the current stand and to time of harvest for the previous stand by comparing the expected density and volume for a healthy stand with the actual density and volume found.

Based on density and volume comparisons alone, one can conclude that damage due to P. weirii in the current stand has not yet reached the proportions of damage in the preceding old-growth stand. However, if mortality continues at a steady rate, the losses accrued in the current stand by an age comparable to that of the preceding stand will undoubtedly be higher than those

found in the preceding stand. Average annual damage rates were calculated for both stands and will be discussed in Chapter VI.

Although both density and volume comparisons were found to be effective in comparing total losses in the two succeeding stands, they do not give any indications of the spatial distribution and correlation of disease from one stand to the next. These can only be assessed by determining the areas affected by the disease in the two stands and how they overlap. The amount of area left out of Douglas-fir production by P. weirii activity as well as the extent of infection of standing trees in the current stand seemed initially to be relatively high for a 60 year-old stand. However, when the infection centers of the previous old-growth stand were reconstructed, it became evident that the level of infection in that stand at the time of its harvest was much greater than is currently found in the succeeding stand. On the average, 51.6% of the current stand area and 82.5% of the previous stand area were infected by P. weirii (Table XIV). This difference in the total areas affected is attributable, for the most part, to greater amounts of Area of Concentrated Mortality in the previous stand (68.4%) than in the current stand (36.3%) (Table XII). The differences in the amounts of Area of Standing Infection were relatively small (Table XIII). A notable exception was the low amount of Area of Standing Infection in the previous stand on plot WC2 . This plot had the highest Total Area of Infection in the previous stand (89.0%) and the lowest in the current stand (42.5%) (Table XIV). Direct comparisons of

this type for each plot revealed that the Total Area of Infection (Table XIV) for a stand does not effectively predict the amount of disease that can be expected in the succeeding stand.

Childs (1970) speculated that: "present differences in infection may be correlated with past differences in infection or in type". Present differences in infection were related to past differences in the type of infection. The types of infection found in the previous stand ranged from obvious centers with few standing trees (plot WC2) to scattered infection with many standing infected trees (plots WC4 and WC5). The lowest amount of diseased area in the current stand was found on plot WC2, where the infection in the previous stand resulted in a large open center covering most of the plot. The Area of Potential Inoculum for WC2 was the lowest of all plots (Table XV). In addition to this, the majority of the previous stand inoculum sources on this plot were old snags and not stumps, as on the other plots (Table XVI). Differences in total infection for the other plots can be attributed to differences in the Area of Potential Inoculum and the composition of the previous stand inoculum sources (Tables XV and XVI). The greater the number and distribution of previous stand stumps infected with P. weirii, the higher the damage level in the succeeding stand.

E. Conclusions.

The results of this study indicated the following trends of disease development in the two succeeding stands studied:

Mortality due to Phellinus weirii was higher in the previous stand than in the current stand. The reduction of Expected Stand Density by P. weirii mortality was also greater in the previous stand. One third of the total trees in both stands were infected by the pathogen. Damage assessments utilizing basal area comparisons revealed similar trends. The proportion of the total basal area killed was higher in the previous stand than in the current stand. One third of the total basal area in both stands was infected by P. weirii. The reduction of Expected Basal Area was again greater in the previous stand than in the current stand.

Estimates of area affected by the disease agreed with the other two damage estimators by indicating a higher amount of damage in the previous stand than in the current stand. The higher Total Area of Infection in the previous stand was attributable to a higher Area of Concentrated Mortality since the Area of Standing Infection was the same for both stands. The previous stand Total Area of Infection did not effectively predict current stand Total Area of Infection.

Damage in the current stand was related to the type and distribution of previous stand inoculum sources. The highest current stand damage was found on the plot with the largest Area of Potential Inoculum and the highest number of infected previous stand stumps.

VI. SPREAD OF PHELLINUS WEIRII IN TWO SUCCEEDING DOUGLAS-FIR STANDS

A. Background and Methods.

1. Association of disease areas in the current and previous stands. Although there has been speculation in the literature on the potential for increasing damage due to Phellinus weirii in stands regenerating on previously diseased sites (Childs 1970; Shea 1958) no attempts have been made to quantify the spread of the disease from one stand to the next. The association of disease in the two succeeding stands examined during this study was investigated by measuring the amount of overlap of diseased areas (see Appendix 3 for maps) in the current and previous stands for each study plot. Area measurements were made using a compensating polar planimeter on 1:200 scale maps of the plots. The areas measured were: currently healthy area overlying previous stand Area of Infection, currently healthy area overlying previous stand Area of Potential Inoculum, current Area of Infection lying beyond previous Area of Infection, and current Area of Infection lying beyond previous Area of Potential Inoculum.

2. Effectiveness of different sources of P. weirii inoculum from the previous stand. It has been shown that previous stand stumps may act as inoculum sources of Phellinus weirii for a succeeding generation of trees (Buckland et al. 1954; Wallis and Reynolds 1965), and that P. weirii can survive saprophytically in these stumps for extended periods of time (Hansen 1979c). However, no attempts have been made to quantify the proportion of infected stumps (and other previous

stand residuals) that actually transmit disease to the succeeding stand. A comparison of the effectiveness of previous stand inoculum sources was made during this study by recording the numbers of residual stumps, snags, and down trees that were associated with root rot in the current stand. Effective carry-over of disease was indicated by the presence of disease in the current stand within the Average Tree Area of the inoculum source. The reliability of this technique was checked by recording the number of uninfected residuals with associated current stand disease.

3. Vegetative incompatibility of P. weirii isolates. Development of laminated root rot in Douglas-fir stands has been studied by testing P.weirii isolates for vegetative incompatibility (Childs 1963,1970). Under conditions for rapid growth of the fungus in culture, "all isolates of a clone are compatible in culture with all other isolates of that same clone and antagonistic to all other clones" (Childs 1970). Incompatibility manifests itself as a narrow dark zone in the agar between the two isolates. This potentially valuable method should be used with some caution. From his study of sexual and vegetative incompatibility in P. weirii, Hansen (1979a) concluded that "formation of lines of demarcation between two isolates of P. weirii seems a very sensitive indication that they differ .... lack of a line of demarcation after pairing is less definitive". Nuclear condition of the isolates must be known to accurately interpret results of the pairings since pairing homokaryotic isolates was found to give erratic reactions that could be confused for "clonal reactions". The nuclear condition of an isolate cannot be easily ascertained due to the lack of clamp

connection formation and irregular conditions in the homo- and heterokaryons (Hansen 1979b). However, all field isolates examined to date have been found to be heterokaryotic (Hansen 1979b).

A limited analysis of the vegetative incompatibility of P. weirii isolates was carried out for each plot to investigate the development of the disease. A minimum of five isolates per plot were collected from current stand mortality. These isolates were taken from trees located near the middle and the corners of the plots to assure coverage of the diseased area. Whenever possible isolates were collected from current stand mortality with P. weirii fruiting bodies to reduce the possibility of isolating homokaryons. The compatibility test was patterned after Child's (1963) "cross-plating" technique. Two isolates to be tested were inoculated about 2 cm apart on a petri plate containing 2% malt agar with 2 mg/liter benomyl. Colonies were allowed to grow out and were examined for formation of "lines of demarcation". Isolates from each plot were plated in all possible combinations. Whenever possible isolates were also taken from previous stand residuals and cross-plated with both previous and current stand isolates.

4. Probability of infection with increasing distance from inoculum source. The effect of distance from inoculum source on the probability of infection has received only limited attention in the literature. Childs (1963) reported that following a clearcut in a stand of young Douglas-fir sawtimber, "decay caused by P. weirii was visible on the fresh surfaces of 62% of the stumps within 30 ft. of trees killed by the disease, on 20% of the stumps at 30-50 ft., and on 4% of those beyond 50 ft.". Hadfield and Johnson (1977) reported that trees within

15 feet of P. weirii caused mortality are usually infected and that "as the distance from the closest tree increases, the percentage of infected trees decreases". This relationship was investigated during our study by measuring distance on 1:200 scale plot maps from randomly selected living trees (both healthy and infected) to nearest P. weirii inoculum by category. Twenty sample trees per plot were selected using random numbers and recorded as healthy or infected. Positive inoculum sources were tallied as follows: previous stand stumps, snags, and down trees, and current stand killed trees. The probability of infection was calculated as the proportion of the total number of trees that were infected by P. weirii in the following distance intervals: 0 to 3 m, 3 to 6 m, 6 to 9 m, 9 to 12 m, 12 to 15 m, and beyond 15 m.

5. Comparison of P. weirii damage at corresponding ages in the two succeeding stands. Several researchers have speculated on the potential for increasing damage due to P. weirii when previously infected sites are regenerated to susceptible species. Shea (1958) stated that P. weirii "can be expected to cause greater damage in second-growth timber than in timber at corresponding ages during the first rotation". Similar predictions have been made by Childs (1963,1970) and Hadfield and Johnson (1977). No attempts have been made to compare damage in two succeeding stands on the same site.

In order to make a comparison of damage at corresponding ages for the two stands examined during this study, average annual damage rates

for the current stand were extrapolated to the age of the previous stand at time of its harvest. The following damage estimates were compared: percent of Expected Stand Density infected or killed by P. weirii, percent of Expected Basal Area infected or killed, and Total Area of Infection as percent of total plot area. Average annual damage rates were calculated assuming constant rates for the life of both stands. The previous stand was estimated to be 300 years old at the time of harvest. Comparisons at a corresponding age were done by extrapolating average annual damage rates in the current stand to 300 years.

B. Results and Discussion.

1. Association of disease areas in the current and previous stands. The location of diseased areas in the current stand was related to the location of previous stand inoculum sources. One third to one half of the previous stand Area of Infection was found to support healthy trees in the current stand (Table XVII). Most of this healthy area is found on the previous stand Area of Concentrated Mortality. This represents successful healthy regeneration on areas that were previously out of production due to infection. This finding is not surprising when one considers that Hansen (1979c) found reduction of P. weirii survival in stumps cut 20 to 50 years previously. If this reduction can be extrapolated to even older inoculum sources then it would appear that trees killed more than 50 years previously would be much less effective as inoculum sources.

Table XVII. Currently healthy area overlying previous stand Total Area of Infection (ps TAI).

Plot	Area of overlap (m^2/ha)	Percent of ps TAI	Percent of total plot area
WC2	4692.8	52.7	46.9
WC3	3766.9	42.5	37.7
WC4	2423.3	37.2	24.2
WC5	2906.2	33.3	29.1
Average	3447.3	41.8	34.5

Table XVIII. Currently healthy area overlying previous stand Area of Potential Inoculum (ps API)

Plot	Area of overlap (m^2/ha)	Percent of ps API	Percent of total plot area
WC2	1154.1	44.8	11.5
WC3	781.1	28.4	7.8
WC4	746.7	30.7	7.5
WC5	630.9	21.8	6.3
Average	828.2	31.1	8.3

It is interesting to note, however, that disease in the current stand could not in all cases be attributed to initial infection from previous stand stumps. In plot WC2 some of the current P. weirii centers could only have originated by infection from old snags, since stumps were not found in the immediate vicinity (none within 20 m of the borders of a few infection centers). This observation prompted the inclusion of all possible sources of infection from the previous stand in the Area of Potential Inoculum.

As can be seen from the maps of the Areas of Potential Inoculum (Appendix 3), inoculum sources from the previous stand were fairly regularly distributed over most of the plots. This lead to a correspondingly wide distribution of infection centers in the current stand. The location of disease in the current stand was related to the distribution of previous stand inoculum sources and not to the configuration of previous stand disease caused openings. The currently healthy area overlying previous stand Area of Infection was larger (34.5% of total area)(Table XVII) than that overlying previous stand Area of Potential Inoculum (8.3% of total area)(Table XVIII). Spread of disease in the current stand beyond the boundaries of the previous infection centers was found to be minimal and varied (Table XIX). However, spread of disease in the current stand beyond the previous stand Area of Potential Inoculum for all the plots was much more consistent (Table XX) and accounted for 63-67% of the currently diseased area. These amounts present a better representation of the tree-to-tree spread in the current stand beyond initial inoculum sources. A similar study on an area with lower disease levels could more easily show unambiguous evidence for tree-to-tree spread beyond initial inoculum sources. The relative differences

Table XIX. Current stand Total Area of Infection (cs TAI) lying beyond the previous stand Total Area of Infection (ps TAI).

Plot	cs TAI beyond ps_2TAI (m^2/ha)	Percent of cs TAI	Percent of total plot area
WC2	42.2	1.0	0.4
WC3	179.6	3.4	1.8
WC4	915.6	18.3	9.1
WC5	296.9	4.4	2.7
Average	351.8	6.8	3.5

Table XX. Current stand Total Area of Infection (cs TAI) lying beyond the previous stand Area of Potential Inoculum (ps API).

Plot	cs TAI beyond ps_2API (m^2/ha)	Percent of cs TAI	Percent of total plot area
WC2	2826.8	66.6	28.3
WC3	3316.4	62.8	33.2
WC4	3324.8	66.3	33.2
WC5	3821.1	62.9	38.2
Average	3322.3	64.4	33.2

in the total amount of spread beyond initial inoculum found in this study were too small to analyze the factors affecting the rate of spread. Comparisons of spread beyond inoculum sources and average stand density (Table XX with Table V) failed to show any relationship. The plot with the highest density had the least amount of spread beyond initial inoculum.

The development of P. weirii root rot in the current stand has not, for the most part, resulted in readily defined infection centers. Only a few large openings have been created, and these do not have a regular margin. Examination of color infra-red aerial photos (supplied by Dr. E. Hansen) of the study plots revealed only these larger openings. The distribution of infected trees around P. weirii-created openings was also variable. Infection in the surrounding trees could have resulted from contact with previous stand inoculum sources in some areas, but many infected trees were found outside the rooting areas of these residuals. Although the average total spread beyond initial inoculum sources was fairly constant among the four plots, this spread was not by any means even. This suggests that the rate of spread varies considerably for different areas on the plots. This observation is somewhat contrary to the theory of disease development presented by Hadfield and Johnson (1978). Infection in standing trees seldom extended five meters beyond the openings, and tended to fill in areas between converging centers. This "bridging" of infection centers was in some cases associated with previous stand stumps, indicating possible infection of the standing trees by contact with infected roots of stumps. However, in most cases the standing infected trees

were not associated with previous stand inoculum sources, indicating possible tree-to-tree spread within the current stand. Healthy appearing individual trees as well as "islands" of healthy trees in infection centers were fairly common. Similar observations were made by Lawson (1979) in a second-growth stand at Black Rock Experimental Forest in the Oregon Coast Range.

2. Effectiveness of different sources of P. weirii inoculum from the previous stand. Table XXI presents the proportions of the current Area of Infection that overlap the rooting areas of infected stumps, snags, and down trees of the previous stand. Ninety-five percent of the old stumps with positive indicators of P. weirii decay were associated with current stand disease. An even higher percentage (96%) of the questionable old down trees were associated with current stand disease. However, many of these old down trees were close to positive stumps. Forty-three percent of the rooting area of uninfected residuals also fell within the current Area of Infection. This latter figure indicates error inherent in this method of estimating inoculum effectiveness. There are several possible reasons for such a high amount of error: 1) infection may have arisen from infected residuals, some of which were adjacent to uninfected residuals, 2) infection in the current stand has spread into areas that were previously healthy (Table XIX), 3) some of the uninfected residuals may have had infection in portions of the root system deeper than those examined.

Table XXI. Overlap of the current Area of Infection on the rooting areas of infected stumps, snags and down trees of the previous stand.

	Categories of previous stand residuals[b]					
	Stump		Snag		Down tree	
	pos.[a]	quest.[a]	pos.[a]	quest.[a]	pos.[a]	quest.[a]
No. of residuals[b]	221	59	153	264	39	28
Residuals overlapped by current Area of Infection	210 (95)[c]	51 (86)	130 (85)	83 (31)	33 (85)	27 (96)

[a]pos. = positive P. weirii, quest. = questionable P. weirii.
[b]totals for all four plots.
[c]percent of total number.

3. Vegetative incompatibility of P. weirii isolates.

The results of the vegetative incompatibility tests for all four plots are shown in Table XXII. The locations of the isolates are shown on the maps of the previous stand disease centers (Appendix 3). Isolates indentified by the same letter are of the same "clone" as determined by the test. These pairings show that there was considerable incompatibility among isolates on the one hectare plots. Childs (1970) interpreted such results as the convergence of separate infection centers in the

Table XXII. Results of vegetative incompatibility test for Phellinus weirii isolates.

+ = compatible reaction of the two paired isolates characterized by fusing of colonies with no discernable lines of demarcation

- = incompatible reaction of the two paired isolates characterized by formation of line of demarcation at interface between the colonies

c = pairing lost due to contamination

* = presence of dark submerged mycelium resembling Phellinus nigrolimitatus

f = isolated from tree with P. weirii fruiting bodies

P = isolated from previous stand source

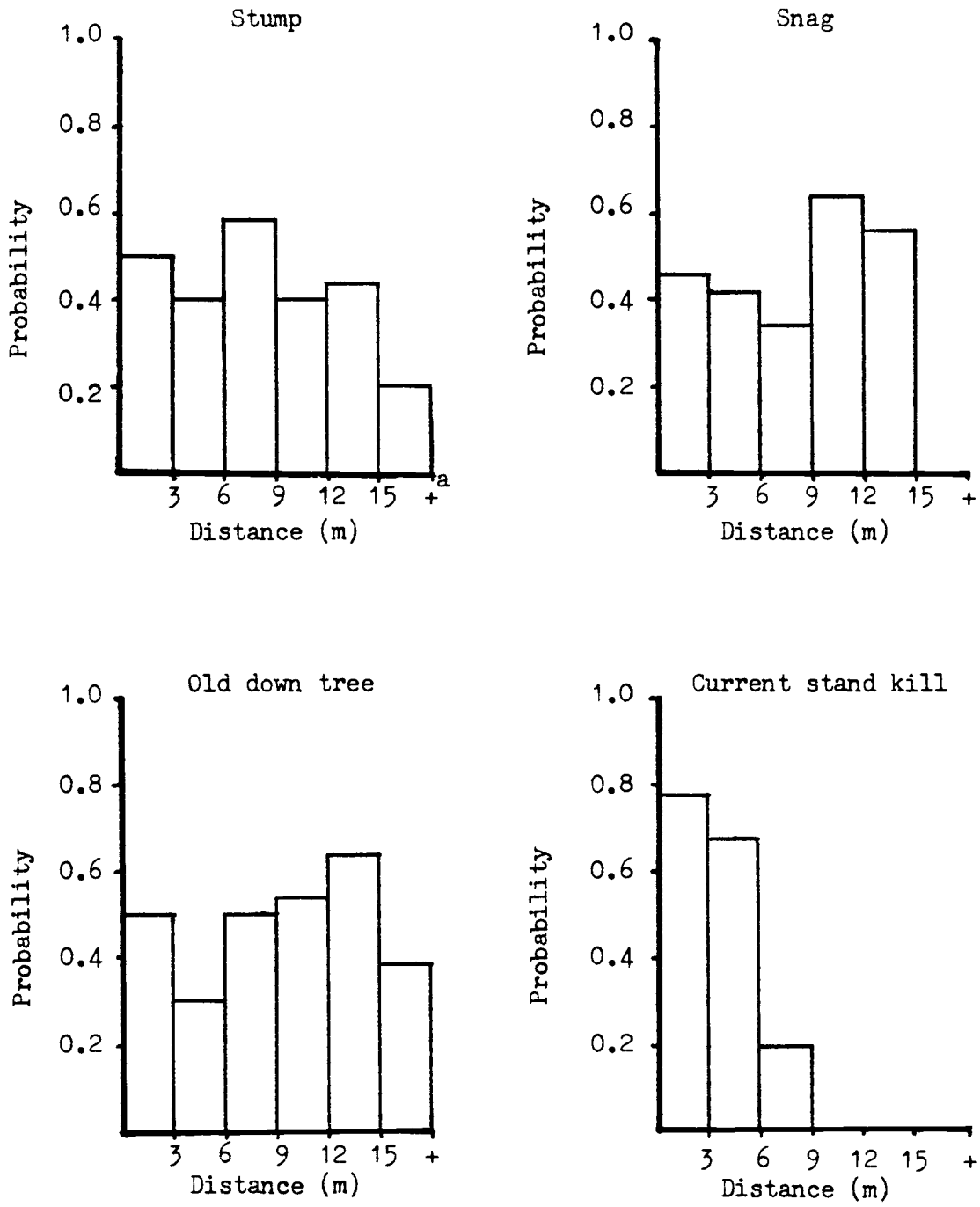
		WC2					WC3					WC4						WC5								
		9	574f	991f	1522f	1943f	96	379	613	514	846	92	239	592	856	915	919P	2f	228f	479f	615	764f	765f	273P	410P	475P
WC2	9	+	-	-	-*	-																				
	574f		+	+	-	+																				
	991f			+	-	+																				
	1522f				+	-																				
	1943f					+																				
WC3	96						+	-	-*	-*	+															
	379							+	c	-*	-															
	613								+*	c	+*															
	514									+*	c															
	846										+															
WC4	92											+	-	+	+	-										
	239												+	-	-	-										
	592													+	+	-										
	856														+	-										
	915															+	+									
	919P																									
WC5	2f																	+	-	c	+	+				
	228f																		+	c	-	-		-		
	479f																			+	c	c				
	615																				+	+				
	764f																					+	+			
	765f																									
	273P																							+	+	+
	410P																								+	+
	475P																									+

current stand originating from different centers in the previous stand. This hypothesis was investigated by comparing the compatibility results of the current stand isolates with the maps of the previous stand disease centers. On plot WC2 the isolates came from different centers in the current stand. The previous stand maps, however, reveal that the whole plot was covered by one large infection center. The three isolates from the southern portion of the plot were all of the same "clone", and the two in the northern portion were different from each other and from the other three. This indicates that the center in the previous stand may have formed as a result of convergence of at least three clones. Another possible explanation is changes in the compatibility factors over time. Plot WC3 yielded mixed results since the compatibility among three of the isolates was not consistent. This may be due to the possible presence of Phellinus nigrolimitatus in one of the isolates (# 613). The results of plot WC4 pairings show compatibility of three isolates, but the irregular pattern of disease in the previous stand did not lend itself to easy analysis of patterns. An isolate from a previous stand stump was cross-plated with an isolate from a current stand kill four meters away. The two isolates were compatible indicating that infection of the current stand tree may have resulted from contact with roots of the infected stump. Plot WC5 pairings indicated compatibility of three isolates. The map of the previous stand disease centers indicates one large center with many standing trees. Based on the results of the test this center must have arisen by convergence of at

least two "clones" of P. weirii. Four isolates were obtained from previous stand sources on plot WC5. Table XXII shows that all of these were compatible with each other. One of these (# 410) was from a very old snag with hollowed out roots. The pairing of a current stand isolate with a previous stand isolate 16 m away resulted in an incompatible reaction indicating involvement of different "clones". More pairings are needed to accurately determine the distribution of "clones" of P. weirii on the plots. Also, the role of Phellinus nigrolimitatus as a possible "cohabitant" must be elucidated before any firm conclusions can be drawn from these results. The presence of dark discoloration of the agar below P. weirii colonies was noticed in a few of the isolates after about one month of incubating. These areas were seen to contain submerged mycelium uncharacteristic of P. weirii. Subculturing was attempted from these areas, and although the success of isolating fungi from these zones was limited, a few apparently pure cultures were obtained. The fungus found in these zones has been tentatively identified as Phellinus nigrolimitatus (Romell) Bourd & G. by using the keys of Nobles (1948 & 1965). This suggests a possible complex of P. weirii with P. nigrolimitatus as has been reported occurring in Idaho (Miller and Partridge, 1973; Chacko and Partridge, 1976).

4. Probability of infection with increasing distance from inoculum source. The results of this test are presented in Figure 6. The probability of infection generally decreases with increasing distance

Figure 6. Probability of P. weirii infection with distance (m) from old infected stumps, snags, and down trees, and current stand killed trees.



[a]distance greater than 15 meters

as expected for a pathogen spreading by root contacts. The steeper this negative slope, the more useful the inoculum source for predicting infection in the surrounding trees. For all of the previous stand inoculum sources the probability of infection remained near 0.5 throughout the distance range measured. Therefore, previous stand residuals are not good indicators of infection in surrounding trees. Previous stand stumps showed a steeper slope than the other categories of residuals (Figure 6). Current stand killed trees showed a much better relationship between distance and probability of infection. No infected trees were found more than 9 m from current stand killed trees. This indicates that the infection of standing trees arises more commonly (but probably not exclusively) from current stand sources than previous stand sources.

5. Comparison of P. weirii damage at corresponding ages in the two succeeding stands. Comparisons of damage were made by extrapolating calculated average annual rates. Due to a lack of data on changes in the rate of damage it was assumed that the rate remains constant throughout the life of the stand. Table XXIII presents average annual rates of increasing damage for the current and previous stands. The annual rates for the current stand were much higher than those for the previous stand (Table XXIII). If the damage proceeds at these rates, the levels of damage in the current stand at 300 years (the approximate age of the preceding stand at harvest) will be considerably higher than those found in the preceding stand, thus substantiating Shea's prediction. However, Childs (1970) found rates of damage to vary with age. Damage rates increased with age up to

60 years but then dropped off for stands 110 years old. Therefore, the above predictions can only be gross estimations without more precise data on damage rate fluctuations.

Table XXIII. Average annual rates of percent increase of Phellinus weirii damage for the current and previous stands.

	Current Stand[a]		Previous Stand[b]	
	Cumulative	Annual	Cumulative	Annual
Percent of Expected Stand Density infected or killed	62.2	1.04	90.4	0.30
Percent of Expected Basal Area infected or killed	51.7	0.86	84.7	0.28
Percent of area in Total Area of Infection	51.6	0.86	82.5	0.28

[a]Cumulative and annual values at age 60.

[b]Cumulative and annual values at age 300.

C. Conclusions.

The results of this study indicated the following trends of disease spread from the previous to the current stand:

The location of diseased areas in the current stand was related to the location of previous stand inoculum sources. The wide distribution of initial inoculum sources led to multiple initial infection of current stand trees. Tree to tree spread of the disease beyond

initial inoculum sources was constant among the four plots and accounted for two-thirds of the Total Area of Infection. Much of the previous stand Area of Concentrated Mortality was found to support currently healthy trees.

Of the previous stand inoculum sources, the stumps with positive indicators of P. weirii infection were most often associated with disease in the current stand. However, a high proportion of uninfected residuals also had current stand disease within their rooting area. This indicates that association with disease in current stand trees is not a good estimator of inoculum effectiveness in a 60 year-old stand.

The vegetative incompatibility test indicated the presence of several "clones" of P. weirii on each of the plots. More pairings are needed to accurately determine the distribution of these clones and how they relate to the disease in the previous stand. Further studies are also needed to elucidate the occurrence of Phellinus nigrolimitatus in a complex with P. weirii.

The probability of infection by P. weirii in standing trees decreases with increasing distance from current stand P. weirii mortality. No infected trees were found more than 9 m from current stand killed trees. Previous stand inoculum sources were not effective in predicting infection in standing trees.

Although damage levels were higher in the previous 300 year-old stand than those in the current 60 year-old stand, the average annual rates of damage increase were much higher for the current stand than the previous stand. If damage continues at these rates the level of damage in the current stand at 300 years will be higher than that found in the previous stand.

VII. SUMMARY

The following list is a synopsis of the most pertinent results of this series of studies on Phellinus weirii damage in two succeeding Douglas-fir stands.

1). Successful diagnosis of Phellinus weirii infection in standing trees requires examination of the crown, root collar, and interior of lower bole of suspect trees for signs or symptoms of the disease. The most common external sign was surface mycelium on the roots. The best indicator of internal decay was visual examination of increment cores.

2). Assessment of disease losses by estimating proportion of trees or volume affected is more meaningful when compared to expected stand density and volume for a healthy stand than when compared to existing density and volume.

3). Damage due to Phellinus weirii in the current 60 year-old stand, as assessed by reduction of Expected Stand Density and Expected Basal Area as well as by the proportion of stand area covered by Total Area of Infection has not reached the levels of damage found in the preceding 300 year-old stand.

4). Assessment of damage due to Phellinus weirii by area affected resulted in higher estimates than did reduction of stand density or volume.

5). Differences in the Total Area of Infection in the current

stand are best related to the type and area covered by inoculum sources from the previous stand.

6). The probability of infection by Phellinus weirii in standing trees decreases with increasing distance from current stand P. weirii mortality, with no infection occurring beyond nine meters.

7). Linear projections of damage rates in the current stand to the age of the previous stand indicate much higher expected losses in the current stand than were found in the previous stand.

Phellinus weirii is an endemic tree pathogen in the Douglas-fir region of western Oregon and Washington. Its presence in the ecosystem is as natural as that of Douglas-fir and the other timber species. The activity of laminated root rot caused by P. weirii in the primeval forest ecosystem most likely led to successional changes in the overstory and not necessarily to "damage" to the ecosystem as a whole. Although large local areas may have been affected by the disease, the relatively slow spread of the disease was probably offset by the gradual encroachment of Douglas-fir onto previously infected areas after the pathogen had died out. Hence, a region-wide equilibrium was most likely established between the host and the pathogen.

Endemic pathogens such as Phellinus weirii can only be termed damaging agents when their hosts are assigned commercial value or when the established equilibrium is affected to favor the pathogen instead of the host. The current value placed on timber has led to the development of intensive management systems for the forests designed for

maximum yield of a desirable product in a minimum of time. For the Oregon Coast Range forests this means repeated rotations of Douglas-fir, the commercially preferred timber species. The high value placed on Douglas-fir and the alteration of successional patterns by continual cropping of a single species greatly elevate the importance and impact of laminated root rot caused by P. weirii.

This study has dealt with the first step in many forest management systems, namely, the total harvest of a virgin stand and its impact on disease development in the succeeding stand. Since regeneration of forests did not concern the early loggers, the area under study was left to regenerate naturally by seeding. The area burned severely after the harvest which probably delayed the regeneration of the succeeding stand. Our analyses of the damage levels in the two stands have led us to conclude that there has been an intensification of P. weirii caused damage in the succeeding stand. The good correlation of previous stand inoculum sources and current stand disease indicate significant carry-over of the disease from the previous stand. The total harvest of the previous stand led to a wide distribution of inoculum sources in the form of infected stumps, snags, and old down trees. This led to multiple initial infections of the regenerating stand and the resulting wide distribution of disease in that stand at 60 years. The levels of disease in the current stand might have been even higher if current regeneration practices (such as planting and suppression of competition) had been implemented after the harvest of the preceding stand. Such measures would have led to more rapid regeneration and thus possibly more initial infections of the current

stand.

The current stand on the study area has not been thinned. Since thinnings (precommercial and commercial) are an important part of intensive management, their effect on disease development must also be studied to accurately predict losses due to P. weirii in a managed forest. Changes in stand density may play an important role in altering disease development.

This study has provided the first known documentation of laminated root rot development in two succeeding stands. The situation studied, namely, a second-growth stand regenerating on a site previously occupied by an infected old-growth stand, will soon be but past history. A similar comparison of second and third generation stands is needed to accurately predict future losses due to the disease in an intensive management system. Greater amounts of potential inoculum sources from the second generation could lead to even higher carry-over of root rot to the third generation stand. Although the results of this study cannot indicate levels of damage to the third stand, they do suggest that accurate predictions of damage from one stand to the next can be made only if the extent of infection in standing trees is known since stumps of these trees are the most effective inoculum sources for the succeeding stand.

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APPENDICES

APPENDIX 1.

LOCATION OF STUDY PLOTS

Travel log to plot locations.

Mi.[a]	Km.	
0.0	0.0	Junction of Hwy 20 and Hwy 34 west of Philomath, Oregon, proceed west along Hwy 20.
1.8	2.8	Turn left onto Woods Creek Road (For. Serv. Road 2005-000).
2.6	4.2	Bridge over small creek and end of pavement, proceed along Woods Creek Road.
6.4	10.3	Rock quarry on right, proceed along Woods Creek Road (road narrows to one lane with turnouts).
8.0	12.9	Intersection with For. Serv. Road 2005-111, stay to right and proceed along Woods Creek Road.
8.3	13.4	Intersection with For. Serv. Road 2005-121, proceed straight along Woods Creek Road.
9.0	14.5	Intersection with For. Serv. Road 2005-122, turn right for plot WC2: proceed 0.25 mi (0.4 km) along road, then bear 180° to WC2-CP1 (Compass Point 1). Proceed along Woods Creek Road for other plots.
9.2	14.8	Intersection with For. Serv. Road 2005-123, for plot WC5 bear 300° for WC5-CP1 (See Plot references for accurate location).
9.5	15.3	Intersection with For. Serv. Road 2005-112, Corvallis Municipal Watershed gate across Woods Creek Road. Bear 180° to plot WC3-CP1. Proceed along Woods Creek Road for plot WC4.
9.7	15.6	Cross small stream, proceed along Woods Creek Road.
10.2	16.4	Plot WC4 located approximately 280° 50 m from road, proceed on to next intersection for accurate bearing and distance to WC4-CP1.
10.3	16.6	Intersection with For. Serv. Road 2005-114, see plot references for location of WC4-CP1.

[a]Mileage figures based on vehicle odometer readings.

Plot references.

Plot	Azimuth and distance to reference point from one of the compass points (CP#) on each plot:
WC2	CP1 to section corner T.12S,R.7W.,Sec.9,10,15,16:
	260° 172 m
WC3	CP1 to concrete post anchoring SE post of Corvallis Municipal Watershed gate across For. Serv. Road 2005-000 at intersection with 2005-112:
	253° 30.8 m
WC4	CP1 to sign post at intersection of For. Serv. Roads 2005-000 and 2005-114:
	215° 203.3 m
WC5	CP3 to first quarter section marker west of section corner T.12S.,R.7W.,Sec.9,10,15,16:
	342° 39.7 m

APPENDIX 2.

LISTING OF COMPUTER PROGRAM FOR PLOTTING

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      CSS FORTRAN  VERSION 3.13          11/28/78  2308

0+001           PROGRAM TREACT
0+002           INTEGER NORTHRO,GRNW,GRNH
0+003           DIMENSION RLABEL(7),ICARD(19),PLABEL(7),ISYP(16)
0+004           CHARACTER CC(76)
0+005           COMMON NORTHRO,CONVER,PSCALE
0+006           COMMON /SPLIT/ IMAP,ISPLIT
0+007           EQUIVALENCE (ICARD,CC)
0+008           INPFLG=0
0+009   C   READ PARAMETER CARD 1
0+010           READ(60,1) NTYPE,(PLABEL(I),I=1,4),ADATE,ISPLIT,(ISYP(I),I=1,16)
0+011   1       FORMAT(A4,4A8,3X,A8,3X,I1,3X,16A2)
0+012           IF(NTYPE.EQ.4HGRID.OR.NTYPE.EQ.4HCOMP) GO TO 100
0+013           WRITE(61,1001) NTYPE
0+014   1001    FORMAT(1- PLOT TYPE NOT SPECIFIED CORRECTLY 1,A4)
0+015           CALL EXIT
0+016   100     CALL DATE (BDATE)
0+017   C   READ PARAMETER CARD 2
0+018           READ(60,2) NORTHRO,GRW,GRNW,GRNH,CONVER,PSCALE
0+019   2       FORMAT(I3,F7.0,2I4,2F10.0)
0+020           IF(NTYPE.EQ.4HCOMP) GO TO 110
0+021           IF(GRW.LT..0001) GO TO 105
0+022           IF(GRNW.LE.0) GO TO 105
0+023           IF(GRNH.LE.0) GO TO 105
0+024           GO TO 110
0+025   105     WRITE(61,3) NORTHRO,GRW,GRNW,GRNH
0+026   3       FORMAT(1- NOT ALL INFORMATION FOR GRID PROVIDED1
0+027         1,/,3X,I4,F15.8,2I6)
0+028           CALL EXIT
0+029   110     IF(ABS(CONVER).LT..0001) CONVER=1.
0+030           IF(ABS(PSCALE).LT..0001) PSCALE=1.
0+031           IF(NTYPE.EQ.4HCOMP) GO TO 115
0+032           PLOTW=GRW*CONVER*GRNW
0+033   C       PLOTWI = PLOT WIDTH IN INCHES
0+034           PLOTWI=PLOTW/PSCALE * .3937007
0+035           IF(PLOTWI.LT.29) GO TO 115
0+036           WRITE(61,4) PLOTWI
0+037   4       FORMAT(1- GRID SCALING AND SETUP CAUSES PLOT TO EXCEED1,
0+038         1/ MAXIMUM WIDTH1,/,1     CALCULATED SIZE = 1,F15.8)
0+039           CALL EXIT
0+040   115     WRITE(61,1002) (PLABEL(I),I=1,4),ADATE,NTYPE,BDATE
0+041   1002    FORMAT(1H0,/,1H0,5X,4A8,/,1 0SURVEY DATE = 1,
0+042         1A8,/,1 0PLOT TYPE = 1,A4,5X,1PLOT DATE = 1,A8)
0+043           IF(NTYPE.EQ.4HCOMP) GO TO 116
0+044           WRITE(61,1003)NORTHRO,GRW,GRNW,GRNH
0+045   1003    FORMAT(10ANGLE ROTATION TO TRUE NORTH = 1,I8,
0+046         1/,10GRID DIVISION WIDTH = 1,F15.6,1 UNITS1,
0+047         2/,10NUMBER OF GRID DIVISIONS WIDE 1,I5,
0+048         3/,10NUMBER OF GRID DIVISIONS HIGH 1,I5,)
0+049   116     WRITE(61,1004)CONVER,PSCALE
0+050   1004    FORMAT(10CONVERSION FACTOR, 1 UNIT = 1,F15.6,1 METERS1,
0+051         1/,10PLOT SCALE 1,F15.6,1 METERS = 1 CENTIMETER1)
0+052           WRITE(61,1005)
0+053   1005    FORMAT(1- LISTING OF SURVEY CARDS1,/,
0+054         1 0CARD   PLT   COMPASS   TREE1,/,
0+055         2 1 TYPE    ID    PT    NUMBER   ANGLE    DIST    SPECIES  DIAM1,
0+056         3 1   DEATH--YR  CONFIRM    CROWN1)
0+057           GRW=GRW*CONVER/PSCALE
0+058   C       GRW NOW EQUALS GRID DIVISION SIZE IN CENT.
0+059           IPTYPE=NTYPE
0+060   121     IF(NORTHRO.GE.0) GO TO 122
0+061           NORTHRO=NORTHRO+360
0+062           GO TO 121
0+063   122     IF(NORTHRO.LE.360) GO TO 123
0+064           NORTHRO=NORTHRO-360
0+065           GO TO 122
0+066   C   NORTHRO NOW EQUALS ROTATION TO NORTH FROM Y AXIS
0+067   123     NORTHRO=360-NORTHRO
0+068           IF(IPTYPE.EQ.4HCOMP) XR=XL=0.
0+069           CALL EQUIP(51,5HFILE )
0+070           YMIN=XMIN=9999999.
0+071           YMAX=XMAX=-9999999.
0+072   117     READ(60,5)NTYPE,ID,ICARD
0+073   5       FORMAT(A1,A3,19A4)
0+074           IF(EOF(60)) GO TO 500
0+075   120     IF(IPTYPE.EQ.4HGRID.AND.NTYPE.EQ.1H0) GO TO 125
0+076           IF(IPTYPE.EQ.4HCOMP.AND.NTYPE.EQ.1H0) GO TO 125
0+077           WRITE(61,6) IPTYPE,NTYPE,ID,ICARD
0+078   6       FORMAT(1- CARD TYPE NOT CORRECT FOR PLOT MODE 1,A4,
0+079         1/,2X,A1,A3,19A4)
0+080           INPFLG=1
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0+081                 GO TO 117
0+082   125           WRITE(61,1006) NTYPE,ID,(CC(I),I=1,20)
0+083   1006          FORMAT(3X,A1,3X,A3,4X,2R1,4X,5R1,4X,3R1,3X,5R1,60R1)
0+084                 IF(NTYPE.NE.1HB) GO TO 130
0+085                 DECODE(15,1007,ICARD(1))IL,IR,IANGLE,DIST
0+086   1007          FORMAT(2X,I2,1X,I2,I3,F5.0)
0+087                 XL=IL*GRW
0+088                 XP=IR*GRW
0+089                 GO TO 135
0+090   130           DECODE(8,1008,ICARD(2))IANGLE,DIST
0+091   1008          FORMAT(I3,F5.0)
0+092   135           IANGLE=IANGLE-180
0+093                 CALL XYCOMP(XL,XP,IANGLE,DIST,X,Y)
0+094                 XL=X
0+095                 XP=Y
0+096   140           READ(60,5) NTYPE,ID,ICARD
0+097                 IF(EOF(60)) GO TO 500
0+098                 IF(NTYPE.NE.1HS.AND.NTYPE.NE.1HT) GO TO 120
0+099                 IF(NTYPE.NE.1HT) GO TO 145
0+100                 DECODE(27,1009,ICARD(1))ITREE,IANGLE,DIST,ISPEC,IDTH,IYR,ICON,ICR
0+101   1009          FORMAT(2X,I5,1X,I3,F5.0,A2,3X,A2,A2,A1,I1)
0+102                 IF(ICR.GT.5) GO TO 153
0+103                 WRITE(61,1009)NTYPE,ID,(CC(I),I=1,40)
0+104   1009          FORMAT(3X,A1,3X,A3,4X,2R1,4X,6R1,3X,3R1,3X,5R1,6X,2R1,5X,3R1,4X,2R
0+105                1,3X,2R1,5X,R1,8X,R1,20R1)
0+106                 GO TO 155
0+107   145           DECODE(13,1010,ICARD(1))LAB,IANGLE,DIST
0+108   1010          FORMAT(2X,A1,2X,I3,F5.0)
0+109                 WRITE(61,1011) NTYPE,ID,(CC(I),I=1,20)
0+110   1011          FORMAT(3X,A1,3X,A3,4X,2R1,5X,3R1,5X,3R1,3X,5R1,6X,2R1,5X,3R1,4X,2R
0+111                1,3X,2R1,5X,R1,8X,R1,20R1)
0+112                 GO TO 155
0+113   153           WRITE(61,1012) NTYPE,ID,ICARD
0+114   1012          FORMAT(*- BAD CARD *,A1,A3,19A4)
0+115                 INPFLG=1
0+116                 GO TO 117
0+117   155           CALL XYCOMP(XL,XP,IANGLE,DIST,X,Y)
0+118                 IF(NTYPE.NE.1HT) GO TO 160
0+119                 WRITE(51,1013)NTYPE,X,Y,ITREE,ICON,ISPEC,IDTH,ICR
0+120   1013          FORMAT(A1,2F15.8,I5,A1,2X,A2,2X,A2,2X,I1)
0+121                 GO TO 165
0+122   160           WRITE(51,1014)NTYPE,X,Y,LAB
0+123   1014          FORMAT(A1,2F15.8,A1)
0+124   165           IF(XMAX.GT.X) GO TO 170
0+125                 XMAX=X
0+126                 GO TO 175
0+127   170           IF(XMIN.LT.X) GO TO 175
0+128                 XMIN=X
0+129   175           IF(YMAX.GT.Y) GO TO 180
0+130                 YMAX=Y
0+131                 GO TO 185
0+132   180           IF(YMIN.LT.Y) GO TO 185
0+133                 YMIN=Y
0+134   185           GO TO 140
0+135   500           ENDFILE 61
0+136                 REWIND 61
0+137                 WRITE(61,1103)
0+138   1103          FORMAT(1H1)
0+139                 CALL PLCTLUN (10)
0+140                 CALL PLOTTYPE (3)
0+141                 IF(IPTYPE.NE.4HGRID) GO TO 400
0+142                 IY=1
0+143                 CALL FNDBOND(XMAX,PXMAX,IXMAX,GRW,IX)
0+144                 IF(IX.EQ.-1) GO TO 190
0+145                 CALL FNDBOND(YMAX,PYMAX,IYMAX,GRW,IX)
0+146                 IF(IX.EQ.-1) GO TO 190
0+147                 IX=0
0+148                 CALL FNDBOND(XMIN,PXMIN,IXMIN,GRW,IX)
0+149                 IF(IX.EQ.-1) GO TO 190
0+150                 CALL FNDBOND(YMIN,PYMIN,IYMIN,GRW,IX)
0+151                 IF(IX.NE.-1) GO TO 200
0+152   190           WRITE(61,1015) PXMIN,PXMAX,PYMIN,PYMAX
0+153   1015          FORMAT(*- PLOT LIMITS UNREALISTIC *,4E15.8)
0+154                 CALL EXIT
0+155   200           IEMP=IXMAX-IXMIN
0+156                 IF(IEMP-GRNW) 220,230,210
0+157   210           IEMP=(XMAX-XMIN)/GRW+.5
0+158                 IF(IEMP.GT.GRNW) GO TO 215
0+159                 PXMAX=XMAX
0+160   -             PXMIN=XMIN
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0+161            GO TO 230
0+162   215      WRITE(61,1100) IEMP
0+163   1100     FORMAT(*- NUMBER OF GRID DIVISIONS WIDE NOT ENOUGH*,/
0+164           1,* NEED AT LEAST *,I8)
0+165            CALL EXIT
0+166   220      IEMP=(GRNW-(IXMAX-IXMIN))/2
0+167            IF(IEMP.LE.0) GO TO 230
0+168            IXMIN=IXMIN-IEMP
0+169            PXMIN=GRW*IXMIN
0+170            IXMAX=GRNW+IXMIN-IEMP
0+171            PXMAX=GRW*IXMAX
0+172   230      IEMP=IYMAX-IYMIN
0+173            IF(IEMP-GRNH)240,250,260
0+174   260      IEMP=(YMAX-YMIN)/GRW+.5
0+175            IF(IEMP.GT.GRNH) GO TO 235
0+176            PYMAX=YMAX
0+177            PYMIN=YMIN
0+178            GO TO 250
0+179   235      WRITE(61,1101) IEMP
0+180   1101     FORMAT(*- NUMBER OF GRID DIVISIONS HIGH NOT ENOUGH*,/
0+181           1,* NEED AT LEAST *,I8)
0+182            CALL EXIT
0+183   240      IEMP=(GRNH-(IYMAX-IYMIN))/2
0+184            IF(IEMP.LE.0) GO TO 250
0+185            IYMIN=IYMIN-IEMP
0+186            PYMIN=GRW*IYMIN
0+187            IYMAX=GRNH+IYMIN-IEMP
0+188            PYMAX=GRW*IYMAX
0+189   250      XSIZE=(PXMAX-PXMIN)*.39370079
0+190            YSIZE=(PYMAX-PYMIN)*.39370079
0+191            IF(XSIZE.LE.29.) GO TO 256
0+192            WRITE(61,1102) XSIZE
0+193   1102     FORMAT(*- SEE PROGRAMMER, IMPOSSIBLE ERROR*,F15.8)
0+194   256      TEMP=YSIZE*6.
0+195            IF(INPFLG) CALL EXIT
0+196   700      REWIND 51
0+197            IF(IPTYPE.NE.4HGRID) GO TO 400
0+198            CALL SIZE(58.,TEMP)
0+199            XBIAS=(58.-XSIZE)/2.
0+200            FACT=.39370079
0+201            CALL SCALE(FACT,FACT,XBIAS,2.,PXMIN,PYMIN)
0+202            CALL VECTORS
0+203            TEMP=IXMIN*GRW
0+204   265      IF(ABS(PXMIN-TEMP).LT..0001) GO TO270
0+205            IF(TEMP.LT.PXMIN) GO TO 270
0+206            TEMP=TEMP-GRW
0+207            GO TO 265
0+208   270      IF(ABS(PXMIN-TEMP).LT..0001) GO TO 280
0+209            IF(TEMP.GE.PXMIN) GO TO 280
0+210   275      TEMP=TEMP+GRW
0+211   280      IF(TEMP.GT.PXMAX) GO TO 285
0+212            CALL PLOT(TEMP,PYMIN,0,0)
0+213            CALL PLOT(TEMP,PYMAX,1,0)
0+214            GO TO 275
0+215   285      TEMP=IYMIN*GRW
0+216   290      IF(ABS(PYMIN-TEMP).LT..0001) GO TO 295
0+217            IF(TEMP.LE.PYMIN) GO TO 295
0+218            TEMP=TEMP-GRW
0+219            GO TO 290
0+220   295      IF(ABS(PYMIN-TEMP).LT..0001) GO TO 310
0+221            IF(TEMP.GE.PYMIN) GO TO 310
0+222   300      TEMP=TEMP+GRW
0+223   310      IF(TEMP.GT.PYMAX) GO TO 315
0+224            CALL PLOT(PXMIN,TEMP,0,0)
0+225            CALL PLOT(PXMAX,TEMP,1,0)
0+226            GO TO 300
0+227   315      ENCODE(7,1016,TEMP)IXMIN,IYMIN
0+228   1016     FORMAT(*(*,I2,*,*,I2,*)*)
0+229            XTEMP=PXMIN-1.27
0+230            YTEMP=PYMIN-1.27
0+231            CALL SYMBOL (XTEMP,YTEMP,0.0,.16,7,TEMP)
0+232            ENCODE(7,1016,TEMP)IXMAX,IYMAX
0+233            XTEMP=PXMAX-1.27
0+234            YTEMP=PYMAX+1.27
0+235            CALL SYMBOL (XTEMP,YTEMP,0.0,.16,7,TEMP)
0+236            GO TO 410
0+237   400      XSIZE=(XMAX-XMIN)*.3937079
0+238            YSIZE=(YMAX-YMIN)*.3937079
0+239            IF(XSIZE.LE.58) GO TO 407
0+240            WRITE(61,1102)
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0+241            INPFLG=1
0+242   407      TEMP=YSIZE+6.
0+243            IF(TEMP.LE.42) GO TO 408
0+244            WRITE(61,1020)
0+245   1020     FORMAT(' YSIZE TOO BIG')
0+246   408      IF(INPFLG) CALL EXIT
0+247            CALL SIZE(58.,TEMP)
0+248            XBIAS=(58.-XSIZE)/2.
0+249            FACT=.39370079
0+250            CALL SCALE(FACT,FACT,XBIAS,2.,XMIN,YMIN)
0+251            CALL VECTORS
0+252            PXMIN=XMIN
0+253            PXMAX=XMAX
0+254            PYMIN=YMIN
0+255            PYMAX=YMAX
0+256   410      XTEMP=PXMIN+5.08
0+257            TEMP=PYMAX+5.08
0+258            DO 411 I=1,7
0+259   411      RLABEL(I) = BLABEL(I)
0+260            CALL SYMBOL(XTEMP,TEMP,0.0,.16,32,RLABEL)
0+261            TEMP=TEMP-2.54
0+262            RLABEL(1)=8HSURVEY D
0+263            RLABEL(2)=8HATE  --
0+264            RLABEL(3)=8DATE
0+265            CALL SYMBOL(XTEMP,TEMP,0.0,.16,24,RLABEL)
0+266            RLABEL(1)=8HPLOT  D
0+267            RLABEL(2)=8HATE  --
0+268            RLABEL(3)=8DATE
0+269            TEMP=TEMP-1.27
0+270            CALL SYMBOL(XTEMP,TEMP,0.0,.16,24,RLABEL)
0+271            IF(.NOT.ISPLIT) GO TO 710
0+272            IMAP=IMAP+1
0+273            RLABEL(1)=8HSPLIT-PL
0+274            RLABEL(2)=8HOT
0+275            TEMP=TEMP+2.54
0+276            CALL SYMBOL(XTEMP,TEMP,0.0,.16,10,RLABEL)
0+277   710      ENCODE(50,1017,RLABEL(1))PSCALE
0+278   1017     FORMAT('PLOT SCALE = ',F15.8,' METERS PER CENTIMETER')
0+279            TEMP=PYMIN-1.27
0+280            CALL SYMBOL(XTEMP,TEMP,0.0,.16,50,RLABEL)
0+281            XTEMP=PXMAX-5.08
0+282            YTEMP=PYMAX+3.81
0+283            TEMP=5.08*PSCALE/CONVER
0+284            CALL PLOT (XTEMP,YTEMP,0.0)
0+285            CALL XYCOMP(XTEMP,YTEMP,0,TEMP,X,Y)
0+286            CALL PLOT (X,Y,1,0)
0+287            TEMP=1.*PSCALE/CONVER
0+288            IF(X.GE.XTEMP) GO TO 418
0+289   418      CALL XYCOMP(X,Y,.135,TEMP,XT,YT)
0+290            CALL PLOT(XT,YT,1,0)
0+291            IF(X.GE.XTEMP) GO TO 419
0+292            X=X-3.81
0+293   419      TEMP=8H NORTH
0+294            CALL SYMBOL (X,Y,0.0,.16,7,TEMP)
0+295            CALL POINTS
0+296   420      READ(51,1018)NTYPE,X,Y,(ICARD(I),I=1,5)
0+297   1018     FORMAT(A1,2F15.8,4A4,A1)
0+298            IF(EOF(51)) GO TO 600
0+299            IF(NTYPE.NE.1HT) GO TO 425
0+300            DECODE(9,1019,ICARD(3))ISPEC,IDTH,ICR
0+301   1019     FORMAT(A2,2X,A2,2X,I1)
0+302            IF(.NOT.ISPLIT) GO TO 720
0+303            DO 730 I=1,16
0+304            IF(ISPEC.EQ.ISYM(I)) GO TO 740
0+305   730      CONTINUE
0+306            IF(IMAP.NE.1) GO TO 720
0+307            GO TO 420
0+308   740      IF(IMAP.NE.1) GO TO 420
0+309   720      CALL XYPLOT(NTYPE,X,Y,ICARD,ISPEC,IDTH,ICR)
0+310            GO TO 420
0+311   425      CALL XYPLOT(NTYPE,X,Y,ICARD,0,0,0)
0+312            GO TO 420
0+313   600      CALL PLOTEND
0+314            IF(ISPLIT.AND.IMAP.EQ.1) GO TO 700
0+315            CALL EXIT
0+316            END

 NO ERRORS FOR TRACT
 LENGTH OF SUBPROGRAM        03376
 LENGTH OF COMMON SPLIT      01002
 LENGTH OF COMMON            00005
```

```
        OS3 FORTRAN  VERSION 3.13          11/28/78  2308

     0+317          SUBROUTINE XYCOMP(XTRAN,YTRAN,IANGLE,DIST,X,Y)
     0+318          INTEGER NORTHO
     0+319          COMMON NORTHO,CONVER,PSCALE
     0+320          DATA (PI=3.1415927)
     0+321          DEGREES=NO*THRO-IANGLE
     0+322  10      IF(DEGREES.GE.0) GO TO 20
     0+323          DEGREES=DEGREES+360.
     0+324          GO TO 10
     0+325  20      IF(DEGREES.LE.360.) GO TO 30
     0+326          DEGREES=DEGREES-360.
     0+327          GO TO 20
     0+328  30      RADIANS=PI*(DEGREES/180.)
     0+329          XDIST=DIST*CONVER/PSCALE
     0+330          X=XDIST*COS(RADIANS)+XTRAN
     0+331          Y=XDIST*SIN(RADIANS)+YTRAN
     0+332          RETURN
     0+333          END

  NO ERRORS FOR XYCOMP
 LENGTH OF SUBPROGRAM      00110
 LENGTH OF COMMON          00006
```

```
      CS3 FORTRAN  VERSION 3.13          11/20/78  230P

0+334          SUBROUTINE ENDBOND(XY,XYR,IXY,GRW,IFLAG)
0+335          IF(XY.LT.0) GO TO 20
0+336          DO 10 I=1,500
0+337          XYR=(I-1)*GRW
0+338          IF(IFLAG) GO TO 11
0+339          IF(XY.GT.XYR) GO TO 10
0+340          IXY=I-2
0+341          XYR=IXY*GRW
0+342          RETURN
0+343    11    IF(XYR.LT.XY) GO TO 10
0+344          IXY=(I-1)
0+345          RETURN
0+346    10    CONTINUE
0+347          IFLAG=-1
0+348          RETURN
0+349    20    DO 30 I=1,500
0+350          XYR=(-I)*GRW
0+351          IF(IFLAG) GO TO 21
0+352          IF(XY.LT.XYR) GO TO 20
0+353          IXY=(-I)
0+354          RETURN
0+355    21    IF(XYR.GT.XY) GO TO 20
0+356          IXY=1-I
0+357          XYR=IXY*GRW
0+358          RETURN
0+359    30    CONTINUE
0+360          IFLAG=-1
0+361          RETURN
0+362          END

 NO ERRORS FOR ENDBOND
 LENGTH OF SUBPROGRAM     0C162
```

```
      CDC FORTRAN  VERSION 3.13           11/28/78  2308

0+353          SUBROUTINE XYPLOT(NTYPE,X,Y,ICHAR,ISPEC,IDTH,ICRN)
0+364          COMMON /SPLIT/ IMAP,ISPLIT
0+355          DIMENSION ICHAR(19)
0+366          ICR=ICRN+1
0+367          IF(NTYPE.NE.1HS) GO TO 10
0+368          XT=X-.205
0+369          YT=Y
0+370          CALL VECTORS
0+371          CALL PLOT(XT,YT,0,0)
0+372          XT=XT+.41
0+373          CALL PLOT(XT,YT,1,0)
0+374          YT=YT-.205
0+375          YT=YT+.3922
0+376          CALL PLOT (XT,YT,1,0)
0+377          YT=Y
0+378          XT=X-.205
0+379          CALL PLOT(XT,YT,1,0)
0+380          XT=X-.102
0+381          CALL SYMBOL (XT,YT,0.1,.08,1,ICHAR)
0+382          CALL POINTS
0+383          RETURN
0+384    10    IF(ISPEC.NE.2HDF.AND.ISPEC.NE.2HML.AND.ISPEC.NE.2HNF) GO TO 30
0+385          IF(IDTH.NE.2HPW) GO TO 20
0+386          GO TO (11,12,12,12,12,16),ICR
0+387    11    MARK=0
0+388          GO TO 17
0+389    12    MARK=2
0+390          GO TO 17
0+391    16    MARK=10
0+392    17    CALL PLOT(X,Y,1,MARK)
0+393          GO TO 60
0+394    20    IF(IDTH.NE.2HVW) GO TO 21
0+395          MARK=24
0+396          GO TO 17
0+397    21    IF(IDTH.NE.2HSE) GO TO 22
0+398          MARK=19
0+399          GO TO 17
0+400    22    IF(ICR.NE.6) GO TO 23
0+401          MARK=10
0+402          GO TO 17
0+403    23    MARK=8
0+404          GO TO 17
0+405    30    IF(ISPEC.NE.2HST) GO TO 35
0+406          IF(IDTH.NE.2HPW) GO TO 31
0+407          CALL PLOT (X,Y,1,28)
0+408          MARK=2
0+409          GO TO 17
0+410    31    MARK=28
0+411          GO TO 17
0+412    35    IF(ISPEC.NE.2HSN) GO TO 40
0+413          IF(IDTH.NE.2HPW) GO TO 36
0+414          MARK=26
0+415          GO TO 17
0+416    36    MARK=18
0+417          GO TO 17
0+418    40    IF(ISPEC.NE.2HOD) GO TO 45
0+419          IF(IDTH.NE.2HPW) GO TO 41
0+420          CALL PLOT (X,Y,1,14)
0+421          MARK=1
0+422          GO TO 17
0+423    41    MARK=14
0+424          GO TO 17
0+425    45    MARK=22
0+426          GO TO 17
0+427    60    RETURN
0+428          END

 NO ERRORS FOR XYPLOT
 LENGTH OF SUBPROGRAM      00372
 LENGTH OF COMMON SPLIT    00002
 RUN
```

APPENDIX 3.

MAPS OF DISEASE AREAS FOR CURRENT AND PREVIOUS STANDS

Key to maps.

Compass point.

Current stand Area of Concentrated Mortality.

Current stand Area of Standing Infection.

Previous stand Area of Concentrated Mortality.

Previous stand Area of Standing Infection.

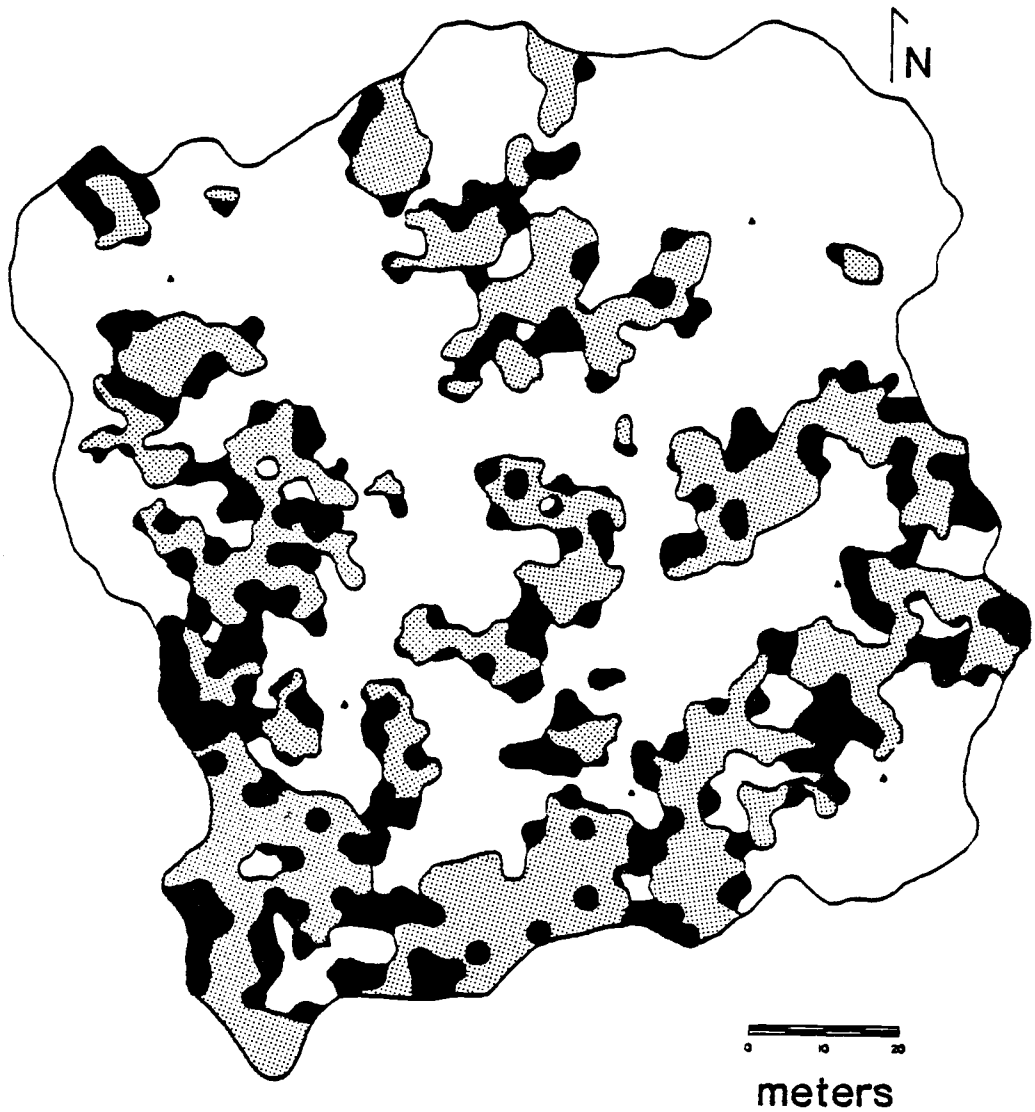
Previous stand Area of Potential Inoculum.

a — Location of current stand P. weirii isolate: same "clone" designated by same letter.

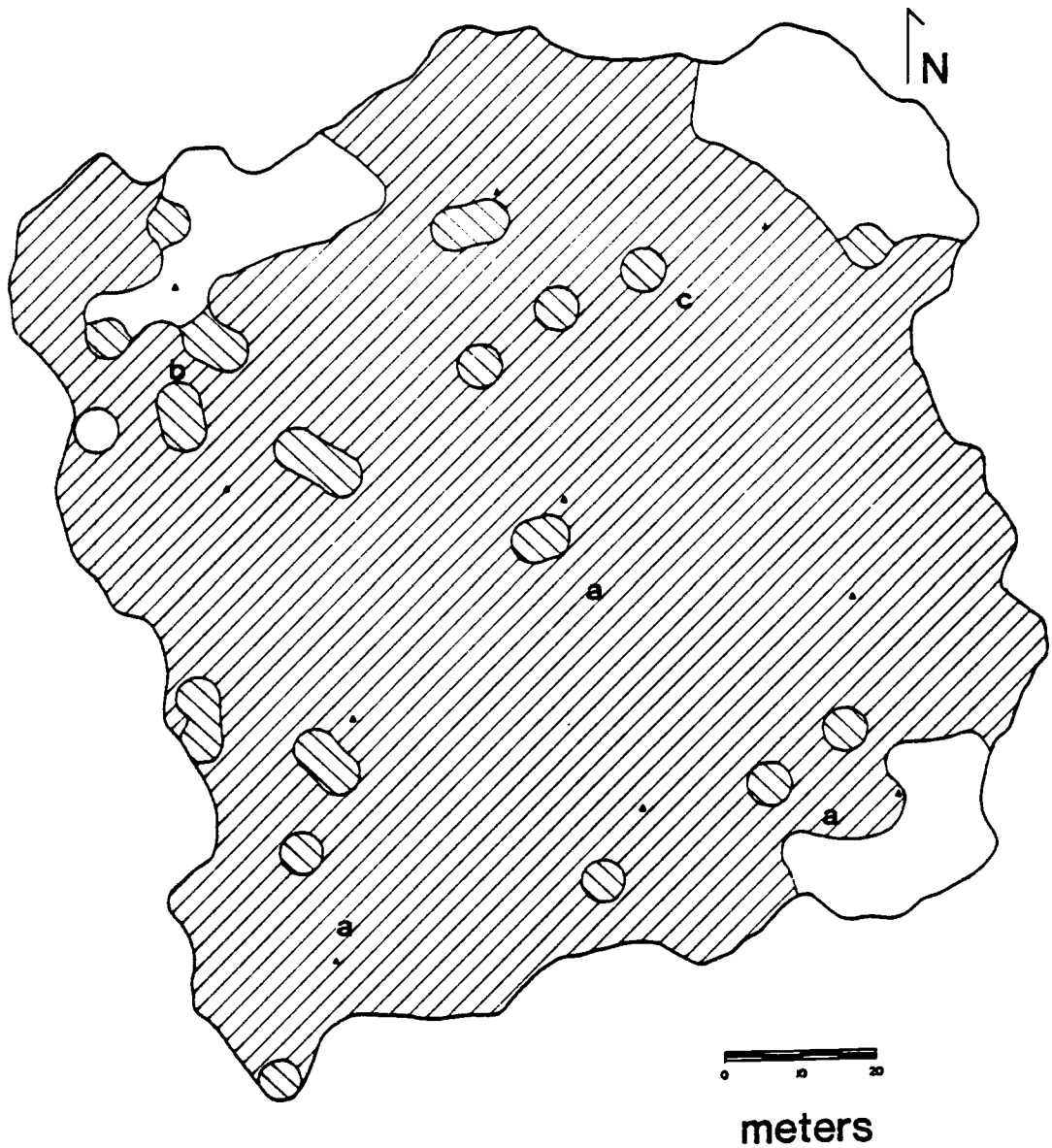
A — Location of previous stand P. weirii isolate: same "clone" designated by same letter.

? — Location of isolate of unknown compatibility.

WC2
Current stand

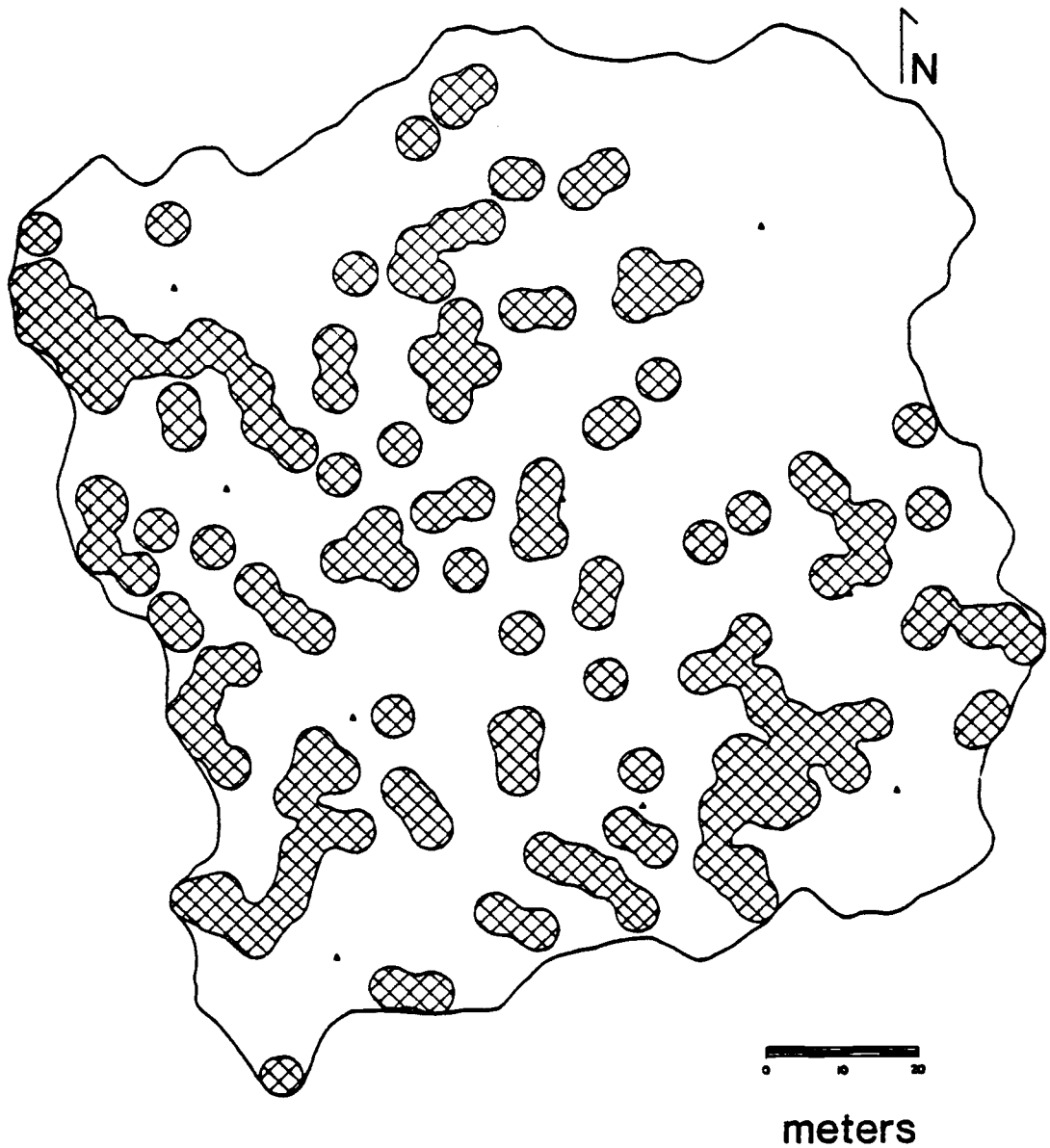


WC2
Previous Stand

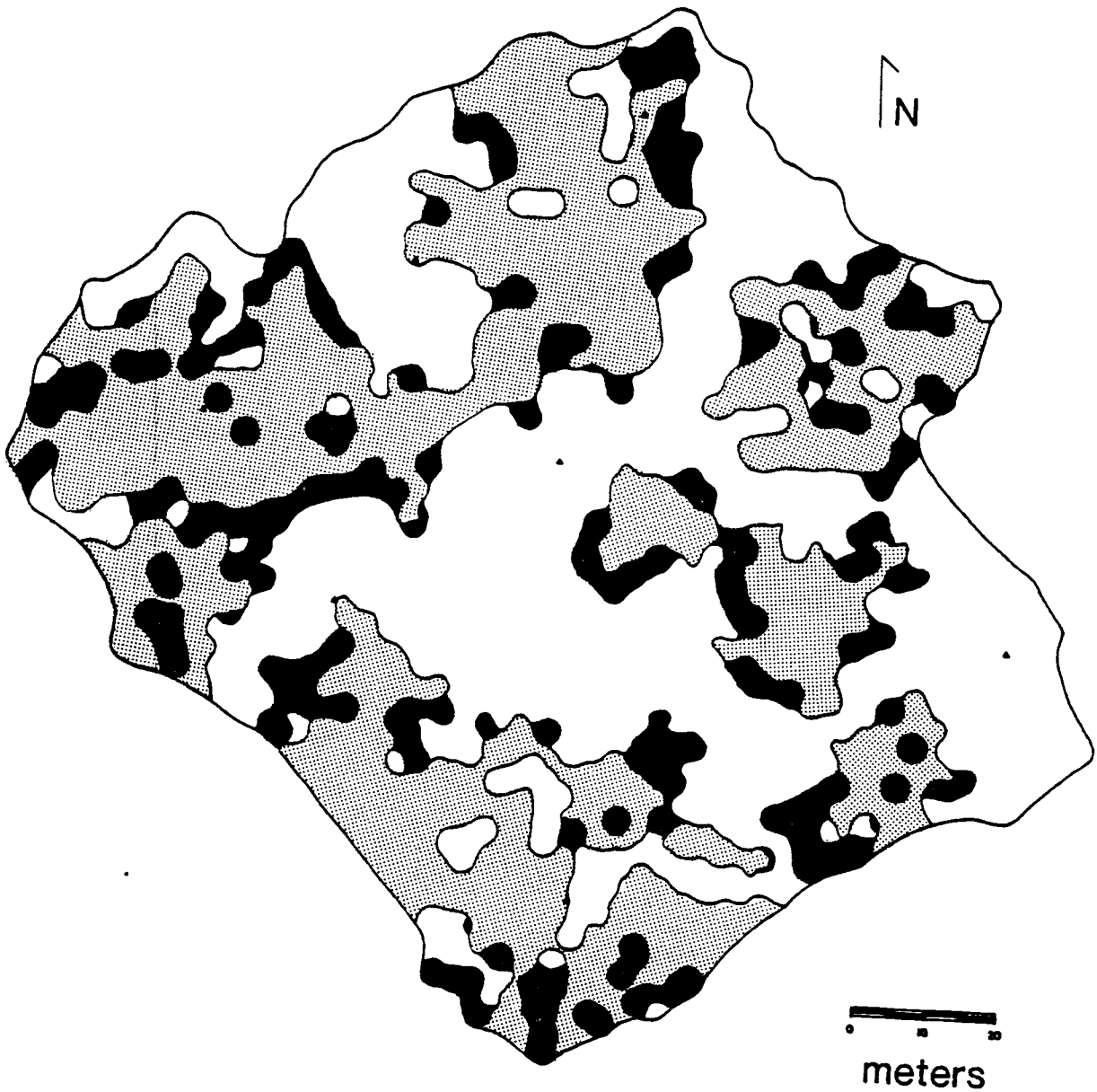


WC2

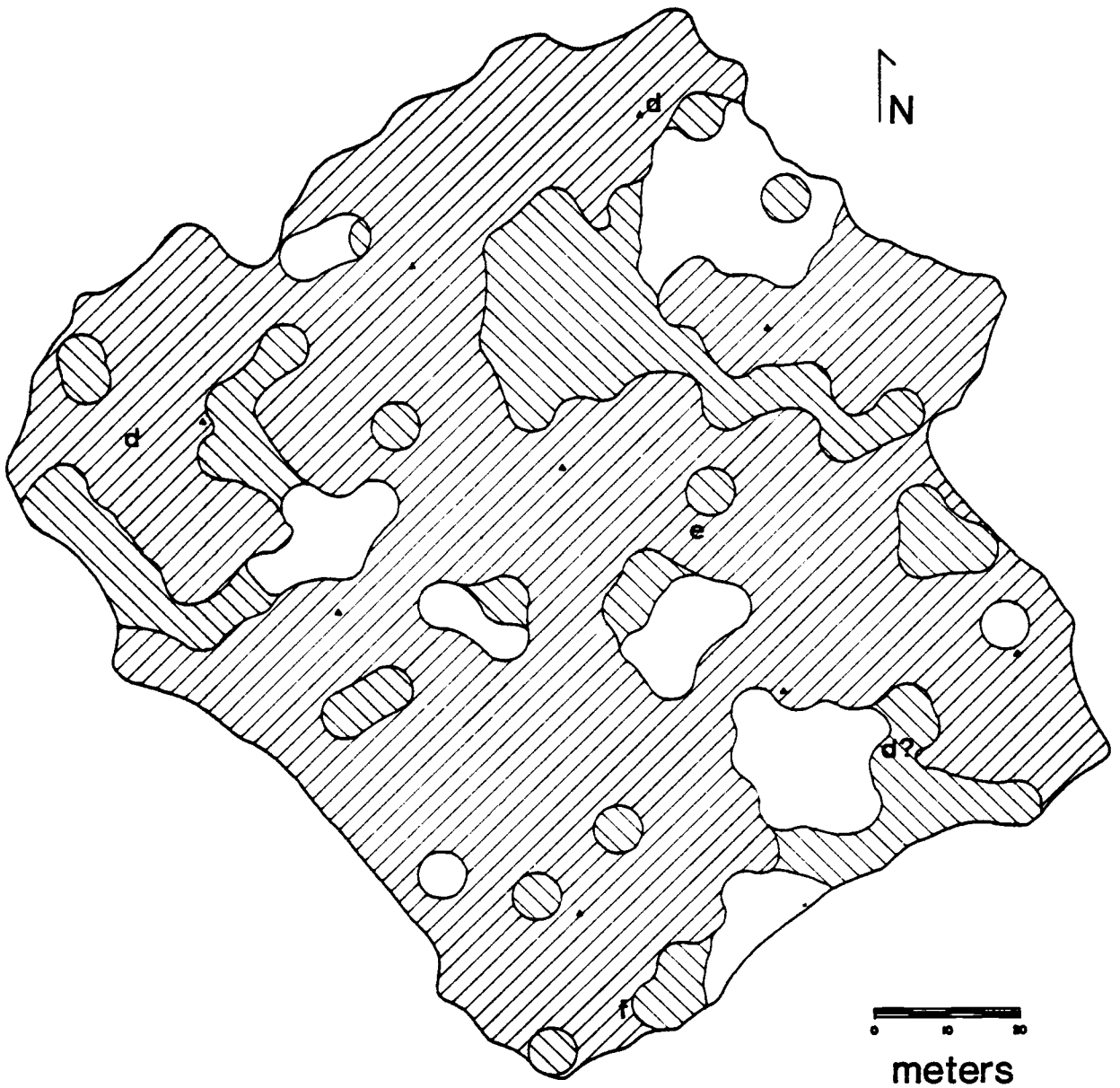
Previous Stand



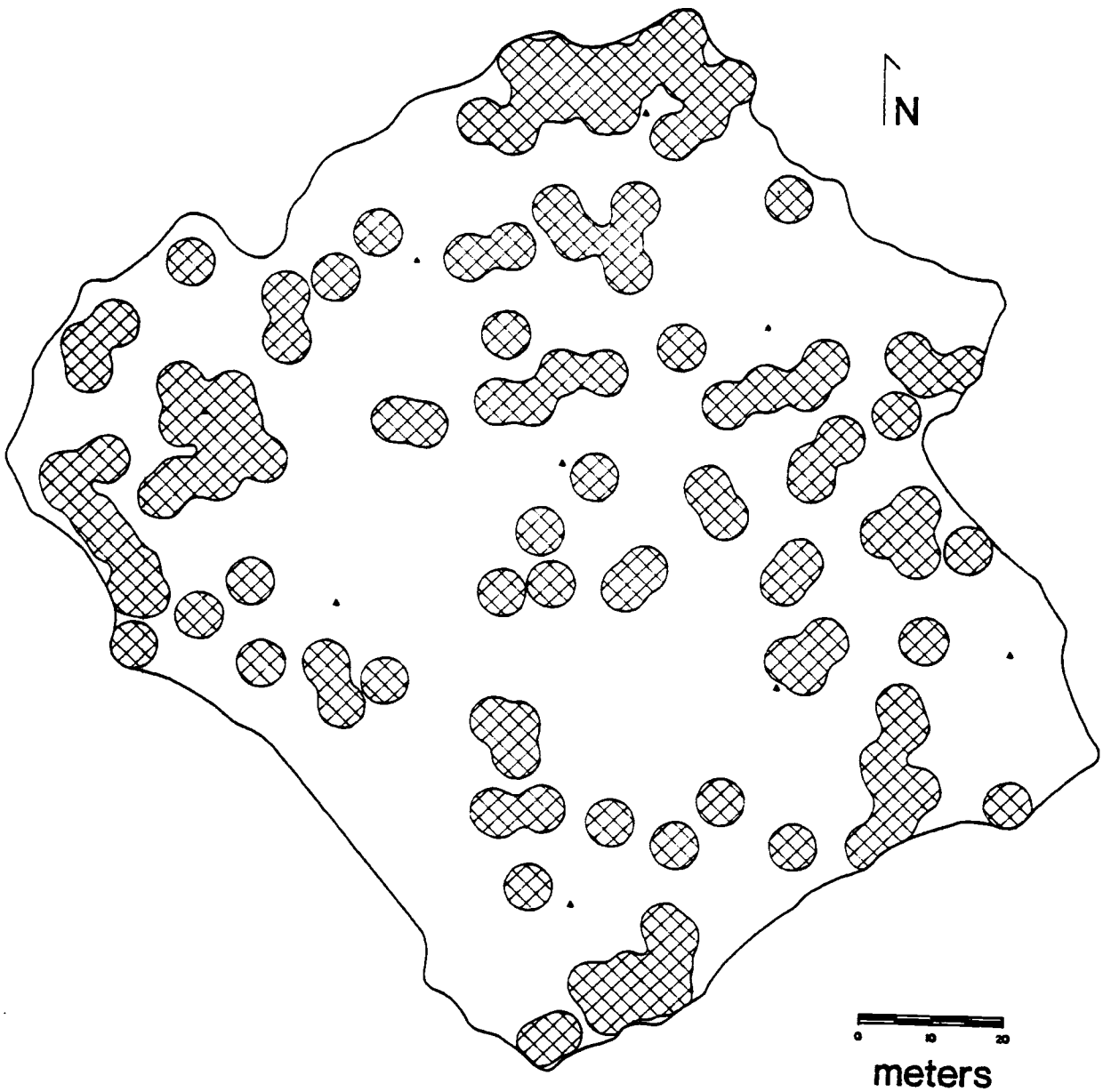
WC3
Current Stand



WC3
Previous Stand

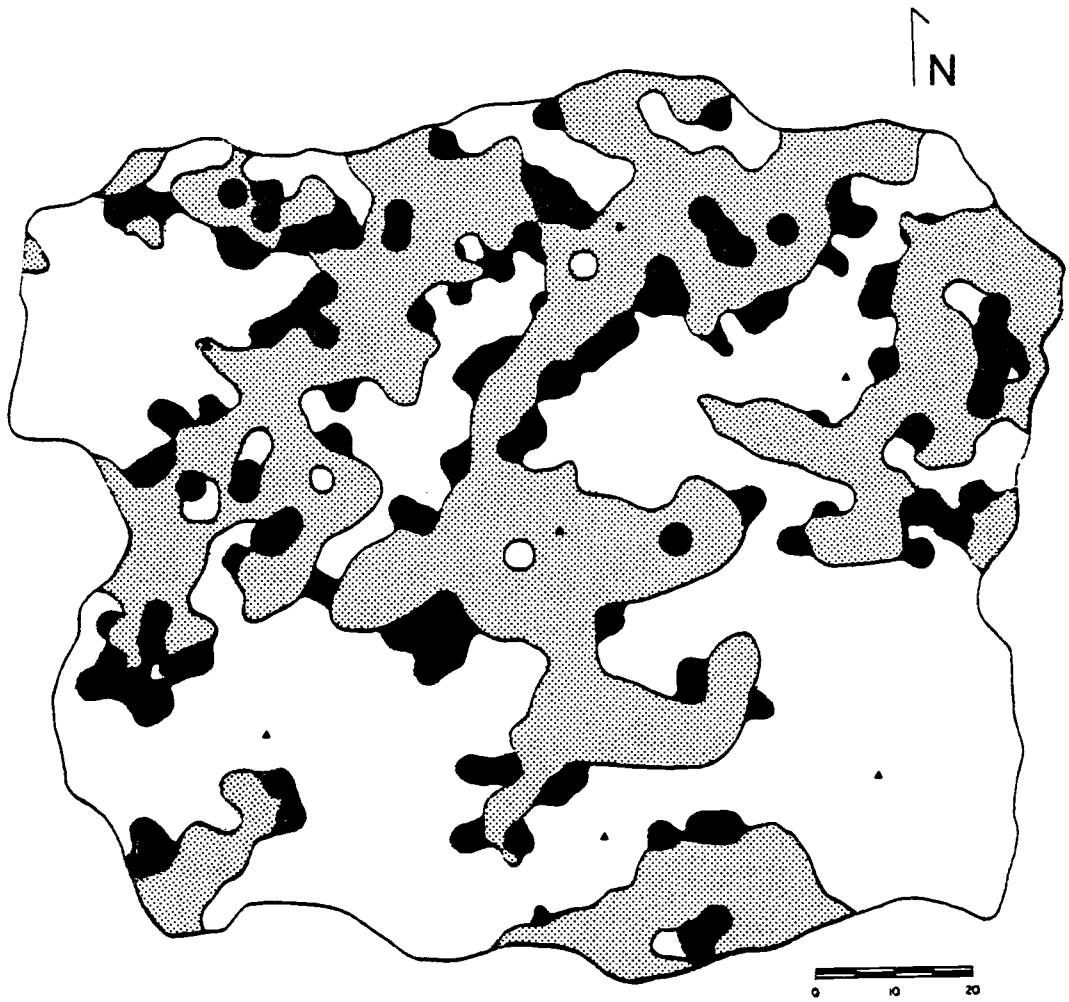


WC3
Previous Stand

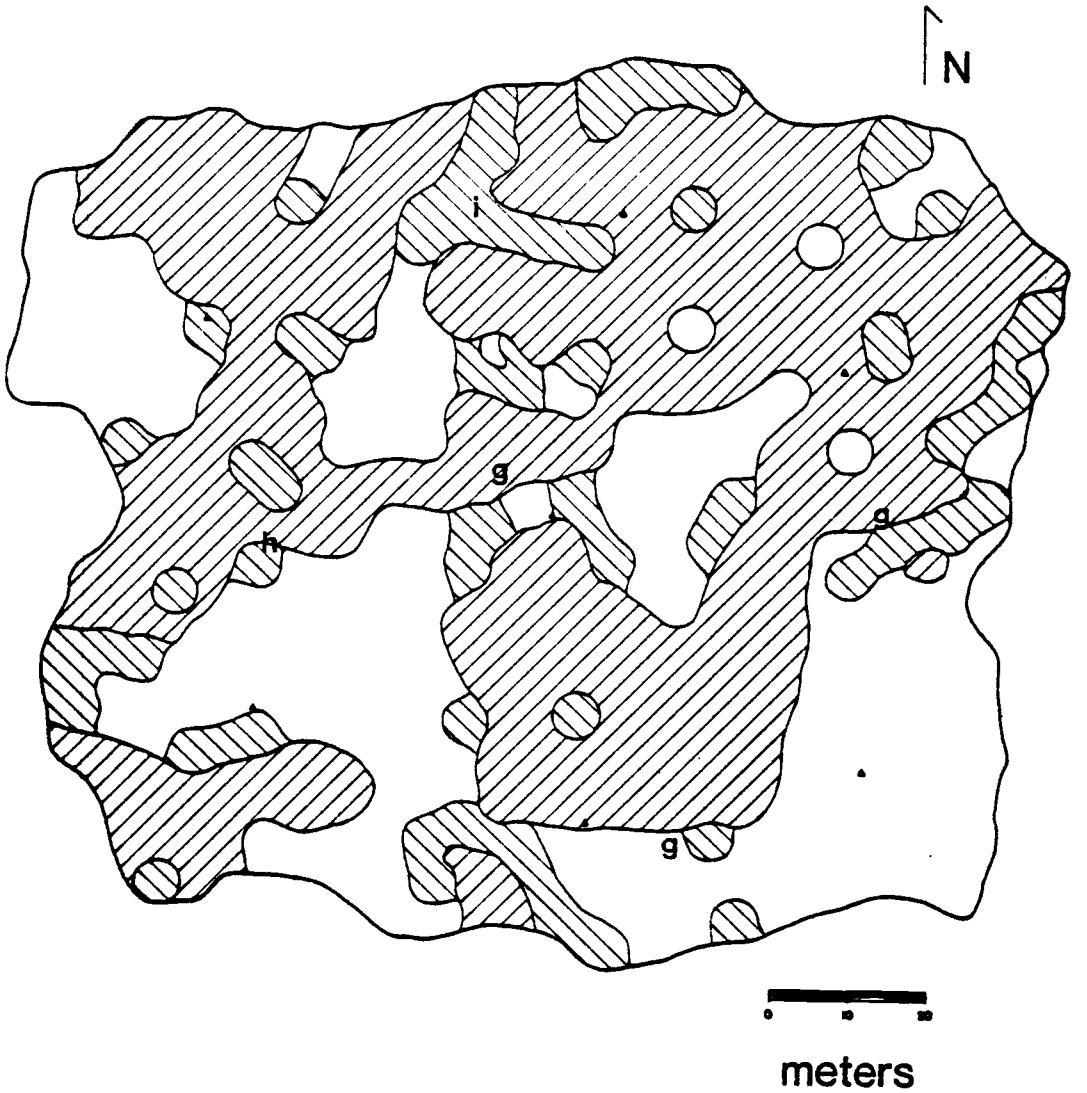


WC4

Current Stand

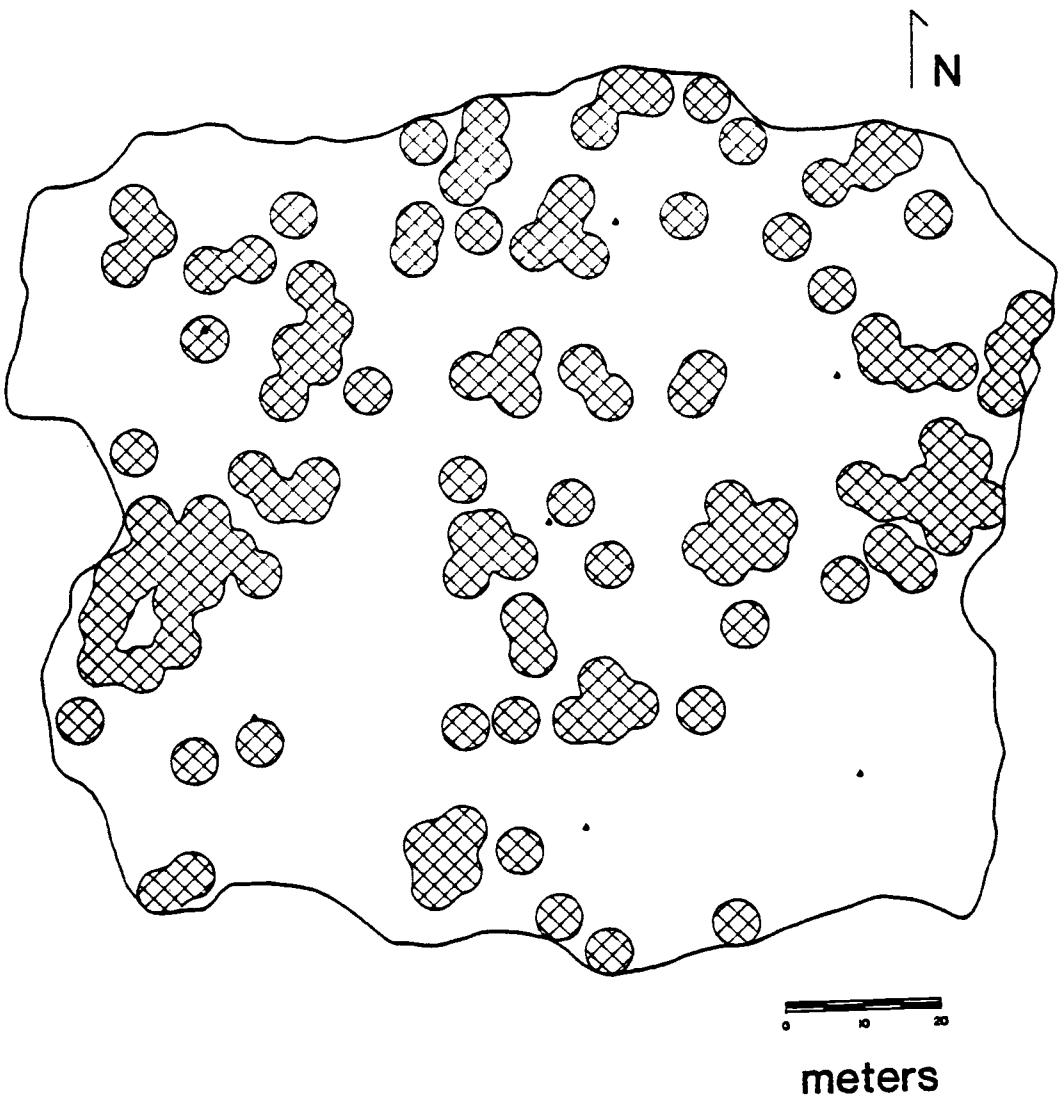


WC4
Previous Stand



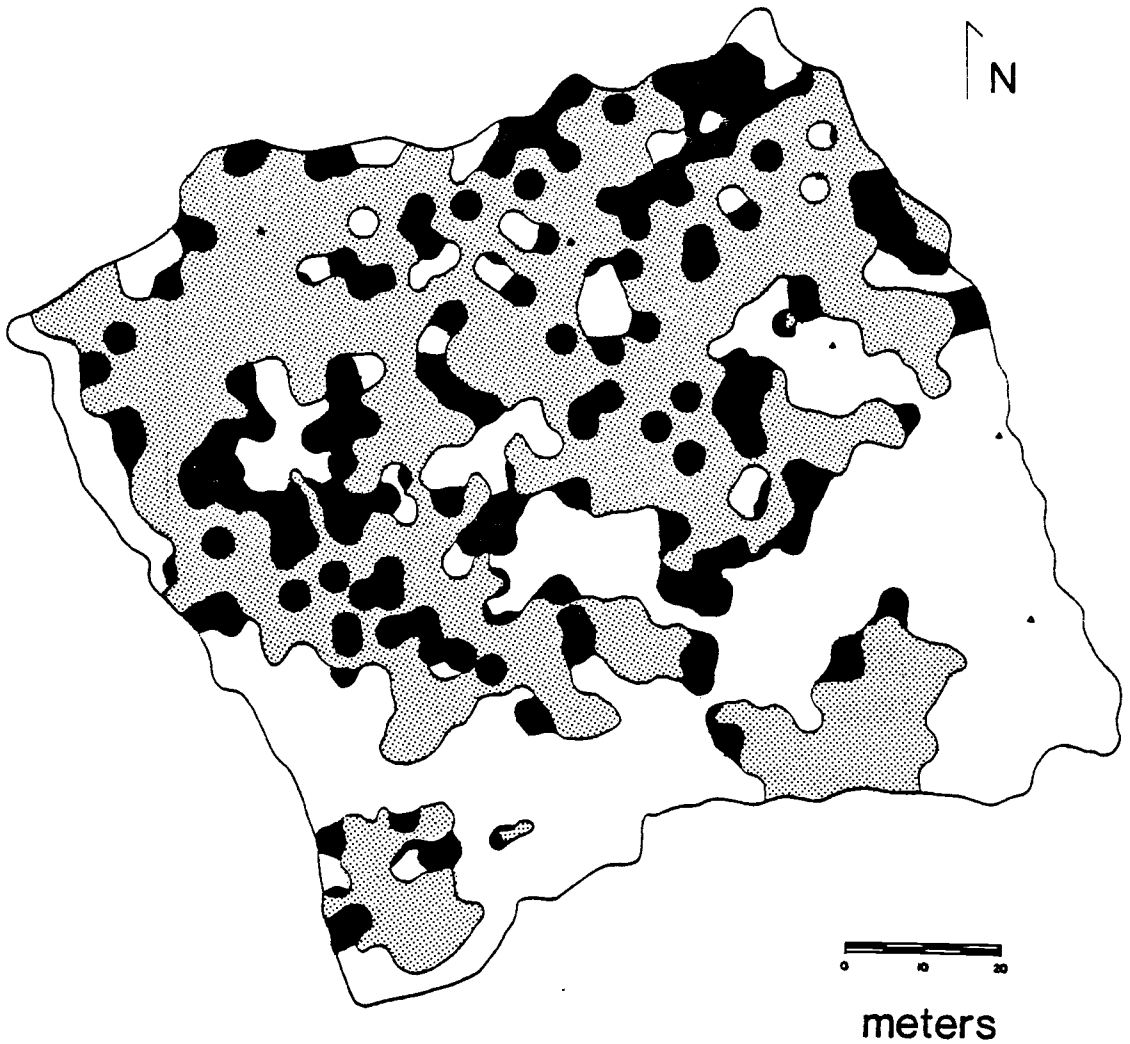
WC4

Previous Stand

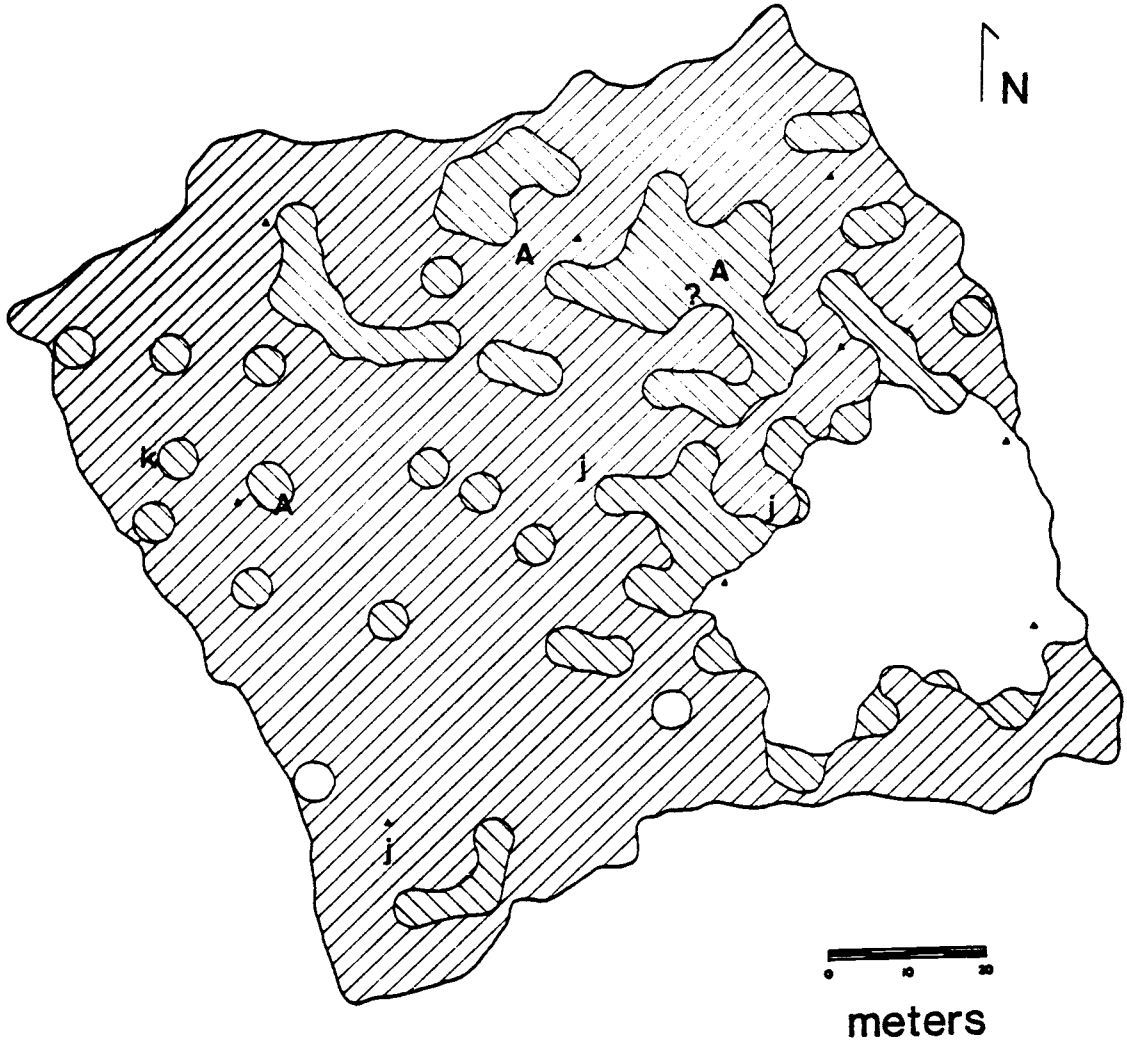
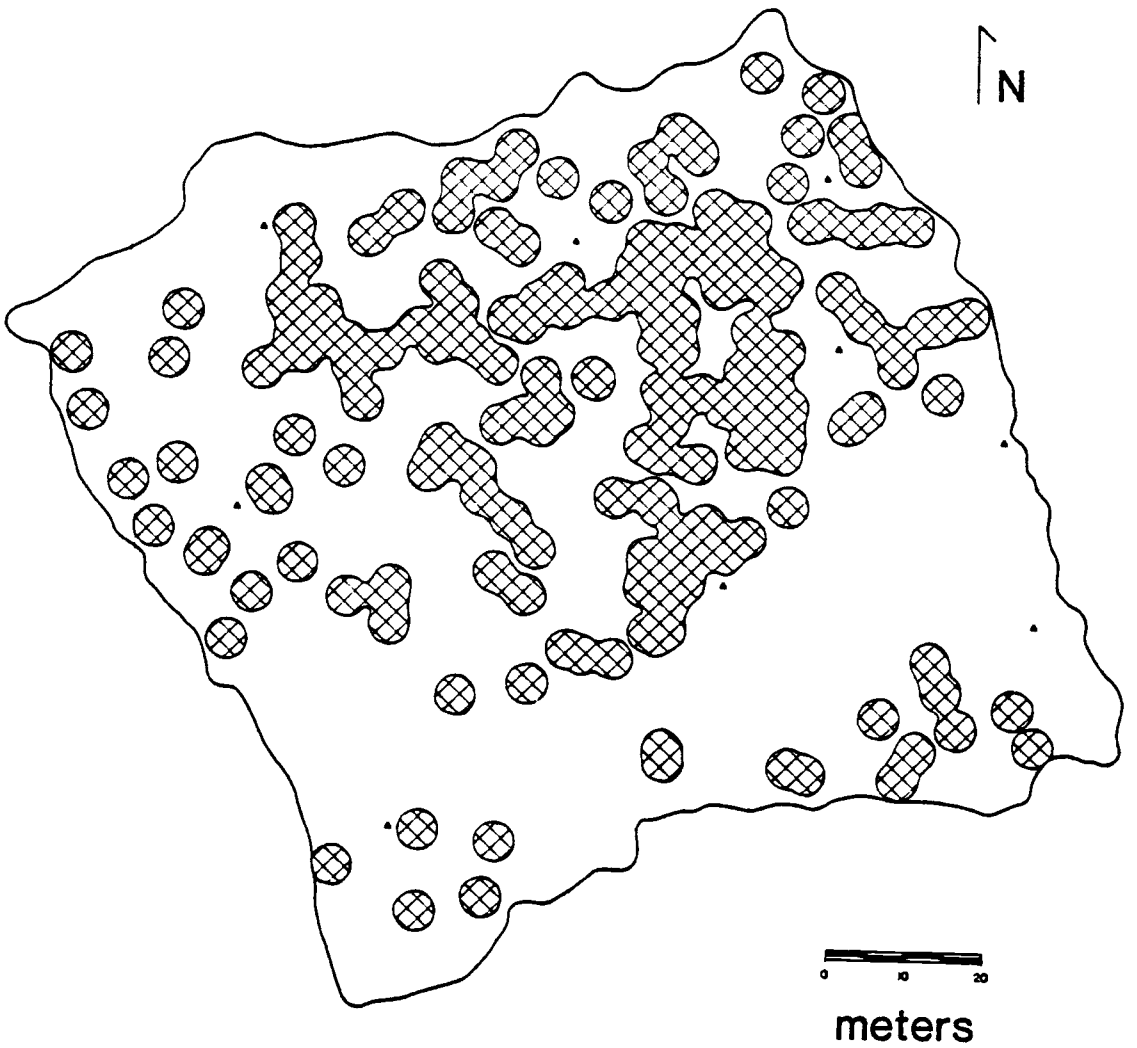


WC5

Current Stand



WC5
Previous Stand



WC5
Previous Stand