

Pathogens, Patterns, and Processes in Forest Ecosystems

Pathogens influence and are influenced by forest development and landscape characteristics

John D. Castello, Donald J. Leopold, and Peter J. Smallidge

Although pathogens are regarded as agents responsible for the specific dynamics of natural forest communities (Dickman 1992, Dinoor and Eshed 1984, Haack and Byler 1993, van der Kamp 1991), they have received little attention at the landscape level, especially compared to catastrophic abiotic disturbances, such as fire blowdowns (Foster and Boose 1992, Heinselman 1973). With so much emphasis today on ecosystem management and the maintenance of natural disturbance regimes, the role of pathogens deserves careful scrutiny. Pathogens (biotic agents that incite disease) differ from many abiotic disturbances by selectively eliminating the less vigorous or genetically unfit individuals of a population, yet the biotic and abiotic agents are similar in that both function to recycle essential elements and to alter forest development and landscape patterns. Pathogen interaction with abiotic disturbance to control the direction and rate of forest succession also has received minimal attention, although such

John D. Castello is a professor and Donald J. Leopold is an associate professor in the College of Environmental Science & Forestry, State University of New York, 1 Forestry Drive, Syracuse, NY 13210. Peter J. Smallidge is an assistant professor in the Program for Applied Ecology & Conservation Biology, Department of Biology, Frostburg State University, Frostburg, MD 21532. © 1995 American Institute of Biological Sciences.

Pathogens can confound forest management objectives

interactions can become important especially when the landscape is modified.

How do pathogens, which are present in all forest ecosystems, influence forest dynamics? They exert their influence primarily through tree mortality, which can occur at both the broad or large scale (forest development) and fine or small scale (gap phase). In this article, we present the hypothesis that pathogens regulate, and in turn are regulated by, patterns and processes in forest ecosystems. We discuss the impacts of pathogens on plant species distributions, forest structure and composition, succession, biodiversity, and landscape pattern. In addition, we discuss how land-use history and subsequent landscape pattern promote disease development. We conclude with a model that attempts to integrate the interaction between pathogens, mortality, and forest development at both the small and large scale.

Species distributions and abundance

Pathogens are effective in controlling species occurrence (Dinoor and Eshed 1984), particularly in the regeneration layer (Grubb 1977). For

example, in a study conducted in Panama, the location of seedling survival of six tree species was greatly influenced by root pathogens. Pathogens caused the large proportion of deaths among shade seedlings of these species, but disease-induced mortality was significantly reduced in light gaps (Augspurger 1984).

The local and regional distribution of eucalypt (*Eucalyptus* spp.) species vary as a result of their susceptibility to root rot caused by *Phytophthora cinnamomi*. In moist soils, this fungus kills susceptible eucalypt species, favoring the establishment and growth of more resistant species. As a result, susceptible eucalypt species tend to occur on drier ridges and resistant species on the moist depressions and swales (Burdon and Shattock 1980).

Brunchorstia pinea, the causal agent of Scleroderma canker of pine, affects Corsican pine (*Pinus maritima*) in northern England most seriously on north-facing slopes (Read 1968). Because the fungus is favored by cold temperatures, infection and mortality tend to occur in pine growing in topographic depressions or openings in the forest canopy where cold air accumulates (Manion 1991).

White pine blister rust, caused by the fungus *Cronartium ribicola*, affects the local distribution of eastern (*Pinus strobus*) and western white pine (*Pinus monticola*). Successful infection of pine, a few weeks of cool, late summer weather including 48 hours of 100% relative

humidity and 68°F, are required. Thus, disease dominates where weather and prevention of white pine. Climatic influences and management has enabled determination of a site-habitat should not be predicted.

Forest structure and composition

A dramatic example of impact is provoking blight, caused by the fungus *Cryphonectria* American chestnut (*Castanea americana*) was a dominant species in the forest throughout much of America until it was killed by the forest canopy blight fungus during the mid-1900s. Many of the changes in species composition and structure in the forest are dominated by chestnut disease (Keever and Sivec 1973, McCune 1980, Stephenson 1980). In current stands, the loss of tree species diversity before the disease; American chestnut had few competitors. However, the species richness of chestnut vary with no single species dominant/codominant. Occupied by chestnut (1986). It is still to be seen the full impact of the changes in forest composition have resulted because some authors (e.g., Platt 1980) suggest that forests eventually dominated by chestnut.

Fungal root diseases caused by those diseases caused by those species and *Phellinus* spp. in the Pacific Northwest, composition, and structure to the extent that communities affected are referred to as "white pine blight" (van der Kamp 1991). Pathogens spread

humidity and temperatures below 68°F, are required (Manion 1991). Thus, disease and mortality predominate where these conditions are met and prevent the establishment of white pine. Knowledge of climatic influences on disease development has enabled the implementation of a site-hazard rating system to determine where white pines should not be planted.

Forest structure and composition

A dramatic example of pathogens' impact is provided by chestnut blight, caused by the introduced fungus *Cryphonectria parasitica*. American chestnut (*Castanea dentata*) was a dominant or codominant species in forest communities throughout much of eastern North America until it was eliminated in the forest canopy by the chestnut blight fungus during the early to mid-1900s. Many studies document the changes in species composition and structure in former chestnut-dominated communities after this disease (Keever 1953, Mackey and Sivec 1973, McCormick and Platt 1980, Stephenson 1986). Typically, current stands have a greater richness of tree species compared to stands before they were affected by disease; American chestnut generally had few codominants. However, the species replacing American chestnut vary with location because no single species has assumed the dominant/codominant role once occupied by chestnut (Stephenson 1986). It is still too early to evaluate the full impact of this disease and the changes in forest dynamics that have resulted because of it, although some authors (e.g., McCormick and Platt 1980) suggest that oak-hickory forests eventually would replace forests once dominated by American chestnut.

Fungal root diseases, particularly those diseases caused by *Armillaria* spp. and *Phellinus weirii* in the Pacific Northwest, can alter structure, composition, and community pattern to the extent that forest communities affected by these diseases are referred to as "root-disease climates" (van der Kamp 1991). Root pathogens spread locally and result



Ash yellows, a bacterial disease of white ash, is thinning stands at Gettysburg National Historic Park. Photos: D. J. Leopold.

in an expansion of canopy gaps (Bloomberg and Reynolds 1985). In the interior forests of British Columbia the pioneer species, Douglas-fir or lodgepole pine (*Pinus contorta*), are killed by *Armillaria ostoyae*, and the gaps become filled with the more disease- and shade-tolerant western hemlock (*Tsuga heterophylla*), western red cedar (*Thuja plicata*), or subalpine fir (*Abies lasiocarpa*; Shaw and Kile 1991). Likewise, the number of conifer species is greater within stands infested by *P. weirii* than in the surrounding uninfested stands, stand age and tree biomass are lower, soil temperatures are higher, and local moisture patterns are altered (Boone et al. 1988). These conditions delay

subsequent tree colonization.

Partial cutting and fire suppression enhance *Armillaria* root disease, which then alters species composition. In the dry mixed-conifer forests of western North America, particularly where ponderosa pine (*Pinus ponderosa*), western white pine, and western larch (*Larix occidentalis*) dominate, selection logging and fire control favor the regeneration of the more root-rot susceptible Douglas-fir (*Pseudotsuga menziesii*) and true firs (*Abies* spp.; Byler et al. 1990, Kile et al. 1991). Likewise, the introduction and spread of white pine blister rust into the northern Rocky Mountains have enhanced mortality by *Armillaria* (Byler et al. 1990). The rust epidemic simulated

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a partial cut by killing western white pine in many forest stands and modifying succession by reducing pine regeneration. This outcome resulted in a shift in species composition from the *Armillaria*-tolerant white pine to the *Armillaria*-susceptible Douglas-fir and grand fir (*Abies grandis*).

Beech bark disease, caused by an interaction between a scale insect, *Cryptococcus fagisuga*, and the canker fungus, *Nectria coccinea*, has had significant impacts on American beech (*Fagus grandifolia*) and northeastern US forest structure and composition. Runkle (1990) documented the changes occurring in just eight years in a mature eastern hemlock (*Tsuga canadensis*) stand in New York affected by beech bark disease. Mortality of American beech was clumped owing to the limited dispersal abilities of the causal agents (Houston et al. 1979), and the surviving beech stems were more regularly dispersed than those in predisease stands (Runkle 1990). On typical hemlock sites, the disease associated with an increase in abundance of hemlock and decreases in the abundance of American beech and yellow birch (*Betula alleghaniensis*; Runkle 1990, Twery and Patterson 1984).

Root rot caused by *P. cinnamomi* has a dramatic effect on structure and composition of the eucalypt forests of the Brisbane Ranges, Victoria, Australia (Weste 1986). The affected forest community changed from open forest with a sclerophyllous understory to one with large gaps dominated by sedges, graminoids, and legumes. On pathogen-free sites tree density increased by 10% from 1975 to 1985, whereas on diseased sites density decreased by 43% (Weste 1986). Susceptible eucalypt species died and resistant species increased. The fate of many rare, endemic eucalypt species is of great concern.

Interacting parameters

The impact of a pathogen on forest structure and composition often is dependent upon many interacting parameters. Menges and Loucks (1984) attempted to predict the level of spread, mortality, and commu-

nity response to oak wilt, caused by the fungus *Ceratocystis fagacearum*, in Wisconsin. Predicted differences in oak mortality were attributed primarily to the efficiency with which the insect vector transmits propagules of the pathogen and less to the amount of inoculum from outside the plot and size of the woodlot. Response was predicted to be extremely variable and dependent upon the initial forest composition. Advanced tree regeneration present during the disease was favored by the loss of red (*Quercus rubra*) and black (*Quercus velutina*) oaks. White oak (*Quercus alba*), black cherry (*Prunus serotina*), and sugar maple (*Acer saccharum*) typically were favored, depending on community type. For example, on mesic sites, oak wilt accelerated succession to forests dominated by sugar maple.

The age and size-class structures and species composition of swamp forests in the US Great Lakes area and Central States, which contain canopy-sized American elms (*Ulmus americana*), have been greatly altered during the past 50 years, at least in part, by Dutch elm disease and/or elm yellows (Parker and Leopold 1983). Elm yellows exacerbates Dutch elm disease by providing additional breeding material for beetles. Elm yellows was first described in 1880, when many elms began to die in Kentucky, Indiana, and Illinois (Garman 1882). Since then, elm yellows and Dutch elm disease have occurred together in the Northeast and Midwest (Lanier et al. 1988).

An increase in the importance of shade-tolerant species due to the death of elms was noted in an Illinois forest where a 60% increase in the basal area of sugar maple was observed (Bogges and Bailey 1964). In an Indiana woods, the death of mature elms resulted in a threefold increase in juvenile elm density between 1926 and 1976 (Parker and Leopold 1983). In a central New York forest American elm did not replace itself in any significant amount (Huenneke 1983). This lack of elm replacement may have been caused by elm yellows, which was epidemic in this region at the time (Lanier et al. 1988).

The density of dead canopy elm affects the community response. Where the density of dead elms was greater than 5 stems/ha, shrub density in southern Wisconsin lowland forests increased significantly. Below this critical density, no community response was detected (Dunham 1986). Lowland forests suffering moderate to severe elm mortality following Dutch elm disease may become shrub dominated. Mortality of American and slippery elm (*Ulmus rubra*) attributed to Dutch elm disease has led to a doubling of the importance value (the average of relative basal area plus relative density plus relative frequency) of white ash (*Fraxinus americana*) through an increase in the abundance of white ash stems (Levenson 1980). White ash has become a major component of second-growth forests in southeastern Wisconsin (Levenson 1981).

Formation of canopy gaps

Diseases play an important role in the dynamics of old-growth forests through the formation of canopy gaps. In mature and old-growth forests, pathogens create a spatially heterogeneous matrix of susceptible and resistant species. In a 350-year old mountain hemlock (*Tsuga mertensiana*) forest in the Oregon Cascades, Cook (1982) determined that stands infested with *P. weirii* for 300 years still contain susceptible species such as hemlock and Pacific silver fir (*Abies amabilis*). However, these species were interspersed amongst patches of the more resistant lodgepole and western white pines and Noble fir (*Abies procera*). The frequency of resistant species has increased and overall stand density has decreased in comparison to noninfested stands. The resultant change in species richness may in turn suppress the rate of mycelial advance within infested stands (McCauley and Cook 1980). Patch-phase processes of disturbance followed by gap colonization maintain a greater number of species, uneven-aged stands, and vegetation mosaics (Worrall and Harrington 1988). In the spruce-fir forests of Crawford Notch, New Hampshire, biotic diseases ac-

counted for 6% of the lowest elevation and butt rot gap makers. Balsam fir is very susceptible to pathogens, and this species at Harrington 1981.

Pathogen impact on site conditions

Pathogens impact site conditions, with significant impact on young mature generating in centers often a tion, whereas (by the fungus. (1984) recorded in nitrogen mineral ability in a mou following dieback. They postulate nitrogen availability assistance of mou *weirii* until nitrogen limiting, at which hemlock again t. In a concurrent controlled condition Waring (1984) nitrogen nutrition site the extent of foliar loss in mountain inoculated with shading increase of the seedling. Matson and Waring increased nitrogen following fungus increase the resin lock regeneration mediate reinfecting stands.

Effect on animal diversity

Tree pathogens impact animal diversity. Structural changes resulting from dieback alter habitats (1987). Both bird diversity and number of bird species related to the intensity of dieback (Arceuthobium

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Pathogen impact on stand and site conditions

Pathogens impact stand and site conditions, which underlie pathogen impact on the forest community. Young mountain hemlock regenerate in *P. weirii* infection sites, whereas older trees are killed by the fungus. Matson and Boone (1984) recorded a twofold increase in nitrogen mineralization and availability in a mountain hemlock stand following dieback due to *P. weirii*.

They postulated that increased nitrogen availability increases the resistance of mountain hemlock to *P. weirii* until nitrogen again becomes limiting, at which point mountain hemlock again becomes susceptible. In a concurrent study under controlled conditions, Matson and Waring (1984) reported that nitrogen nutrition significantly reduced the extent of foliage damage and loss in mountain hemlock seedlings inoculated with *P. weirii*, whereas shading increased the susceptibility of the seedlings to the fungus. Matson and Waring postulated that increased nitrogen and light levels following fungus-induced mortality increase the resistance of the hemlock regeneration and prevent immediate reinfection of the regenerating stands.

Effect on animal species

Tree pathogens indirectly affect the richness and diversity of animal species. Structural changes in the forest resulting from diseases can affect animal habitats (Franklin et al. 1987).

Both bird diversity and the number of bird species were directly related to the intensity of dwarf mistletoes (*Arceuthobium* spp.) in Colorado



Forest regeneration within gap beneath an American beech that was killed by beech bark disease in the Adirondack Park, NY.

ponderosa pine forests (Mlot 1991). Similarly, approximately half of the nesting sites of the endangered northern spotted owl (*Strix occidentalis*) are in dwarf mistletoe brooms (Irwin et al. 1989). In addition, the number of mule deer (*Odocoileus hemionus*) and elk (*Cervus elaphus*) pellets were higher in affected versus nonaffected stands (Bennetts et al. 1991). In the Southeast, the red-cockaded woodpecker (*Picoides borealis*) constructs its nesting cavity in living southern pines, particularly those with heartrot (Walters 1991). Conversely, chestnut blight and beech bark disease in the eastern United States presumably have greatly lowered mast (nuts used as food by wildlife) production, lowering the diversity

of wildlife species until other species provide a replacement food source.

Land-use history, management practices, and disease

Land-use history has an impact on disease development. For example, in central New York, extensive forest lands were cleared for agriculture, so that by 1930 only 8% of the land was forested and coverage was fragmented. However, extensive land abandonment and subsequent reforestation increased the forest cover to 40% by 1980 (Nyland et al. 1986). Yet, unlike the original forest cover, these forests are aged, structurally homogeneous,

called islands with increased edge and lower species richness occur within a heterogeneous landscape. White ash is a scattered tree species in undisturbed eastern deciduous forests (Braun 1950, Burns and Honkala 1990), but it is one of the most abundant species following land abandonment (Zipperer et al. 1990). The increased incidence of white ash decline since 1930 may be the result of a more homogeneous, even-aged host population (Smallidge et al. 1991b).

Ash yellows, a bacterial disease of white ash and the principal causal agent of ash decline (Matteoni and Sinclair 1985), may function as a natural thinning agent in white ash stands of old-field origin. The disease may accelerate forest development beginning at stem exclusion, where trees compete with one another for available resources leading to mortality of the less competitive individuals (Oliver 1981). Mortality, growth decline, and ash yellows are most common in stands at this stage of development (Han et al. 1991, Smallidge et al. 1991b). The disease may accelerate forest development until it reaches the understory reinitiation stage.

In the northeastern United States, white ash stands that developed on abandoned agricultural fields in the early 1900s have reached the stem-exclusion stage of forest succession. The population of trees susceptible to ash yellows is expected to diminish over time due to the effects of disease, competition, and the coalescence of forest islands.

Ash yellows, drought, stand characteristics, landscape pattern, and radial growth of white ash are interrelated (Smallidge et al. 1991a). However, the relationship between ash yellows and landscape pattern is more speculative. Forest islands with dense stands of young white ash are not contiguous with larger mature forests, and so they may be more sensitive to drought, more susceptible to ash yellows, or closer to potential vectors of the pathogen. In addition, the inadvertent establishment of biological corridors (i.e., the network of abandoned agricultural fields) could enhance vector movement, thus corridors may be involved in the increased incidence

of ash yellows in fragmented forest landscapes.

The littleleaf disease of shortleaf pine (*Pinus echinata*), caused in part by *P. cinnamomi*, in the Piedmont of the southeastern United States has been exacerbated by changing land-use history, species composition, and forest type. The rolling hills of this region were cleared, cropped, and abandoned several times between 1700 and the 1930s. Each wave of cropping and abandonment facilitated soil erosion and resulted in a thin topsoil covering clay subsoils with poor internal drainage, low fertility, and limited aeration. Shortleaf pine seeded into abandoned fields and much of the land reverted to pine, although the natural vegetation of the Georgia Piedmont is oak-hickory-pine forest (Turner 1990). Fire suppression allowed the trees to become established (Oak and Tainter 1988). As the trees aged on these poor sites they became susceptible to attack by *P. cinnamomi*, which ultimately killed them. Since the 1920s, planted and natural coniferous forests have increased on the Georgia Piedmont (Turner 1990) and elsewhere in the Southeast, and many occur on littleleaf-prone sites.

Management practices influence the effects of pathogens on forest structure and composition. During the last 100 years, fire suppression has resulted in forest stands in the Yosemite Valley of California with basal areas in excess of 110 m²/ha. This stocking level in association with drought, bark beetle attack, ozone stress, and annosus root rot (caused by the fungus *Heterobasidion annosum*) led to extensive mortality of ponderosa pine in the 1970s (Sherman and Warren 1988). Incense cedar (*Calocedrus decurrens*) was not affected, and it has replaced ponderosa pine as the dominant species. Sherman and Warren suggested that these forests may eventually revert to a patchwork of mixed conifers and open oaks, similar to that which existed prior to 1850.

Variation in fire frequency and forest management activities facilitate the development of otherwise inconsequential diseases. Fusiform rust, caused by the fungus *Cronartium fusiforme*, was of minor im-

portance until the natural distribution of native pine species was altered (Dinus 1974). In the nineteenth century, longleaf pine (*Pinus palustris*) was extensively logged in this region. Loblolly (*Pinus taeda*) and slash (*Pinus elliottii*) pines, which are more susceptible than longleaf pine to fusiform rust, colonized cutover longleaf pine sites. Loblolly and slash pines also were widely planted to replace longleaf pine because of their rapid early growth and ease of planting. Oaks, which are the alternate hosts of the fusiform rust fungus, also increased in abundance as longleaf pine was cut and fire-suppression policies became common (Dinus 1974). These activities resulted in an increase in fusiform rust, from a curiosity in 1930, to a major problem today in both plantations and natural forests in the southern United States.

In lodgepole pine forests of the Rocky Mountains, landscape features, disturbance, and stand structure and composition interact with the incidence and severity of dwarf mistletoe. Structural heterogeneity increases the rate of spread of dwarf mistletoe. Spread rates are fastest from overstory trees to adjacent younger and smaller trees, whereas spread through even-aged stands is much slower (Hawksworth and Johnson 1989). Likewise, spread is 1.5 times faster in stands in which the canopy has not closed than in stands with closed canopies.

Wildfires enhance regeneration of mistletoe-susceptible seral tree species such as lodgepole pine (Hawksworth and Johnson 1989). Dissimilarities in fire history may explain why dwarf mistletoe is more prevalent in the upper foothills than in the lower foothills of the Rocky Mountains of Alberta. During the past 100 years, wildfires were extensive and complete in the lower foothills; the lodgepole pine stands that developed in these burned areas are essentially free of mistletoe because of the stands' structural homogeneity and closed canopies. In the upper foothills, however, topographic and structural diversity prevented the development of large burns and promoted mixed size and age classes. Many mistletoe-infected trees survived and inoculated the

development (1972), why lodgepole pine is usually found on slopes (Hawksworth and Johnson 1989).

Fire caused an increase in mistletoe in lodgepole pine forests. In the 1930s, fire frequency peaked (1890 and 1930) and mistletoe incidence peaked (the point of maximum stand is in Laven 1990).

More than 50% of mistletoe in lodgepole pine near stands (Laven et al. 1988) is attractive to disseminating mistletoe (Hudler 1990) provide the same spruce (Hawksworth and Johnson 1989).

Where a progressive regeneration process is used for fire control, mistletoe inoculum level and structure of species (Laven et al. 1988) are primary parameters and understory of central and eastern Australia. In these forest stands, logging, all pathogens in forests, the severity of damage is related to the age of the older trees. Logging frequency and intensity appear to be a factor. Selective removal of trees from the inoculum (Kellas et al. 1990).

In eastern Australia, dieback and decline caused by *P. cinnamomi* severe after timber logging, wildfire (Laven et al. 1986). The impact of timber logging is affected by a combination of moisture and

atural distribu... developing regeneration (Baranyay species was al... (1972), which may partially explain n the nineteenth... why lodgepole pine dwarf mistletoe pine (*Pinus*... is usually more common on ridge sively logged... and slope sites than on bottom sites y (*Pinus taeda*... (Hawksworth 1958). *elliottii*) pines... Fire control, similarly, may have isceptible that... increased the amount of dwarf form rust, color... mistletoe in some lodgepole pine leaf pine sites... forests. In central Colorado, fire fre- pines also were... quency peaked between 1860 and eplace longleaf... 1890 and then declined, while dwarf er rapid early... mistletoe incidence has increased to planting. Oaks... the point today where nearly every ate hosts of the... stand is infested (Zimmerman and , also increased... Laven 1984). *ingleaf pine wa*... More than 66% of isolated dwarf- sion policies be... mistletoe infection-centers in a is 1974). These... lodgepole pine forest were located an increase in... stand openings (Hawksworth a curiosity is... et al. 1987). Such openings are an oblem today is... attractive habitat for birds, which l natural forest... disseminate seeds of dwarf mistle- ted States. ... toe (Hudler et al. 1974). Birds may e forests of the... provide the mechanism for long-dis- landscape fea... tance spread of the pathogen d stand struc... (Hawksworth et al. 1987). n interact with... Where *Armillaria* species are ag- verity of dwarf... aggressive root-rot pathogens, man- l heterogeneity... agment practices (e.g., logging and spread of dwarf... fire control) may affect disease ex- ates are faster... pression directly by impact on es to adjacent... oculum levels or indirectly by alter- : trees, where... ation of species composition or stand -aged stands... structure (Kile et al. 1991). *Armil- wksworth and*... *luteobubalina* is an aggressive ewise, spread... primary pathogen of many eucalypt stands in which... and understory species in the forests closed than in... of central and southwestern West- anopies. ... er Australia (Kile 1981). Most of regeneration... these forests have a long history of e seral tree spe... logging. Although this fungus is le pine (Hawk... pathogenic on eucalypts in unlogged 1989). Dissim... forests, the greatest incidence and ry may explain... severity of disease has been associ- : is more preva... ated with repeated selection-cutting oothills than i... of the older trees (Edgar et al. 1976). of the Rock... Logging frequency rather than in- eta. During the... tensity appears to be the critical dfires were ex... factor. Selection cutting places re- in the low... signal trees in close proximity to sole pine stand... inoculum from fresh-cut stumps se burned area... (Kellas et al. 1987). of mistletoe be... In eastern Victoria, Australia, ' structural ho... dieback and mortality of eucalypts, ed canopies. l... caused by *P. cinnamomi*, became however, top... severe after the combination of log- al diversity pre... ging, wildfire, and high rainfall (Fagg ment of larg... et al. 1986). The distribution and mixed size an... impact of the pathogen were af- mistletoe-infecte... fected by a combination of high inoculated th... moisture and temperature and low

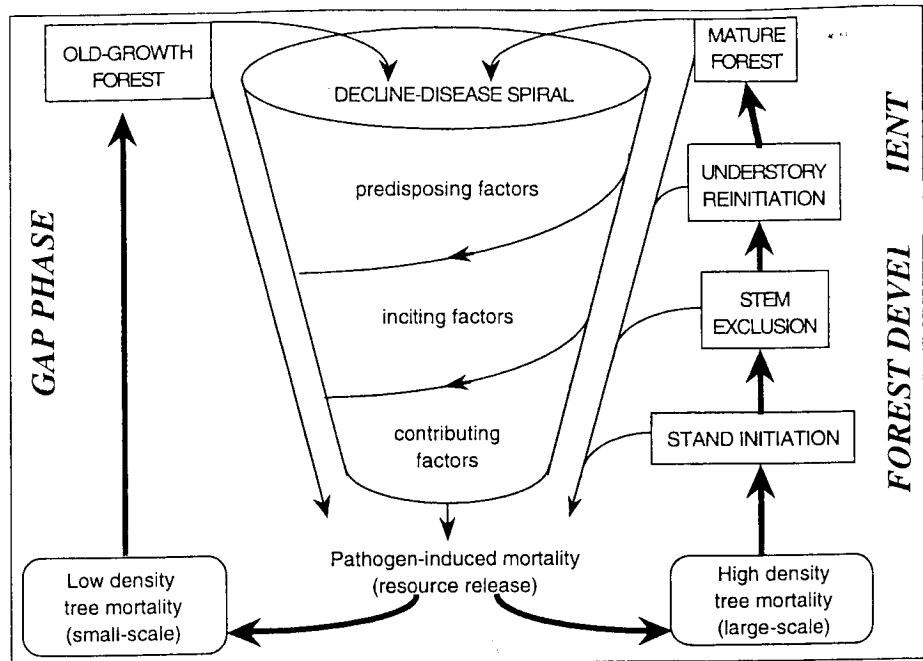


Figure 1. The role of pathogens in tree death and forest development. The model integrates Manion's decline-disease spiral (Manion 1991), Oliver's model of forest development following major disturbance (Oliver 1981), and gap-phase dynamics. Some pathogens function as agents of mortality most effectively at specific stages of forest development, some at many stages, and others as predisposing, inciting, or contributing factors in the death of mature trees. Upward arrows indicate the direction of forest development and downward arrows indicate mortality.

microbial populations and fertility (Weste and Marks 1987). Logging increased soil moisture and temperature by reducing vegetation cover and decreasing evapotranspiration. In addition, the creation of logging roads altered soil drainage patterns and facilitated the spread of the fungus. Wildfire, which followed logging, killed some eucalypts outright and weakened many others, perhaps increasing their susceptibility to the fungus.

Fire is the most important large-scale disturbance factor that contributes to landscape heterogeneity in the Pacific Northwest (Dickman and Cook 1989). However, fire episodes are strongly influenced by climate and by factors that cause fuels to accumulate. Insects and pathogens, including bark beetles, dwarf mistletoe, western gall rust (caused by *Endocronartium barknessii*), and comandra rust (caused by *Cronartium comandrae*), are important causes of mortality in lodgepole pine stands 100–200 years of age. Climatic factors and pathogens interact to influence the frequency of fire (Dickman and Cook 1989). Addi-

tionally, older stands that contain *P. weirii*-killed mountain hemlock are most susceptible to fire. root-rot infestations may incite the initiation and spread of fire. Interactions among climate, fire, and disease over long periods of time are responsible for cyclical changes in landscape pattern in these forests (Dickman and Cook 1989).

Forest-management practices have fragmented extensive tracts of original forests in the Pacific Northwest, increasing the incidence of some soil-borne tree diseases. The so-called staggered-setting system of timber harvesting (the creation of small, widely interspersed clear cuts) was developed in part to minimize the impact of windthrow on forest edges bordering clearcuts as well as for aesthetic reasons. This practice has resulted in a patchwork of old-growth and young-growth forests intersected by extensive road networks (Perry 1988). The networks have altered drainage patterns and increased incidence of Port-Orford-cedar (*Chamaecyparis lawsoniana*) root rot and black stain root disease (Hansen et al. 1986).

Pathogens and forest stage: A conceptual model

Pathogens affect pattern and process in forested landscapes. Their effects are exerted primarily through tree mortality or reduced competitive ability, which may occur at the small scale (gap phase) or at the large scale (forest development), and at any stage from stand initiation to old growth. Pathogens may also contribute to mortality as part of a decline complex involving predisposing, inciting, and contributing factors. To conceptualize the important role of pathogens in forest development, we have devised a model that integrates the decline-disease spiral (Manion 1991), Oliver's model of forest development (Oliver 1981), and gap-phase dynamics (Pickett and White 1985, Watt 1947; Figure 1).

The concept of the decline-disease spiral involves the interaction of many interchangeable factors on a population of mature trees (Manion 1991). Predisposing stresses lessen the ability of otherwise healthy trees to respond to pathogens or injury-inducing agents and are generally long term in duration. These stresses set the stage for inciting factors, which are generally short term in duration and may be either physiological or biotic. Contributing factors represent a collection of biotic or environmental factors that interact with predisposed trees and inciting factors to cause decline and eventual death. The precise set of interacting factors depends upon the specific decline scenario. Accordingly, pathogens may function as either predisposing, inciting, or contributing factors within a complex interaction that leads to mortality (Figure 1).

Forest development following major disturbance (i.e., one that basically removes most or all of the overstory trees) can be separated into these four stages (Oliver 1981): stand initiation, stem exclusion, understory reinitiation, and old growth. Following major disturbance, new individuals of trees, shrubs, and herbs appear from seeds, roots, or advanced regeneration during the stand-initiation stage. Soon, in the stem-exclusion stage,

further recruitment is prevented but changes in species dominance can occur due to differences in species growth rates and longevities. Later, during the understory reinitiation stage, new individuals of trees, shrubs, and herbs appear. The old-growth stage is reached as the advanced tree regeneration is able to grow into the canopy after the overstory trees die from various agents within a stand. Barring an additional catastrophic disturbance, one to a few canopy-dominant trees die at various points within the stand and throughout time, which allows one to a few trees to regenerate in a process known as gap-phase dynamics (Pickett and White 1985, Watt 1947).

Some pathogens may predominate at a particular stage of forest development, whereas others may occur across all stages (Figure 1). For example, leafspot of black cherry (*Prunus serotina*), caused by the fungus *Blumeriella jaapii*, causes defoliation and death of seedlings less than 30 cm in height (Stanosz 1989). The disease is widespread in black cherry-sugar maple stands on the Allegheny Plateau and is locally severe. In some stands, cumulative mortality of two-year-old seedlings has exceeded 70%.¹ In conjunction with herbivory, this disease can significantly impact regeneration of black cherry at both the stand-initiation and understory-reinitiation stages of forest development.

Ash yellows-induced mortality, however, is much more prevalent during stem exclusion. It may function as a natural thinning agent, in concert with competition-induced mortality, to push stands toward understory reinitiation.

Pathogens also have been shown to alter competitive relationships in short-rotation willow forests. Infection of willow by the rust fungus, *Melampsora epitea*, altered the competitive hierarchy between willow clones, which lowered the canopy height of infected individuals and decreased their ability to compete for light. All noninfected individuals survived, but 87% of infected individuals died within three years

¹G. Stanosz, 1994, personal communication. University of Wisconsin, Madison, WI.

(Verwijst 1993).

On the other hand, pathogens such as dwarf mistletoe and the root and butt rot fungi (e.g., *Armillaria* spp. and *Phellinus weirii*), because of their slow and localized spread, are likely to function primarily as gap makers in mature and old-growth forests. The changes in diversity and the specific patterns that result from mortality of selected overstory trees are likely to depend upon the site and species involved (Holah et al. 1993). In conjunction with fire and wind, however, these pathogens may also induce widespread mortality, which leads ultimately to stand regeneration. *Armillaria* root rot has also been implicated as a contributing factor in declines of many forest tree species including birch, maple, white pine, and oak predisposed by age, drought, or insect defoliation (Manion 1991). Viruses have been detected in many forest tree species (Nienhaus and Castello 1989), and their role as potential predisposing factors in forest decline has been postulated (Manion 1991, Nienhaus and Castello 1989). To date, viruses have been detected in red spruce (*Picea rubens*; Jacobi and Castello 1992, Jacobi et al. 1992), yellow birch (*Betula alleghaniensis*; Berbee 1957, Gotlieb and Berbee 1973), white ash (Castello et al. 1984), European beech (*Fagus sylvatica*; Hamacher and Quadt 1994), European silver fir (*Abies alba*; Flachmann et al. 1990), and Norway spruce (*Picea abies*; Nienhaus 1985). All of these species have experienced decline symptoms in recent years. However, as yet, the role of virus infection in forest decline still remains speculative.

Disease and ecological balance

Disease is essential to ecological balance in a natural forest. Because most are heterotrophic microorganisms, pathogens help to break down and release elements sequestered within trees and, by increasing mortality, they facilitate succession and help to maintain genetic, species, and age diversity.

Forest land managers, however, generally view pathogens not as essential to ecosystem function but

rather as an impediment to forest health. With managed forests, managers propose the same forest may or may not be sustainable without human management.

As forest managers move toward sustained yield systems, the health of the system has become a primary concern. Past management practices (e.g., fire suppression, abandonment, partial cutting) have had ecological consequences, including an increase in tree mortality (e.g., tree root rot, ash yellows, root rot). These diseases often require active management.

People must understand the effects of pathogens on forest health. It is necessary to know what pathogens are present within a forest, what the conditions are for outbreaks, how they act with other factors, and how development and management may influence the impact of other factors. Understanding how disease develops and spreads is essential to forest management.

Applied research to address forest decline is essential to protect it has only just begun. In the interim, pathogens must continue to conform to forest management practices.

Acknowledgments

We thank R. I. D. Manion, D. J. J. Worrall, and two anonymous reviewers for their comments on drafts of this manuscript.

References

- Augsburger, C. K. 1989. The impact of a tropical tree species on a temperate forest. *Ecology* 70: 1000-1010.
- Baranyay, J. A. 1990. Dwarf mistletoe in Alberta and Saskatchewan. Report BC-29, Canadian Forest Centre, Canada.

... as nuisances that interfere with management objectives. We propose that pathogens are themselves forest managers whose effects may or may not be compatible with human management objectives.

As forestry becomes more focused on sustained production and ecosystem health, management activities have become more intensive. Past management activities and land use (e.g., fire suppression, land abandonment, road building, and partial cutting) have changed the ecological balance resulting in an increase in the severity and/or impact of tree diseases (e.g., fusiform rust, ash yellows, and *Armillaria* root rot). The impact of these diseases often frustrates forest land managers.

People must learn to manage forests within the constraints imposed by the pathogens there. It is necessary to know what pathogens are present within a given forest ecosystem, what their host ranges are, what conditions are conducive to epidemic outbreaks, how the pathogens interact with other factors, how the development and spread of one disease may influence the spread and impact of other diseases, how the development of disease is likely to affect forest dynamics, and, conversely, how such dynamics affect disease development.

Applied, interdisciplinary research to address such questions is essential to proper management, but it has only just begun. In the interim, pathogens are likely to continue to confound and dictate forest management objectives.

Acknowledgments

We thank R. L. Burgess, S. Cook, P. D. Manion, D. J. Raynal, G. Stanosz, J. J. Worrall, and anonymous reviewers for their critical reviews of drafts of this article.

References cited

- Augsburger, C. K. 1984. Seedling survival of tropical tree species: Interactions of dispersal distance, light-gaps, and pathogens. *Ecology* 65: 1705-1712.
- Baranyay, J. A. 1972. Silvicultural control of dwarf mistletoe in young lodgepole stands in Alberta and British Columbia. Internal Report BC-29. Pacific Forest Research Centre, Canadian Forestry Service, Victoria, BC, Canada.

- Bennetts, R. E., G. C. White, and F. G. Hawksworth. 1991. Dwarf mistletoe: A forest pest revisited from a new perspective. Pages 24-25 in *Proceedings of a Symposium, Biodiversity of the Rocky Mountains, 1991 March 12-15*. Colorado State University, Fort Collins, CO.
- Berbee, J. G. 1957. Virus symptoms associated with birch dieback. *Can. Dept. Agric. Sci. Serv. Forest Biol. Div. Bi-mon. Prog. Rep.* 13: 1.
- Bloomberg, W. J., and G. Reynolds. 1985. Growth loss and mortality in laminated root rot infection centers in second-growth Douglas-fir on Vancouver Island. *Forest Science* 31: 497-508.
- Boggs, W. R., and L. W. Bailey. 1964. Brownfield Woods, Illinois: Woody vegetation and changes since 1925. *Am. Midl. Nat.* 71: 392-401.
- Boone, R. D., P. Sollins, and K. Cromack Jr. 1988. Stand and soil changes along a mountain hemlock death and regrowth sequence. *Ecology* 69: 714-722.
- Braun, E. L. 1950. *Deciduous Forests of Eastern North America*. The Free Press, New York.
- Burdon, J. J., and R. C. Shattock. 1980. Disease in plant communities. *Applied Biology* 5: 145-219.
- Burns, R. M., and B. Honkala. 1990. *Silvics of North America*. Vol. 2: *Hardwoods*. USDA Agriculture Handbook No. 654. USDA Forest Service, Washington, DC.
- Byler, J. W., M. A. Marsden, S. K. Hagle. 1990. The probability of root disease on the Lolo National Forest, Montana. *Can. J. For. Res.* 20: 987-994.
- Castello, J. D., L. A. Amico, and M. T. O'Shea. 1984. Detection of tobacco mosaic and tobacco ringspot viruses in white ash trees by enzyme-linked immunosorbent assay. *Plant Dis.* 68: 787-790.
- Cook, S. A. 1982. Stand development in the presence of a pathogen, *Phellinus weirii*. Pages 159-163 in J. E. Means, ed. *Forest Succession and Stand Development Research in the Northwest: Symposium Proceedings, 26 March 1981*. USDA Forest Service, Forest Research Laboratory, Corvallis, OR.
- Dickman, A. 1992. Plant pathogens and long-term ecosystem changes. Pages 499-520. in G. C. Carroll and D. T. Wicklow, eds. *The Fungal Community: Its Organization and Role in the Ecosystem*. Mycology Series, Vol. 9.
- Dickman, A., and S. Cook. 1989. Fire and fungus in a mountain hemlock forest. *Can. J. Bot.* 67: 2005-2016.
- Dinoor, A., and N. Eshed. 1984. The role and importance of pathogens in natural plant communities. *Annu. Rev. Phytopathol.* 22: 443-466.
- Dinus, R. J. 1974. Knowledge about natural ecosystems as a guide to disease control in managed forests. *Proc. Am. Phytopathol. Soc.* 1: 184-190.
- Dunn, C. P. 1986. Shrub layer response to death of *Ulmus americana* in southeastern Wisconsin lowland forests. *Bull. Torrey Bot. Club* 113: 142-148.
- Edgar, J. G., G. A. Kile, and C. A. Almond. 1976. Tree decline and mortality in selectively logged eucalypt forests in central Victoria, Aust. For. 39: 288-303.
- Fagg, P. C., B. K. Ward, and G. R. Featherston. 1986. Eucalypt dieback associated with *Phytophthora cinnamomi* following logging, wildfire, and favorable rainfall. *Aust. For.* 49: 36-43.
- Flachmann, M., D. E. Lesemann, B. Frenzel, and R. Koenig. 1990. Isometric virus-like particles in *Abies alba* Mill. and *Abies* species: Partial purification, improved detection by means of immunoelectron microscopy. *J. Phytopathol. (Berl.)* 129: 193-202.
- Foster, D. R., and E. R. Boose. 1992. Patterns of forest damage resulting from catastrophic wind in central New England, USA. *J. Ecol.* 80: 79-89.
- Franklin, J. F., H. H. Shugart, and M. E. Harmon. 1987. Tree death as an ecological process. *BioScience* 37: 550-556.
- Garman, H. 1882. The elms and their diseases. *Ky. Agric. Exp. Stn. Bull.* 84: 51-75.
- Gotlieb, A. R., and J. G. Berbee. 1973. Line pattern of birch caused by apple mosaic virus. *Phytopathology* 63: 1470-1477.
- Grubb, P. J. 1977. The maintenance of species richness in plant communities: the importance of the regeneration niche. *Biol. Rev. Camb. Philos. Soc.* 52: 107-145.
- Haack, R. A., and J. W. Byler. 1993. Insects and pathogens: Regulators of forest ecosystems. *J. For.* 91: 32-37.
- Hamacher, J. A., and A. Quadt. 1994. Isolation of cherry leafroll and broome mosaic viruses from European beech and transmission to beech seedlings. *Plant Dis.* 78: 849-853.
- Han, Y., J. D. Castello, and D. J. Leopold. 1991. Ash yellows, drought, and decline in radial growth of white ash. *Plant Dis.* 75: 18-23.
- Hansen, E. M., D. J. Goheen, P. F. H. J. J. Witcosky, and T. D. Schumacher. 1986. Biology and management of black-stain root disease in Douglas-fir. Pages 13-19 in O. T. Helgeson, ed. *Forest Pest Management in Southwest Oregon*. Forest Research Laboratory, Oregon State University, Corvallis, OR.
- Hawksworth, F. G. 1958. Survey of lodgepole pine dwarfmistletoe on the Roosevelt, Medicine Bow, and Bighorn National Forests. Station Paper 35. USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, CO.
- Hawksworth, F. G., T. H. Nicholls, and L. M. Merrill. 1987. Long-distance dispersal of lodgepole pine dwarf mistletoe. Pages 220-226 in C. A. Troendle, M. R. Kaufmann, R. H. Hamre, and R. P. Winokur, technical coordinators. *Management of Subalpine Forests: Building on 50 Years of Research; Proceedings of a Technical Conference, 1987 July 6-9, Silver Creek, CO*. USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, CO.
- Hawksworth, F. G., and D. W. Johnson. 1989. Biology and management of dwarf mistletoe in lodgepole pine in the Rocky Mountains. USDA General Technical Report RM-169. USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, CO.
- Heinselman, M. L. 1973. Fire in t

forests of the Boundary Waters Canoe Area, Minnesota. *Quaternary Research* 329-382.

J. C., M. V. Wilson, and E. M. Hansen. 1993. Effects of a native forest pathogen, *Phellinus weirii*, on Douglas-fir forest composition in western Oregon. *Can. J. For. Res.* 23: 2473-2480.

Houston, D. R., E. J. Parker, and D. Lonsdale. 1979. Beech bark disease: Patterns of spread and development of the initiating agent *Cryptococcus fagisuga*. *Can. J. For. Res.* 9: 336-344.

Hudler, G., T. Nicholls, D. W. French, and G. Warner. 1974. Dissemination of seeds of the eastern dwarf mistletoe by birds. *Can. J. For. Res.* 4: 409-412.

Huenneke, L. F. 1983. Understorey response to gaps caused by the death of *Ulmus americana* in central New York. *Bull. Torrey Bot. Club* 110:170-175.

Irwin, L. L., J. B. Buchanan, and E. L. McCutchen. 1989. Distribution and biology of the spotted owl nest sites in the eastside national forests, Washington. Unpublished report. National Council of the Paper & Pulp Industry for Air & Stream Improvement, Corvallis, OR.

Jacobi, V., and J. D. Castello. 1992. Infection of red spruce, black spruce, and balsam fir seedlings with tomato mosaic virus. *Can. J. For. Res.* 22: 919-924.

Jacobi, V., J. D. Castello, and M. Flachmann. 1992. Isolation of tomato mosaic virus from red spruce. *Plant Dis.* 76: 518-522.

K. C. 1953. Present composition of some stands of the former oak-chestnut forest in the southern Blue Ridge Mountains. *Ecology* 34: 44-54.

Kellas, J. D., G. A. Kile, R. G. Jarrett, and B. J. T. Morgan. 1987. The occurrence and effects of *Armillaria luteobubalina* following partial cutting in mixed eucalypt stands in the Wombat Forest, Victoria. *Aust. For. Res.* 17: 263-276.

Kile, G. A. 1981. *Armillaria luteobubalina*: a primary cause of decline and death of trees in mixed species eucalypt forests in central Victoria. *Aust. For. Res.* 11: 63-77.

Kile, G. A., G. I. McDonald, and J. W. Byler. 1991. Ecology and disease in natural forests. Pages 102-121 in C. G. Shaw III and G. A. Kile, eds. *Armillaria Root Disease*. USDA Agriculture Handbook No. 691. USDA Forest Service, Fort Collins, CO.

Lanier, G. N., D. C. Schubert, and P. D. Manion. 1988. Dutch elm disease and elm yellows in Central New York: Out of the frying pan into the fire. *Plant Dis.* 72: 189-194.

Levenson, J. B. 1980. The southern-mesic forest of southeastern Wisconsin: Species composition and community structure. Contributions to Biology and Geology Report. Milwaukee Co. Public Museum, Milwaukee, WI.

1981. Woodlots as biogeographic units in southeastern Wisconsin. Pages in R. L. Burgess and D. M. Sharpe, eds. *Forest Island Dynamics in Managed Landscapes*. Springer-Verlag, New York.

Mackey, H. E. Jr., and N. Sivec. 1973. The present composition of a former oak-chestnut forest in the Allegheny Mountains of western Pennsylvania. *Ecology* 54: 915-919.

Manion, P. D. 1991. *Tree Disease Concepts*. 2nd ed. Prentice-Hall, Englewood Cliffs, NJ.

Matson, P. A., and R. D. Boone. 1984. Natural disturbance and nitrogen mineralization: wave-form dieback of mountain hemlock in the Oregon Cascades. *Ecology* 65: 1511-1516.

Matson, P. A., and R. H. Waring. 1984. Effects of nutrient and light limitation on mountain hemlock: susceptibility to laminated root rot. *Ecology* 65: 1517-1524.

Matteoni, J. A., and W. A. Sinclair. 1985. Role of the mycoplasma disease, ash yellows, in decline of white ash in New York State. *Phytopathology* 75: 355-360.

McCauley, K. J., and S. A. Cook. 1980. *Phellinus weirii* infestation of two mountain hemlock forests in the Oregon Cascades. *For. Sci.* 26: 23-29.

McCormick, J. F., and R. B. Platt. 1980. Recovery of an Appalachian forest following the chestnut blight or Catherine Keever—you were right! *Am. Midl. Nat.* 104: 264-273.

Menges, E. S., and O. L. Loucks. 1984. Modeling a disease-caused patch disturbance: oak wilt in the midwestern United States. *Ecology* 65: 487-498.

Mlot, C. 1991. Diversity and dwarf mistletoe. *BioScience* 41: 755.

Nienhaus, F. 1985. Infectious diseases in forest trees caused by viruses, mycoplasma-like organisms and primitive bacteria. *Experientia* 41: 597-603.

Nienhaus, F., and J. D. Castello. 1989. Viruses in forest trees. *Annu. Rev. Phytopathol.* 27: 165-186.

Nyland, R. D., W. C. Zipperer, and D. B. Hill. 1986. The development of forest islands in exurban central New York State. *Landscape and Urban Planning* 13: 111-123.

Oak, S. W., and F. Tainter. 1988. How to identify and control littleleaf disease. USDA Forest Service Protection Report R8-PR 12. USDA Forest Service, Atlanta, GA.

Oliver, C. D. 1981. Forest development in North America following major disturbances. *For. Ecol. Manage.* 3: 153-168.

Parker, G. R., and D. J. Leopold. 1983. Replacement of *Ulmus americana* L. in a mature east-central Indiana woods. *Bull. Torrey Bot. Club* 110:482-488.

Perry, D. A. 1988. Landscape pattern and forest pests. *Northwest Environ. J.* 4: 213-228.

Pickett, S. T. A., and P. S. White, eds. 1985. *The Ecology of Natural Disturbance and Patch Dynamics*. Academic Press, New York.

Read, D. J. 1968. Some aspects of the relationship between shade and fungal pathogenicity in an epidemic disease in pines. *New Phytol.* 67: 39-48.

Runkle, J. R. 1990. Eight years change in an old *Tsuga canadensis* woods affected by beech bark disease. *Bull. Torrey Bot. Club* 117: 409-419.

Shaw, C. G. III, and Kile, G. A. 1991. *Armillaria Root Disease*. USDA Forest Service Agriculture Handbook No. 691. USDA Forest Service, Fort Collins, CO.

Sherman, R. J., and R. K. Warren. 1988. Factors in *Pinus ponderosa* and *Calocedrus decurrens* mortality in Yosemite Valley, USA. *Vegetatio* 77: 79-85.

Smallidge, P. J., Y. Han, D. J. Leopold, and J. D. Castello. 1991a. Management implications of ash yellows in northeastern hardwood stands. *Northern Journal of Applied Forestry* 8: 115-118.

Smallidge, P. J., D. J. Leopold, and J. D. Castello. 1991b. Structure and composition of forest stands affected and unaffected by ash yellows. *Plant Dis.* 75: 13-18.

Stanosz, G. 1989. Relationship of seedling height and dolomitic lime application to black cherry leafspot severity in northern Pennsylvania. Abstract. *Phytopathology* 79: 1143.

Stephenson, S. L. 1986. Changes in a former chestnut-dominated forest after a half century of succession. *Am. Midl. Nat.* 116: 173-179.

Turner, M. G. 1990. Landscape changes in nine rural counties in Georgia. *Photogrammetry Engineering and Remote Sensing* 56: 379-386.

Twery, M. J., and W. A. Patterson. 1984. Variations in beech bark disease and its effects on species composition and structure of northern hardwood stands in central New England. *Can. J. For. Res.* 14: 565-574.

van der Kamp, B. J. 1991. Pathogens as agents of diversity in forested landscapes. *For. Chron.* 67: 353-354.

Verwijst, T. 1993. Influence of the pathogen *Melampsora epitea* on intraspecific competition in a mixture of *Salix viminalis* clones. *Journal of Vegetation Science* 4: 717-722.

Walters, J. R. 1991. Application of ecological principles to the management of endangered species: the case of the red cockaded woodpecker. *Annu. Rev. Ecol. Syst.* 22: 505-523.

Watt, A. S. 1947. Pattern and process in the plant community. *J. Ecol.* 35: 1-22.

Weste, G. 1986. Vegetation changes associated with invasion by *Phytophthora cinnamomi* of defined plots in the Brisbane Ranges, Victoria, 1975-1985. *Aust. J. Bot.* 34: 633-648.

Weste, G., and G. C. Marks. 1987. The biology of *Phytophthora cinnamomi* in Australasian forests. *Annu. Rev. Phytopathol.* 25: 207-229.

Worrall, J. J., and T. C. Harrington. 1988. Etiology of canopy gaps in spruce-fir forests at Crawford Notch, New Hampshire. *Can. J. For. Res.* 18: 1463-1469.

Zimmerman, G. T., and R. D. Laven. 1984. Ecological interrelationships of dwarf mistletoe and fire in lodgepole pine forests. Pages 123-131 in F. G. Hawksworth and R. F. Scharpf, technical coordinators. *Biology of Dwarf Mistletoes: Proceedings of the Symposium, 1984 August 8*. General Technical Report RM-111. USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, CO.

Zipperer, W. C., R. L. Burgess, and R. D. Nyland. 1990. Patterns of deforestation and reforestation in different landscape types in central New York. *For. Ecol. Manage.* 36: 103-117.

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Darwin (1859) theory of evolution premise that natural selection promotes the spread of traits that enhance survival and fecundity. From the outset Darwin realized that natural selection was widespread and diverse. Darwin's theory defied explanation. Darwin's theory of survival or reproduction was the gauge of success. Darwin's theory was a burdensome armament in males of animals. Accountants proposed one of the generalities of evolution, and he took (Darwin 1859) his personal correlation (Darwin 1991) to existence. So did natural selection that Darwin evoked a natural selection, sexual selection. Darwin proposed that instead of traits that enhance survival, sexual selection spread and maintained. Geoffrey E. Hill is a professor in the Department of Wildlife Science and Cultural Experiment, Hall 331, Auburn University, AL 36849-5414. Hill's research interests include natural plumage color and mate preferences of birds in North America. © 1995 Institute of Biological