Plant Diseases and the Conservation of Tropical Forests

Conservation planners need to consider the roles diseases play in natural communities

Gregory S. Gilbert and Stephen P. Hubbell

Plants are the primary producers on which all other members of an ecosystem depend. Because of the central importance of their hosts, plant pathogens drive many ecological and evolutionary processes in natural ecosystems. Disease-causing organisms can regulate host populations and/or modify their genetic composition, restrict host distribution at various spatial scales, promote or reduce community diversity, mediate plant-herbivore and plant-plant interactions, create canopy gaps, and reduce host growth or reproduction and thus affect the availability of food for animals. They also may drive the evolution of species, sex, and host defenses (see reviews in Alexander 1992, Augspurger 1988, Barbosa 1991, Burdon 1991, Dickman 1992, Herms and Mattson 1992, Parker 1992). For all of these reasons, the role of plant diseases in natural ecosystems deserves greater attention in conservation biology efforts. Still, despite the recent acceptance of plant pathology into basic ecological and evolutionary theory, the scientific community has yet to recognize the implications of plant diseases for forest conservation science.

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Physical disturbances such as selective logging are major inducers of forest diseases

In most recent books on conservation biology, discussion of disease is either absent or is restricted to the potential dangers diseases pose for small, isolated populations of large, rare mammals. Plant diseases seldom receive more than a brief mention. Nonetheless, many disease-related themes, such as threats of extinction from introduced diseases in populations restricted to isolated fragments, apply equally well to plants as to large wild mammals. Particularly in highly diverse tropical forests with complex interactions among plants and animals, the potential for ecosystem-wide repercussions from plant diseases may be even greater, due to cascade effects, than those from diseases affecting animals. As tropical forests dwindle and disappear, and conservation efforts focus increasingly on ecosystems rather than on single species, it is becoming crucial that planners include plant diseases as one of many important factors when formulating conservation policy.

This article reviews some important factors in the development of plant diseases in natural communities, discusses the effects of plant diseases, and then focuses on known and postulated roles of diseases in tropical forests, devoting particular attention to the influence of plant diseases on conservation considerations. Of special interest is the importance of disease in fragmented forest reserves and the importance of plant diseases as links between protected areas and their agricultural surroundings. Because there is scant literature on diseases in natural ecosystems in the tropics, the article draws freely from studies of temperate systems and uses those data to hypothesize about phenomena in tropical environs.

Factors influencing disease development

In natural systems plant diseases may be present at epidemic (rapidly spreading, high incidence) or endemic (persistent over time, often low incidence) levels. Disease epidemics, particularly those caused by introduced pathogens or disturbances in an ecosystem, may provoke dramatic, permanent changes in a forest community. Such changes are unacceptable in the context of conservation efforts. On the other hand, endemic diseases may kill individual trees in a forest but play a key role in normal community function.

Plant diseases may be caused by fungal, bacterial, protozoan, viral, and nematode parasites that lead to some alteration in the plant's physiological activities. However, the presence of a pathogenic microorganism in an ecosystem no more

denotes the presence of a disease than does the presence of the plant. For disease to develop, three indispensable factors must all be present: a virulent pathogen, a susceptible host, and a conducive environment (Agrios 1988). Therefore, when considering the role of plant diseases in conservation efforts aimed at tropical forests, it is essential to appreciate the importance of all three factors and how they interact.

Endemic plant diseases are common components of all natural ecosystems. In contrast, most documented epidemics have resulted from some kind of human intervention or climatic vagaries that bring the three components together in a novel fashion. Anthropogenic factors important in epidemics of tree diseases include physical disturbance of an ecosystem through logging, road building, agriculture, or other such activities; air pollution or other industrial stresses; introduction of a new pathogen into a region; introduction of host species into a region where a pathogen already exists on other hosts; and large-scale plantings of single species of hosts. Unusual weather events, which may or may not be anthropogenic, may induce disease epidemics where susceptible hosts and virulent pathogens had previously coexisted at endemic levels. These events may include flooding, drought, or severe storms, or long-term climatic shifts.

The most serious tree disease epidemics have come from introductions of pathogens into regions with large populations of susceptible hosts. For example, after the introduction of the fungus *Cryphonectria parasitica* to North York, New York, from Asia in 1904 (Anagnostakis 1987), chestnut blight led to the ecological extinction of American chestnut (*Castanea dentata*), a once-dominant component of the forests of the eastern United States. In south Western Australia, the oomycete *Phytophthora cinnamomi* was introduced in the jarrah (*Eucalyptus marginata*) forests near the turn of the century and then spread in gravel during road construction (Shearer and Tippett 1989). This pathogen infects not only the jarrah but also many other plant species, and its aggressiveness and broad host range (more than 1000 host species) has led to the destruction of vast tracts of forest in Western Australia (Dickman 1992). However, where *P. cinnamomi* is endemic to undisturbed native vegetation (i.e., in South Africa), it apparently causes only occasional mortality of scattered plants from a variety of species (Von Broembsen and Kruger 1985).

Dispersal and persistence of a pathogen are crucial in determining whether a destructive epidemic is likely to arise from an initially patchy distribution of an introduced pathogen. Greater host densities, host growth, and heterogeneity of pathogen reproductive rates all increased the persistence of a pathogen in a model of a system likened to one of perennial evergreen tropical plants (Onstad and Kornkven 1992). In the moist tropical forest of Barro Colorado Island in Panama, the denser populations of common tree species sustain a greater diversity of wood-decay fungi (both pathogenic and nonpathogenic) than do rarer trees.[1] Many pathogens have dual methods of dispersal (short versus long distance); for example, oak wilt employs mycelial growth for neighbor-to-neighbor dispersal and ascospore production for insect-mediated, long-distance dispersal (Menges and Loucks 1984). The rarity of dispersal of the oak wilt pathogen into patches of healthy oaks is the primary constraint on disease spread. In agricultural systems, the rapid production of short-distance propagules is of primary importance early in disease focus formation, but as host availability decreases in the local area (disease saturation), long-distance transport becomes increasingly important.

Physical disturbances such as selective logging are major inducers of forest diseases. Where disturbance-incited diseases are present, the death toll from selective logging may be much greater than that directly attributed to logging practices, and it may affect many more species. Such indirect effects of logging are often overlooked in conservation biology.

The classic example comes from the North Temperate Zone, where airborne spores of the fungus *Heterobasidion annosum* infect cut stumps of a range of coniferous tree hosts. The fungus grows through the stump into the roots and can infect neighboring uncut trees through root grafts (Sinclair et al. 1987). The story is similar with widespread *Armillaria* spp., which colonize stumps of dead or cut trees, then form rhizomorphs (thick bundles of fungal hyphae) that move along roots and through the soil, infecting and killing the healthy roots of neighboring trees. *Armillaria luteobubalina* root rot of eucalyptus is seriously aggravated in logged sites, although it is present also in virgin forests (Kile 1983). Once uncommon in Montana forests, Douglas fir (*Pseudotsuga menziesii*) and grand fir (*Abies grandis*) are now dominant species due to the reduced density of white pine (*Pinus monticola*) as a result of selective logging, fire control, and the introduction of white pine blister rust. This shift in dominance is associated with high levels of root diseases in these firs (Byler et al. 1990).

Some pathogens require wounds in order to successfully infect a plant. Wounds to trees caused by road construction and logging provide infection sites for pathogens (and may attract insect vectors), which once established can spread to neighboring trees (Kellas et al. 1987). *Pestalotiopsis* spp., the most commonly isolated leaf pathogen on Barro Colorado Island, requires leaf damage to cause pathological infections.[2] Garcia-Guzman and Dirzo (1991) reported that for both understory and canopy plant species in Los Tuxtlas, Mexico, leaves could be attacked by herbivores or a combination of herbivores and pathogens, but almost never by pathogens alone. Inoculation experiments showed that the fungi isolated from those leaves required simulated herbivory in order to cause disease. These data suggest a strong interaction among herbivorous insects and plant pathogens. The results of any event that would change the abundance or species composition of her-

[1] G. S. Gilbert, A. Ferrer, and J. Carranza, manuscript in preparation.

[2] G. S. Gilbert, 1993, personal observation.

bivorous insects in a forest preserve could be magnified through the insects' interactions with plant pathogens.

Introduction of hosts into new areas where they escape pressures from pests present in their native environs and the planting of large populations of single species (whether native or introduced) have often led to serious disease problems. The most dramatic example of this effect is the spectacular spread of coffee rust throughout all coffee-growing areas of the Americas after the rust's introduction in Brazil (Thurston 1984). In this case, both host and pathogen were introduced into a new environment.

The development of many disease epidemics is density dependent, making reforested areas particularly vulnerable; witness the array of serious pest problems plaguing many important plantation species (Hilje et al. 1991). Holmquist (1992) recently reported the loss of thousands of hectares of Caribbean pine (*Pinus caribbea*) on plantations in Venezuela by the polyfagous fungus *Botryodiplodia theobromae*. Apparently the fungus is present in all of the more than 300,000 ha of pine in that country, causing wood staining but little mortality; however, when trees are additionally stressed due to high host density, poor soil fertility, drought, or insect attack, the infections become lethal, sometimes destroying stands of up to 1000 ha in just a few months. *Acacia mangium* plantations in Costa Rica have suffered losses of up to 50% from attack by the fungus *Ceratocystis*, which is introduced via insect vectors (Nichols and Gonzáles 1992).

Climatic shifts or perturbations, whether caused by shifting land-use patterns or natural cycles, are often crucial factors in inciting disease epidemics. Many important disease-related diebacks can be tied to a trigger event such as severe drought. For instance, *Botryosphaeria* branch dieback of California chaparral vegetation was related to a five-year drought (Brooks and Ferrin 1994). On Barro Colorado Island, a fungus-induced canker disease is associated with the rapid decline of the canopy tree *Ocotea whitei* (Gilbert et al. 1994) and perhaps other members of the Lauraceae. Most likely, a severe drought in 1982–1983, following a year of record rainfall, incited the epidemic. The disease has now nearly saturated the population of *O. whitei* on the island, and it remains to be seen what fate awaits the tree population.

Many important forest diseases have more diffuse causes and fall into the class of diseases called forest declines. Manion and Lachance (1992) recently provided an overview of decline diseases, where so-called predisposing factors (poor soil, climatic change, genetic susceptibility, and old age) allow so-called inciting factors (defoliating insects, drought, and air pollution) to open avenues for fungi, viruses, bacteria, and nematodes to infect and kill normally resistant hosts. It is difficult to ascribe blame for diseases of such complex etiology, with no single identifiable causal agent, but factors such as acid rain, air pollution, severe drought, and cohort senescence are often considered important (Franklin et al. 1987).

Of greatest importance for integrating plant pathology into tropical forest conservation is a better knowledge of the kinds of diseases present in natural tropical ecosystems and the life histories of the pathogens. How the pathogens respond to environmental factors, how they spread, and how diseases affect survival or reproduction of the hosts are essential issues. Only a few diseases have been studied in any detail in tropical forests outside of managed second-growth forests or plantations. Further theoretical and empirical exploration of how diseases spread in heterogeneous plant communities with various distances between susceptible host populations (within or among forest preserves) would be invaluable in determining which kinds of pathogens are most likely to cause problems.

Direct negative effects of disease

Disease epidemics may impede or foil efforts at species or ecosystem protection. In particular, tree species with highly restricted geographical distributions are vulnerable to invading pathogens that may quickly drive a population (or a species) to ecological extinction. Even if the epidemic runs its course before the population is eliminated, the aftereffects of the disease may seriously impede population recovery through reduced seed production (Jarosz et al. 1989), reduced growth rates in infected survivors (Gavin and Peart 1993), or by reducing the population to a point where stochastic events are important. A forest may require hundreds of years to recover from numerous tree diseases (Dickman 1992) because serious pathogens can often survive as saprophytes long after an epidemic is over. American chestnut still exist as stump sprouts in eastern US forests but never grow beyond the sapling stage, because once individuals reach 10–20 cm in diameter (15–20 years of age) they are reinfected by the blight pathogen from reserves in the stump and root systems (Anagnostakis 1987). Mountain hemlock (*Tsuga mertensiana*) regenerating in laminated root rot–induced dieback patches (caused by the fungus *Phellinus weirii*) do not always become infected immediately, perhaps because the increased soil nutrient levels and light availability from the death and decomposition of older trees boost the regenerating individuals' resistance (Waring et al. 1987). McCauley and Cook (1980) found that time to reinfestation of a disease-devastated area ranged from 88 to 165 years.

If the pathogen is virulent on only a subset of the population, an epidemic could severely reduce both host population size and genetic diversity, leaving the remaining individuals that much more vulnerable to outbreaks of other pests or to environmental fluctuations. If the disease is more severe on juveniles than adults (Gilbert et al. 1994) or vice versa, an epidemic may leave in its aftermath an even-aged cohort of individuals, making the population again more susceptible to additional pest problems. Plants stressed by one pathogen may be more susceptible to damage from other minor pathogens, leading to formation of disease complexes.

Reductions in plant genetic di-

versity from stochastic fluctuations, population isolation, or other selective agents (Lande 1988) may predispose plant populations to epidemic-level diseases. Although genetic uniformity does not necessarily lead to more serious disease problems in natural systems (Parker 1988, Roy 1993), it can predispose populations to massive disease outbreaks caused by introduced or endemic pathogens (Agrios 1988).

A disease epidemic that seriously reduces or eliminates any particular species in an ecosystem certainly has strong implications for conservation efforts. The real danger to such efforts, however, lies in the possibility that a keystone plant species might be affected. Terborgh (1986) discusses the importance of palm nuts, figs, and nectar sources to animal populations in the Amazonian forest of Cocha Cashu, Peru. Similarly, on Barro Colorado Island, fruit production by almendro *(Dipteryx panamensis)* during a period of normally low food abundance is of crucial importance to many animal species (Bonaccorso et al. 1980). In such cases, should a disease epidemic strike one of the important keystone species, the potential for cascade effects in the ecosystem is tremendous. Not only could bird and mammal populations be directly affected, but as these animals search for alternative foods, other plant populations could be seriously disturbed (Foster 1982).

As tropical conservation efforts increasingly and necessarily turn toward the opportunistic protection of small, fragmented tracts of available habitat, the probability that epidemics will have a major impact on individual tree species in the forest fragments is growing. Forest fragmentation and the creation of so-called edge forests are major effects of increasing deforestation in the Amazon (Skole and Tucker 1993). The likely introduction of pathogens through human activities and the environmental changes associated with habitat fragmentation and intensive land use in surrounding areas (e.g., hydrological changes, increased wind and desiccation) can both contribute to the development of disease epidemics in forest preserves.

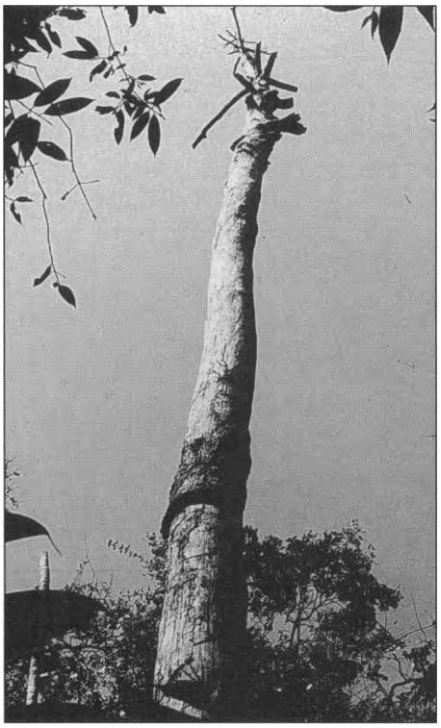

Figure 1. Diseased or dead standing trees serve both as sources of pathogen inoculum for further disease development and as important habitats for a range of forest organisms.

Direct positive effects of disease

Not all of the effects of plant disease in natural systems are negative. Diseases can act as selective agents that promote plant diversity (Connell 1971, Janzen 1970), create canopy gaps and other disturbances that can be important in community dynamics (Worrall and Harrington 1988), and provide food and housing for animals (Figure 1). Daily et al. (1993) recently highlighted the importance of a heartrot fungus (*Fomes ignarius)* in a species complex including sapsuckers (*Sphyrapicus nuchalis*), swallows (*Tachycineta bicolor* and *Tachycineta thalassina*), and aspen trees (*Populus tremuloides*). The sapsuckers excavate holes in fungus-infected aspens, and two swallow species depend on these holes for nest sites. Daily and colleagues described a keystone species complex that included the host tree, *F. ignarius*, and the sapsuckers; without the fungus, the sapsuckers would be unable to create cavities in the aspens. This fact indicates the role of the fungus as a keystone species in the system.

Perhaps the most influential theory of the role of pests in natural communities is that proposed independently by Connell (1971) and Janzen (1970). In the Janzen-Connell model, species-specific pests (insects) can be transmitted from the mother tree directly to offspring, or the pests may respond in a density-dependent manner to the abundance of offspring beneath the crown of a reproductive adult. The increased pest pressure on juveniles close to conspecific adults is likely to cause proportionately greater mortality of nondispersed offspring compared with those growing at some distance away. The Janzen-Connell model suggests that the disproportionate mortality of a tree's own nearby offspring may make that space available for colonization by nonsusceptible species (or genotypes), thus promoting or maintaining plant diversity. In the original model, insect pests were the driving force, but Augspurger and Kelly (1984) provided empirical evidence that pathogens can produce the same effects. On Barro Colorado Island, they showed that *Pythium*-induced seedling mortality (damping-off) of Carcuera *(Platypodium elegans)* was more severe both at high seedling density and close to conspecific adults. Because seeds not dispersed away from susceptible parent trees are more likely to succumb to damping-off than those dispersed farther away, damping-off should reduce local recruitment of susceptible species and permit that space to be colonized by resistant species. This effect has the potential to maintain species diversity in the forest, although it remains to be tested.

Additional support for the Janzen-Connell hypothesis comes from examination of the spatial distribution of a canker disease of saplings (probably ten years old and older) of *O. whitei* on Barro Colorado Island. Canker incidence and sapling mortality are greater near conspecific adults than far away; they are also greater at high sapling density than at low density (Gilbert et al. 1994). The incidence of mortality and disease is not a simple result of suppressed growth in the shade of a large canopy tree, because saplings

of *O. whitei* growing near adults of the nonsusceptible but confamilial Aquacatillo *(Beilschmiedia pendula)* were healthier than expected at random. Since 1982, the increased mortality to saplings located near adults has shifted the distribution of *O. whitei* saplings steadily away from conspecific adults; the pattern of mortality matches that expected if the canker disease were the cause behind the shift in spatial distribution (Gilbert 1993). Other, less common species of Lauraceae (e.g., *Nectandra cissiflora*) that also show symptoms of the canker disease exhibited higher mortality and lower recruitment (1982–1990) in areas of the forest where *O. whitei* was common than in areas where it was rare.[3] These observations suggest that the population dynamics of various susceptible hosts may be spatially linked through the actions of a common pathogen.

Seed dispersal beyond the parental neighborhood can function as a parasite avoidance mechanism even within species. When local parasites are more harmful to hosts than are unfamiliar parasites, as can occur through gene-for-gene coevolution of host and pathogen, dispersal of pathogen-free seed to neighborhoods with genetically different conspecifics can provide escape from particularly harmful pathogens (Ladle et al. 1993).

In tropical forests, where many trees have long-lived leaves, the crown of a canopy tree may provide opportunities for local or genotype-specific adaptation of pathogens. As propagules of the pathogen wash from the tree crown to the understory, those pathogens reaching that tree's offspring below may be particularly virulent on that genotype (Gilbert 1995). The literature on reciprocal inoculation and common garden experiments suggests that except for newly introduced plant pathogens, which in some cases can initially lead to epidemics, pathogens are generally more virulent on local, coevolved hosts than on distant host populations.[4] Our knowledge of genetic variability within tree populations in tropical forests is growing (Hamrick and Loveless 1986), but to what degree disease resistance is variable within and among populations remains largely unknown.

Localized physical disturbances in tropical forest structure are considered to be important factors in population dynamics of particular tree species and in the maintenance of species diversity (Brokaw 1985). Many tree species require canopy gaps and the high light levels they provide for rapid growth and successful recruitment to adulthood. Disturbances may form from catastrophic events, such as hurricanes, but more often single trees tip up or snap off due to wind or water stresses. Often trees that fall are already dead as a result of the action of pathogens, or they have been weakened by heartrot, root rot, butt rot, or canker fungi, all of which predispose them to physical damage. Worrall and Harrington (1988) showed that up to 40% of canopy gaps in a New Hampshire forest were associated with root rot and butt rot fungi. In Atlantic Panama, 23% of dead black mangroves were associated with a single species of butt- and heartrot polypore, and 26% of dead white mangroves with a single, different species.[5] In addition to the role of disease-related gaps in sapling growth, such pathogen-induced disturbances also may lead to uneven-aged stand development, which may be important in reducing pest pressure.

Plant diseases that differentially affect the plant genotype or species can influence plant intra- and interspecies interactions. A rust-resistant subpopulation of *Salix viminalis* gained a competitive advantage over its susceptible counterpart after a rust epidemic differentially reduced the height of plants with the susceptible genotype (Verwijst 1993). The Cascade Mountains of Oregon offer an example of an endemic forest disease increasing stand species diversity through manipulation of competitive hierarchies. As expanding margins of infection of laminated root rot (caused by *Phellinus weirii*) move into old forest dominated by mountain hemlock, most of the large hemlocks die. The less susceptible (but also less competitive and thus rare) Pacific silver fir (*Abies amabilis*), among other tree species, then exploit the reduced competition with hemlocks, substantially increasing stand diversity (Dickman 1992). On the other hand, in forests strongly dominated by silver fir, *Phellinus* can eliminate the hemlocks and other uncommon but susceptible species, substantially reducing stand diversity.

Building our understanding of the role of endemic diseases in so-called normal functioning of tropical forest ecosystems may provide the keys to predicting the likely effects of introduced pathogens or induced diseases. Of particular importance to preserving the tremendous biological diversity in tropical forests is an understanding of how diseases interact with other key ecological processes, such as competition and herbivory, and the cascade effects of diseases throughout the ecosystem.

Density and dominance

High dominance (monoculture plantations as an extreme) or high densities of single species is often an invitation to epidemic disease (see review in Burdon and Chilvers 1982). For instance, laminated root rot of mountain hemlock spreads much faster (34 cm/yr) through stands strongly dominated by mountain hemlock than through more diverse conifer stands (23 cm/yr; McCauley and Cook 1980). Factors that increase the density or dominance of a particular tree species may also increase the probability or severity of disease problems by facilitating the establishment and maintenance of specialized or particularly virulent fungi in the host population. In a mangrove forest in Panama, where saline-inundated soils lead to a community completely dominated by three tree species, only 4 species of lignicolous fungi (of 38 found) represented 64% of all macroscopic fungal samples.[6] Each of the four fungi was restricted to a

[3]G. S. Gilbert, S. P. Hubbell, and R. B. Foster, manuscript in preparation.

[4]E. A. Herre and G. S. Gilbert, manuscript in preparation. Smithsonian Tropical Research Institute, Panama.

[5]G. S. Gilbert and W. P. Sousa, manuscript in preparation.

[6]See footnote 5.

single host species. In contrast, in the highly diverse forest on Barro Colorado Island (310 woody species in 50 ha), the 5 most common polypore fungi (of 46 species) represented a total of only 38% of all polypore collections, and all of these fungi were host generalists.[7] Although host-specialized fungi exist in diverse tropical forests, they are apparently uncommon and may be limited to the most common hosts.[8] Forest reserves may be particularly susceptible to host density effects. Also, selective logging may increase the relative density of particular host species that are not of economic interest and put those populations at risk as well. Species may be lost in forest reserves through demographic stochasticity, whereas isolated reserves may not receive sufficient numbers of immigrant species to replace those lost (Shafer 1990).

Where important seed dispersers and/or predators, such as toucans, curassows, primates, and rodents, are lost (through hunting, habitat restrictions, disease, or stochastic extinctions), unusually high densities of offspring may form around parent trees. In these situations, density-dependent disease development (exacerbated Janzen-Connell effects) can lead to complete mortality of those offspring, and the lack of dispersers to move seeds into low density or low disease areas may have a severe impact on reproductive success. Inouye (1981) showed that when rodents were removed from a desert system, the density of a plant species on which they fed increased, as did the proportion of those plants infected by a parasitic fungus. If threshold levels for disease epidemics are common (Jennersten et al. 1983), such high seedling densities may provide the focus for epidemic development, with serious repercussions on the reproduction of affected host species. Because trees seldom recover from infections, temporary increases in density and short-lived epidemics can have long-lasting effects on a susceptible population, even if initial mortality is low. In addition, stresses from pathogenic infections at sublethal levels may predispose the hosts to mortality from a wide range of other stresses that are usually relatively innocuous.

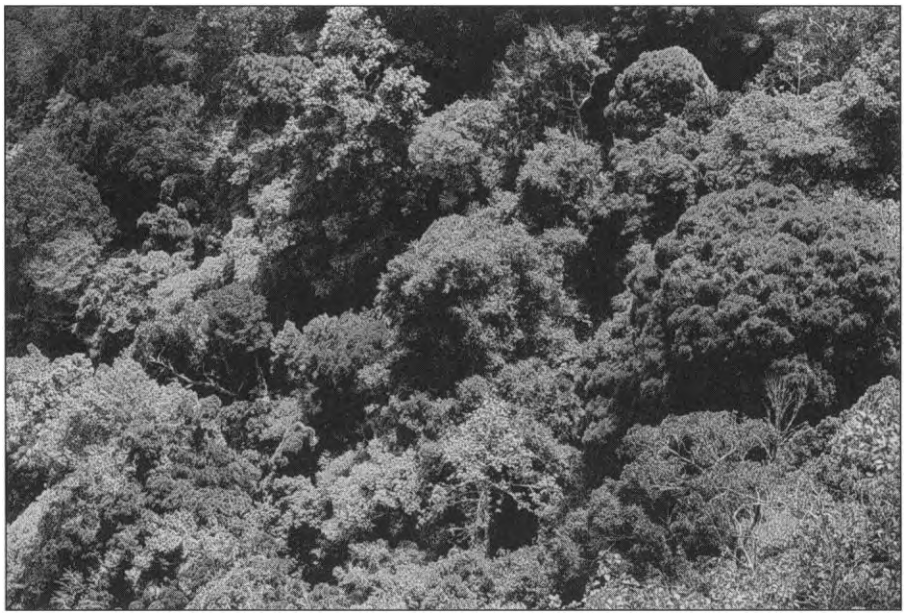

Figure 2. Understanding how the host range of pathogenic fungi affects the spread of tree diseases through highly diverse tropical forests may be key to evaluating the threat diseases pose to conservation efforts.

The idea that pathogenic microorganisms and large animals occupy similar niches in a forest ecosystem is not necessarily intuitive. However, conservation planners must be aware that freeing plant populations from animal herbivore pressures could enhance the probability of disease epidemic formation.

In tropical forests, the high diversity of tree species may reduce the spread of disease through simple physical interference, because intervening nonsusceptible plants could impede the spread of air- or soilborne pathogens (Figure 2). Therefore, increasing the relative density of host species, regardless of constant absolute density, may predispose populations to pathogen attack. However, high species diversity does not necessarily prevent the spread of disease, because many, if not most, plant pathogens are not species-specific. The appropriate unit for study may be a functional species, designated from a pathology perspective as species sharing common pathogens. For instance, the canker disease of *O. whitei* on Barro Colorado Island, mentioned earlier, apparently affects most of the species of Lauraceae on the island, although at various frequencies.[9] At the other extreme, only certain genotypes may be susceptible to a given pathogen, and the level of diversity of interest is the genetic rather than the species level.

Interactions with agriculture

Natural ecosystems can serve as reservoirs for agricultural pests. This has been well documented in a variety of tropical systems. Many pathogens important in tropical agriculture (e.g., fungi such as *Colletotrichum, Cercospora,* and *Pestalotiopsis*) are common on host plants in the tropical moist forest of the Barro Colorado Nature Monument in Panama.[10] Sequeira (1958) searched areas around coffee plantations for wild hosts of the fungus *Mycena citricolor*, the causal agent of the still economically important leaf spot disease. He found 150 host species from 45 families with natural infections from the fungus. However, the fungus can also be found on Barro Colorado Nature Monument, far from any coffee plantation.[11]

[7]See footnote 1.
[8]G. S. Gilbert, N. Talaro, C. A. Howell, and A. Symstad, submitted manuscript.
[9]See footnote 3.
[10]See footnote 2.
[11]G. S. Gilbert, 1992, personal observation.

Perhaps the best-documented disease of tropical regions with interactions between wild hosts and cultivated crops is bacterial wilt, caused by *Pseudomonas solanacearum*. Wild *Heliconia* populations and other plant species harbor the bacterium, which can infect bananas (*Musa acuminata)* planted on cleared lands (Sequeira and Averre 1961). In Côte d'Ivoire, rubber trees (*Hevea brasiliensis*) planted on sites recently cleared of forest suffer from the root rots *Rigidoporus lignosus* and *Phellinus noxius*. These fungi spread from infested stumps into the rubber trees, then quickly spread throughout the planting (Chadoeuf et al. 1993).

Although plant pathologists have traditionally emphasized the impact of natural communities as sources of infection for agronomic crops, the potential is probably considerably greater for agricultural areas surrounding forest fragments to serve as epidemic foci for diseases in natural communities. This phenomenon, which has not been well studied, is probably much more common than the literature indicates. Dobson and May (1986) discuss the classic example from the animal literature of the devastating effects of the rinderpest myxovirus on wildebeest (*Connochaetes taurinus)* and African buffalo (*Syncerus caffer*) in the Serengeti, where the virus was introduced by the Italian army along with herds of cattle. Agriculture-to-wildlands transfer of plant disease is illustrated by the spread of chestnut blight from New York City parks to eastern woodlands (Anagnostakis 1987). Widespread monoculture agriculture invites the development of disease epidemics by providing high densities of genetically uniform hosts, often planted under environmental conditions outside their native range. Seed or planting material brought in from outside a region is often contaminated with pathogens. Epidemics of disease in vegetable fields, fruit orchards, or tree plantations may result in high inoculum densities of the pathogen, which can then penetrate adjacent forested areas through either wind or insect vectors. As forest preserves become more and more fragmented and contact between native and agricultural crops increases, one would expect to see many more examples of such transfers.

One group of pathogens that should be of particular concern is the heteroecious rusts. These fungi require two unrelated host plant species to complete their life cycle. Several important rust diseases in the North Temperate Zone involve one cultivated and one wild host (e.g., *Gymnosporangium juniperi-virginianae* on apple and juniper, *Cronartium ribicola* on pine and currants, *Puccinia graminis* on wheat and barberry). Comandra blister rust uses lodgepole pine (*Pinus contorta*) and *Comandra umbellata* (rhizomatous perennial in sagebrush sites) as alternate hosts. Rust incidence on pine is much greater in stands adjacent to or up to 10 kilometers downwind of *Comandra* sites, which serve as inoculum sources (Jacobi et al. 1993). Sweet-fern rust (*Cronartium comptoniae*) is much more severe on both *P. contorta* and *Pinus banksiana* (both are alternate hosts to sweet-fern [*Comptonia peregrina]*) provenances collected from areas outside of the geographic range of sweet-fern (Hunt and Van Sickle 1984). Lack of selective pressures from the rust in outlying areas may have left pine populations highly susceptible, and introducing the alternate host and the rust to those areas could have a serious impact on pine populations. This scenario is not unlikely to emerge in tropical systems as larger areas of tropical forest are converted to agricultural uses.

Tropical forests face additional indirect pressures from diseases in agricultural systems. Introduction of crop diseases into previously unaffected agricultural regions, or anthropogenic factors that incite previously controllable diseases to epidemic proportions, may lead to rapid cutting of unprotected forests in search of so-called uncontaminated lands. This scenario is especially important for industrial-scale agriculture, such as the banana industry in Central America. When the fungal pathogen *Fusarium oxysporum* f. sp. *cubense* was introduced from Asia into the Americas with new varieties of banana in the first half of this century, Panama disease of banana (fusarial wilt) caused massive losses in plantations in Central America and South America: At least 100,000 acres of bananas were destroyed and abandoned (Thurston 1984). Facing bankruptcy, banana companies were forced to move into virgin forest for new plantings in order to escape the ravages of the disease (Wellman 1972). Eventually, disease-resistant varieties came into common use, and Panama disease is no longer a major commercial threat. In Trinidad in the late nineteenth century, a bacterial disease called Moko completely eliminated plantain production. Later, as the disease spread throughout Latin America in the 1960s, it destroyed millions of banana and plantain plants (Thurston 1984). In part as insurance against destruction of plantations by diseases such as Panama disease, Moko, and unknown diseases that may emerge in the future, banana companies have purchased thousands of hectares of forested land maintained in its natural condition in Central America. Although these set-aside lands are temporarily protected, in the future these valuable tropical forests could be rapidly lost as a result of disease epidemics occurring hundreds of miles away, should the companies feel compelled to abandon established plantations for virgin land.

The relationship between inoculum density and epidemic development is critical to the interactions between tree plantations or agriculture and disease development in forests. Buildup of massive amounts of inoculum of forest-endemic pathogens in adjacent plantations of susceptible hosts may provide enough inoculum to initiate epidemics in forest systems. Identifying common pathogens that cross the lines between forest and agricultural hosts is likely to be a useful next step. Because of the great potential for agricultural diseases to affect forest sites, management strategies may have to include policies for use of agricultural lands surrounding forest reserves, as well as management of the forests themselves.

Although usually considered distant from the science of conservation biology, research into the con-

trol of plant diseases in agricultural crops should be given high priority. Effective methods of disease control are likely to reduce the probability that new pests will be introduced into natural systems or that epidemics will develop from pathogens already present, and that large- and small-scale agriculturists will abandon so-called contaminated lands and destroy intact forests. Further study of disease processes in natural forest communities may contribute concepts or natural products useful in the control of agricultural diseases.

Plant diseases and forest reserve design

Understanding how rapidly and how far disease epidemics can spread through species-rich tropical forests and predicting how long it is likely to take for a disease-aftermath patch of forest to be clean of the pathogen are of particular interest for conservation planning. How large must a reserve be to permit recolonization of a disease-recovered area by a susceptible species before the epidemic eliminates the host species entirely? Available data from temperate forest epidemics (see above and Dickman 1992) suggest that the recovery process is generally much slower than the spread of disease and that individuals recolonizing aftermath areas are likely to be infected by the remaining pathogen, extending the recovery process even further. However, scientists have little information about disease spread or recovery in tropical forests, and describing such disease processes should be a priority.

Plant pathology is germane to the issue of whether one large or multiple small reserves of tropical forests are preferable (Shafer 1990). Dividing a reserve into various distinct parts might offer some protection against disease epidemics, if transmission of the pathogen between reserve sites is unlikely. However, advantages to such fragmentation may be nullified when agriculture and edge effects are important in introducing pathogens or inciting an epidemic. Assessing the likelihood of agriculture-reserve disease transfer, modes of long- and short-distance dispersal of tropical pathogens, and the probabilities of inter-reserve pathogen transfer over various distances is necessary before planners can make reliable recommendations about the pros and cons of reserve designs with respect to plant disease.

Conclusion

Conservation biology is perhaps the most interdisciplinary of all the biological sciences, but plant pathology has played only a peripheral role until recently. Common pathogens can forge a tight linkage between agriculture and fragmented and edge forests. The probability that introduced diseases will wreak havoc in protected ecosystems should spur phytopathologists to become involved in conservation biology and conservation planners to incorporate plant pathology into conservation strategies.

Without adequate attention to the roles of plant disease in the conservation biology of tropical ecosystems, our best efforts to preserve the majestic buttressed trees, large mammal fauna, and the beautiful array of bird and insect species in tropical forests may be foiled by the power of the microscopic components of biological diversity.

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