Ecology of plant diseases and other microorganism-plant interactions

by

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Every plant in a tropical forest interacts intimately with microorganisms from the time it germinates as a seed until the mature plant dies and returns to the soil. An appreciation of the ecology of these interactions is essential for understanding how tropical forests work as well as for effective forest management. Microorganisms can benefit the host plant through improved nutrition and defense against pests, or they can cause deleterious effects including reduced growth and reproduction, or even death. Decomposition of plant material by microbes is critical to nutrient cycling. Although plant diseases may have negative effects on individual plants, disease should be viewed as an important, natural part of the function of any natural ecosystem. Plant diseases can play key roles in regulating the dynamics and distributions of plant populations, determining the composition and diversity of plant communities, facilitating successional processes, providing wildlife habitat, and determining the success of restoration efforts. This chapter begins with an introduction to the major groups of microorganisms in tropical forests, then discusses aspects of their interactions with plants, and finally explores the impact of plant-microbe interactions on tropical forest ecology. The emphasis throughout will be on plant diseases, but other important plant-microbe interactions will also be included.

CHARACTERISTICS AND DIVERSITY OF MICROORGANISMS

Fungi.

The most important group of microorganisms in tropical forests is the fungi (Figure 1). The Kingdom Fungi includes a diverse group of eukaryotic organisms that obtain their energy through consumption of organic carbon (i.e., they are heterotrophic). These "True Fungi" grow primarily as hyphae, long tubes with an active growing tip. Hyphae are usually divided into cells by crosswalls called septa. The septa are not complete: cytoplasm and organelles can pass through pores in the septa, allowing exchange of nutrients and cellular components through the collection of hyphae called the mycelium. The fluidity of cellular

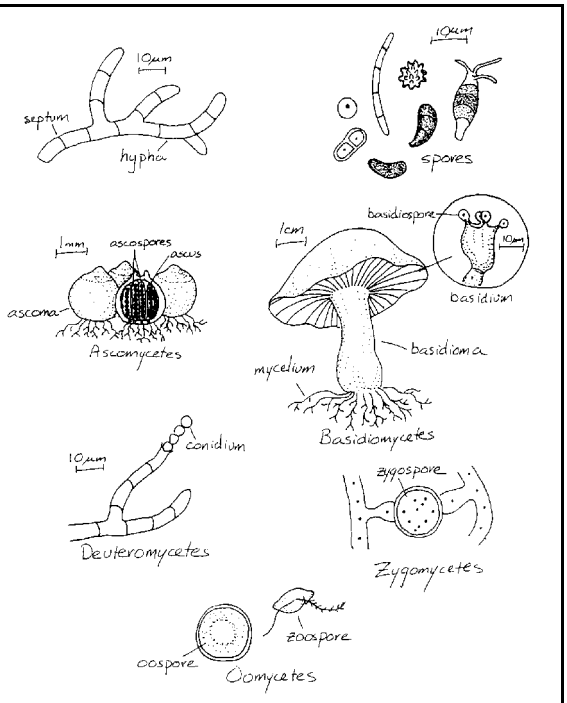

Figure 1. Key features and groups of fungi important in tropical forests.

components is an important characteristic of fungi, and we say that fungi are functionally coenocytic. Each compartment (not really a true cell, because of this coenocytic attribute) has one or more nuclei. As such, each hypha, or even the tip of a hypha, can continue to grow when separated from the mycelium as a whole. In this way, fungi can grow as clones indefinitely. Although individual hyphae are small (perhaps 5 μm in diameter) a single clonal individual may cover many hectares (Smith *et al*. 1992). The mycelium is the vegetative thallus of a fungus, and is the site of acquisition of nutrients. Because fungi consume preformed organic carbon by secreting enzymes into their substrate and absorbing the simple, digested carbon sources, the mycelium usually grows within its substrate.

Fungi reproduce by making spores. Spores are important in the dispersal of fungi, floating in air currents, splashing in raindrops, moving in flowing water, or being carried by insect (or other animal) vectors. The nuclei in spores may be the products of asexual (mitotic) division, so that when a spore germinates the hypha that grows from it is an exact, clonal copy of the parent mycelium. Such asexual spores are called conidia. In most cases fungal nuclei are haploid. Fungi can also reproduce sexually, where two haploid nuclei fuse to create a diploid nucleus, which then undergoes meiosis to produce recombined haploid nuclei. When sexual spores germinate, the resulting mycelium is genetically (and therefore, potentially phenotypically) different from its parent strains; such variability among fungal individuals is important in the ecological and evolutionary dynamics of plant-fungal interactions. Spores may be short lived and function mainly for short-distance dissemination and infection of new hosts, or be highly resistant to desiccation, radiation, and other damaging factors, remaining viable and dormant for decades.

Fungi include a diversity of organisms - 69,000 named species, with estimates of up to 1,500,000 species total (Hawksworth 1991; Pimm *et al*. 1995). In tropical forests as much as three-fourths of fungal species collected are new to science (Hawksworth & Rossman 1997). Any of a number of introductory mycology (the study of fungi) textbooks and three excellent dictionaries (Hawksworth *et al*. 1995; Ulloa 1991; Ulloa & Herrera 1994) will provide an overview of fungal diversity. Most of the ecologically important True Fungi in tropical forests belong to one of four groups: Ascomycetes, Basidiomycetes, Deuteromycetes, and Zygomycetes. Ascomycetes are the most diverse group, including inconspicuous semi-microscopic leaf-inhabiting fungi as well as large "dead man's fingers" commonly associated with decaying wood in tropical forests. Ascomycetes reproduce sexually by producing ascospores in structures called ascomata (singular ascoma; formerly these structures were called ascocarps). Basidiomycetes included mushrooms and shelf fungi; such showy reproductive structures are called basidiomata (formerly basidiocarps) and produce sexual basidiospores. Rusts are also basidiomycetes, but their reproductive structures are usually small and seldom showy, and they often have very complex life cycles producing up to five different kinds of spores. Most Ascomycetes and some Basidiomycetes also produce asexual conidia as part of their life cycle. Additionally, a large group of microscopic fungi have no known sexual state, and thus reproduce strictly through production

of conidia or by fragmentation of the mycelium. These are known as "Imperfect Fungi" or Deuteromycetes, and represent a large portion of the fungal diversity in tropical forests. Phylogenetically, most Deuteromycetes are really Ascomycetes, but due to the lack of known sexual reproductive structures are classified into their own group for convenience. The last group of True Fungi is the Zygomycetes, microscopic fungi that reproduce sexually by producing zygospores. These fungi are inconspicuous, but some of them play an important ecological role in their beneficial associations with plant roots in mycorrhizal associations (see below). Other Zygomycetes commonly decompose seeds, pollen, and even bread in the kitchen!

Oomycetes are another ecologically important group traditionally studied by mycologists, and often referred to as fungi. They are particularly noted for their importance in causing diseases of seedlings. They are not True Fungi, however, but rather Protists. They grow as hyphae without septa, have cell walls made of cellulose (rather than the chitin found in true fungi), reproduce sexually by producing oospores, and asexually by producing motile zoospores. Zoospores are released in saturated soils and swim through water-filled pores until contacting a suitable substrate: for this reason Oomycetes are often called "water molds". For simplicity many people refer to all the organisms usually studied by mycologists as fungi (with a small "f"), and use Fungi (big "F") to refer to the True Fungi.

We classify fungi into divisions based on their reproductive structures. However, the fruiting bodies that we see above the ground, on a leaf, or on a dead trunk may represent only a small portion of the fungal body when compared to the mycelium in the substrate. Even with sexual structures present, fungi are often difficult to identify using only morphological characters. Molecular tools may be needed to accurately identify certain fungi.

Bacteria.
The Kingdom "Bacteria" (Monera) is comprised of single-celled, prokaryotic organisms with a broad range of ecological strategies. Some are autotrophic, producing their own energy through photosynthesis or chemosynthesis, whereas most are heterotrophic. Heterotrophic bacteria feed by secreting enzymes that allow the absorption of carbon compounds from their substrate, as do fungi. Most bacteria are single-celled organisms, growing through cell division known as "binary fission". Bacteria grow exponentially; as each cell divides, and the two resulting progeny cells then divide to produce two new progeny each, and those four into eight, and so on. A single bacterial cell can rapidly produce a colony of millions of identical cells. In most cases each cell is physically separated from other cells in the colony, but in one group of bacteria, the Actinomycetes, the cells do not separate but rather form long chains that superficially resemble fungal hyphae. Actinomycetes are responsible for the "earthy" smell of soil, and form nitrogen-fixing associations with the roots of some tree species. Most bacteria fall into one of two large groups, the Gram-negative and Gram-positive bacteria, which are differentiated based on their cell-wall characteristics(Gerhardt *et al*. 1994). Of most importance in this chapter are Gram-negative bacteria, whose ecological roles range from disease agents to fixation of nitrogen in association with leguminous plants. Another unusual group of bacteria are the phytoplasmas, small, cell-wall-less bacteria that live in the vascular system of plants and can cause diseases. The principal sources of information on bacterial taxonomy and biology are Holt (1984-1989) and Balows et al. (1992); Gerhardt et al. (1994) offer a compilation of the most important methods for working with bacteria.

Viruses.
Viruses are particles of DNA or RNA packaged in a protein coat. They are unable to reproduce on their own; instead they infect the cells of a host plant and utilize the hosts cellular machinery to replicated their genetic material and package it as infectious virus particles. Plant-infecting viruses are known to produce a number of important diseases on trees worldwide (Nienhaus & Castello 1989). Many are transmitted from plant to plant by insect vectors, especially insects with piercing and sucking mouthparts. The importance of viruses in tropical forests is entirely speculative,

however, because they are almost completely unstudied in those ecosystems.

KINDS OF PLANT-MICROBE INTERACTIONS

Plants and microorganisms interact in many ways. The simplest interaction is that of fungi and bacteria decomposing dead plant material. Fungi are particularly important in the decomposition of wood, where extracellular enzymes break down complex organic molecules such as lignin and cellulose into ever-simpler molecules, converting highly structured plant material into soil, and making available trapped nutrients for plant uptake.

Much more complex are the interactions between microbes and living plants. The microbe and plant enter into a symbiosis (living together) where the plant is called the host and the microbe the symbiont. Symbioses may take many forms. The symbiont may obtain its nutrition from the host to the host's detriment, a type of symbiosis termed parasitism. Alternatively, the symbiont may live in the host but without causing significant negative effects: a relationship called commensalism. Finally, mutualism is a symbiosis where both the symbiont and the host benefit from the relationship. Unfortunately, particular plant-microbe interactions are rarely easy to categorize because these three types of symbioses fall along a continuum. As we will see below, the confusion increases further when a microbe is a commensal or even a mutualist at one stage in its life cycle, and a parasite at another - even on the same host!

A plant is diseased when a persistent agent (usually a parasite) disrupts the normal functions or form of the host plant, leading to impairment or death of the plant or parts of the plant. Disease is differentiated from injury by the persistence of association between the disease agent and the host (thus a machete cut is not a disease), and from simple parasitism in that the effect on the host are more extensive or damaging than would be expected from the simple removal of nutrients or water from the host. When a parasite causes a disease on the host, it is called a pathogen.

For all types of symbioses the association may be obligate (required for survival for the symbiont, the host, or both) or facultative (optional for either or both). Obligate pathogens can grow and reproduce only on a living host plant. Other pathogens also have saprotrophic phases (consuming dead organic matter), and only facultatively infect and cause disease on plants. Opportunistic pathogens facultatively attack plants that are already sick or dying from severe stress or damage from other pathogens. Some may cause little damage to the plant, but act almost as a saprotroph, primarily consuming tissue killed by an earlier pathogen.

NATURAL HISTORY OF DISEASES

The disease triangle.

The nature of a symbiosis depends on three interacting factors: the host, the symbiont, and the environment. Whereas the genetic makeup of both the plant and the microbe are critical in determining the range of possible outcomes of a symbiosis, the environment influences what the final outcome of the interaction will be. This tripartite phenomenon is traditionally discussed within the context of plant diseases (Figure 2), but is easily extended to any plant-microbe interaction. In the case of a plant disease, a virulent pathogen must come in contact

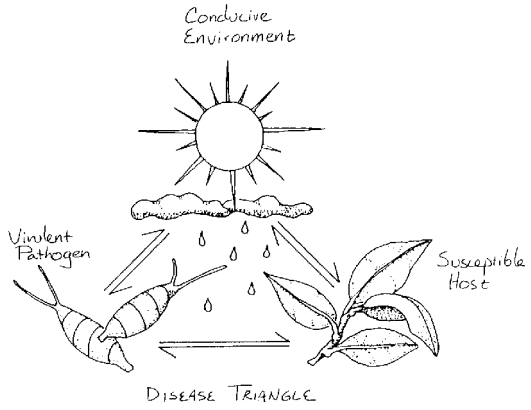

Figure 2. Plant disease development requires a virulent pathogen, a susceptible host, and a conducive environment.

with a susceptible host in order for disease to develop. However, even if spores of a pathogenic fungus do land on a susceptible host plant, if the environmental conditions are too dry, too wet, too hot, or too cold for the spores to germinate, infection will not take place and disease will not develop, although there is potential for disease development under the appropriate environmental conditions. Similarly, a fungus might infect but not cause disease in a host plant growing vigorously in moist, fertile soil in adequate sunlight, but the same fungus may kill a host plant should the interaction take place under more stressful (to the plant) conditions. Changes in the environment during tenure of a symbiosis can change the nature of the interaction.

Symptomology.
As mentioned above, disease is a negative disruption of the normal functions or structure of a plant, caused by a persistent agent. We know a plant is diseased because of the symptoms it expresses. Symptoms are responses of the host to the pathogen, and may include death, wilting, necrosis (death of tissues), chlorosis (loss of green in tissue), stunting, cankering, wood decay, abnormal growth, fruit loss, and many others. In addition to plant symptoms, signs of the pathogen are often visible. Signs include reproductive structures or mycelium of fungi, or bacterial ooze that can be seen associated with disease symptoms.

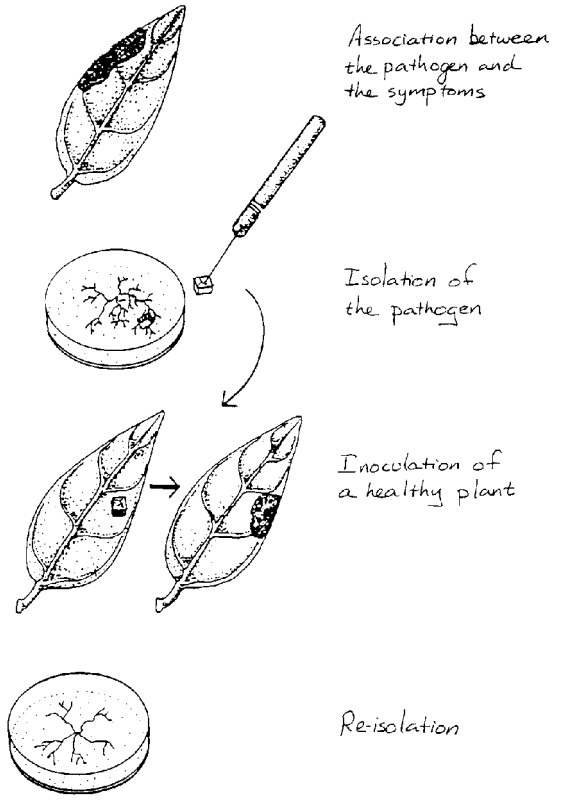

Figure 3. Koch's proof of pathogenicity is essential for showing what causes a disease. Constant association between a pathogen and symptoms, isolation of the pathogen into pure culture, reproduction of symptoms after inoculation of a healthy host, and re-isolation of the pathogen are the key steps. See Box 1 for a more detailed discussion.

Proof of pathogenicity.
Specific combinations of signs and symptoms are often diagnostic for particular kinds of diseases. However, it is important to remember that finding signs of a given fungus associated with diseased tissue does not necessarily mean that that fungus caused the disease (recall the discussion on opportunistic infections). In order to establish that a given set of symptoms is due to a particular microorganism, it is necessary to complete Koch's Proof of Pathogenicity, where the pathogen is isolated into pure culture and then used to cause disease in a healthy plant (see Figure 3 and the Proof of Pathogenicity box). This is the only reliable way of assigning cause of a disease to the appropriate pathogen, but it does have its limitations. Whereas for many diseases isolating the pathogen in pure culture (see Isolating Microorganisms box) and inoculating hosts is relatively easy, some obligate pathogens (e.g., rusts) cannot be grown in pure culture. For others, reproducing (or even recognizing) the environmental conditions appropriate for disease development may be extremely difficult. Some diseases may take many years for symptoms to develop (particularly true for some tree diseases). In the case of rare or endangered species, infecting hosts experimentally may be unethical. Disease complexes, which have multiple agents acting in concert, are particularly difficult to manipulate experimentally. In each of these cases, Koch's Proof of Pathogenicity is impractical, and a body of evidence in its entirety

BOX 1

Koch's Proof of Pathogenicity

In order to show that a particular pathogen is the cause of a plant disease, it is not sufficient to show that it is present, because the microorganism maybe only casually associated with the plant. Koch's four-step proof of pathogenicity provides a formal structure for establishing the cause of a disease. Figure 3 illustrates the process.

1. There must be a consistent association between the microbe and the disease symptoms. This can be established through direct observation, if the microbe produces visible identifiable structures (signs) on the host, through culturing of microorganisms on microbiological media, or through the use of genetic probes.

2. The microbe must be isolated into pure culture, away from its host. This is usually accomplished by growing the microorganism on microbiological media, but in the case of obligate pathogens, purification of the putative pathogen (e.g. viruses) is considered sufficient.

3. Inoculation of the pure culture into a healthy host must produce the original disease symptoms. Care must be taken to provide the appropriate environmental conditions for disease development, or false negative reactions are likely. It is critical to include control plants (mock inoculated in the same way as the target plants, but without the putative pathogen) to compare host responses to wounding with disease development.

4. The microbe must be re-isolated from the infected host, showing the it established infection and growth in the host, and that the symptoms were not due to a casual contaminating organism, or simply an effect of stress from wounding of the host. Control plants must not develop symptoms.

must be considered in assigning cause to a particular pathogen.

Disease cycles.
In order to understand where, when, and how a disease will affect tropical forest communities it is helpful to look at the key phases in the disease cycle. Each host and pathogen combination is unique, and it is an error to say "plant diseases work like this" - because there are more exceptions than rules. However, all diseases have several processes in common: dissemination of the pathogen, propagule germination, penetration and infection of the host, the host response, and growth and reproduction of the pathogen, which lead back to dissemination. The key to understanding the ecology of a particular disease lies in the details of how particular pathogens move about and infect their hosts, how different hosts respond, and how the environment influences these processes. Figure 4 shows an idealized life cycle diagram of a plant disease.

Dissemination.
Spores are the primary means of dissemination of pathogens from one host individual to another. Spores may be powdery or have appendages to increase their buoyancy in air or water currents, be flagellated to permit swimming through water, or be borne in sticky masses to facilitate transmission by insect vectors. Large, thick-walled, pigmented spores are likely to be resting structures for survival during adverse environmental conditions, and not be very important in pathogen movement. Looking at the kinds of spores produced and where they are produced (i.e., on the surface of a leaf or

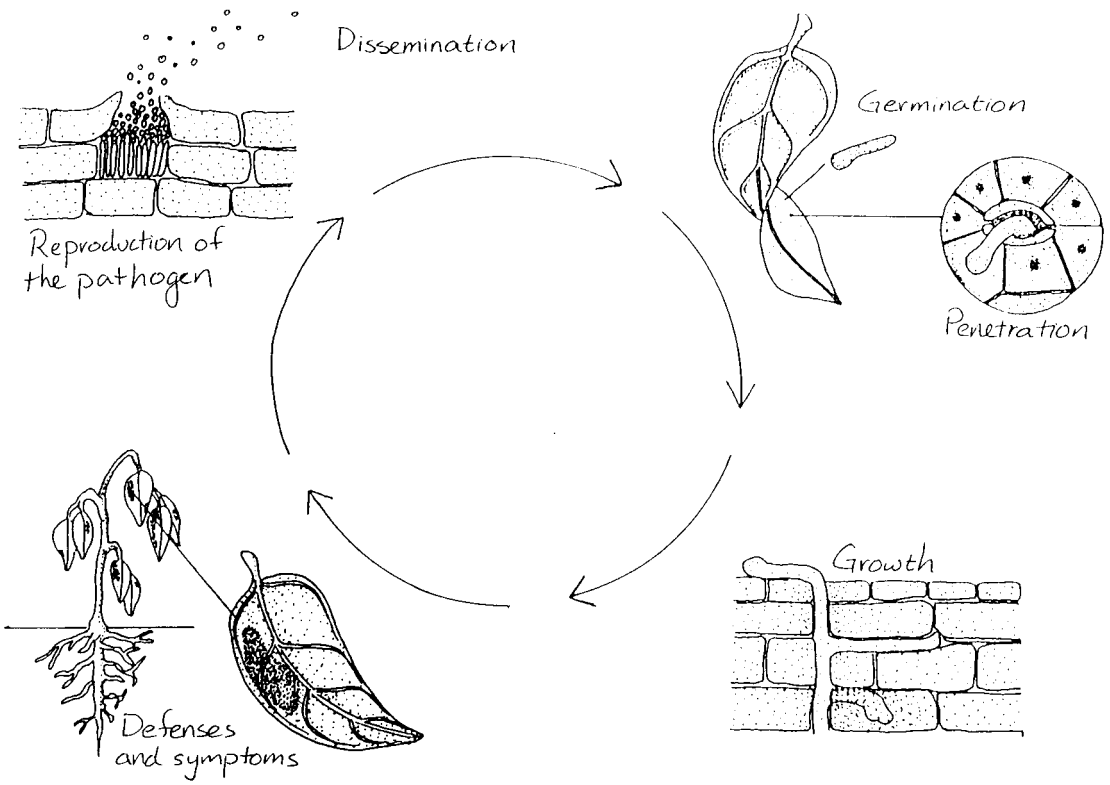

Figure 4. The life cycle of a typical plant disease involves dissemination, germination of spores, penetration and growth through the host, a host response, and reproduction of the pathogen.

embedded in stem tissue) provides clues to where the pathogen is most likely to be successful. For instance, Oomycetes produce flagellated zoospores that require large, water-filled pores in the soil to move from one host to another; thus we would expect that diseases caused by Oomycetes (such as damping-off of seedlings) would be most severe in areas where the soil periodically becomes saturated, and minimal in sunny areas like forest gaps where the soil dries out quickly after a rain. For example, damping-off of seedlings of *Platypodium elegans* (Fabaceae) was least severe in canopy-gap environments in a lowland tropical moist forest in Panama (Augspurger & Kelly 1984).

Germination.

In many cases, however, spore germination and subsequent infection of the host may be far more important than spore dissemination in the epidemiology of plant diseases. Indeed, plants in tropical moist forests are inundated by a constant "spore rain". On Barro Colorado Island (BCI) in Panama, Petri plates filled with fungal culture media and placed in the forest understory on a still, dry day in the rainy season caught 9.3 ± 3.3 spores cm^{-2} hr^{-1}; this includes only readily culturable fungi with air-borne spores, many of which are not plant pathogens (G. S. Gilbert, unpublished data). Extrapolating across a 24-hour period, a 50-cm^2 plant leaf would receive approximately 11,000 spores per day! Clearly, only a small fraction of these spores germinates and successfully infects a given leaf. The vast majority blow or wash off of the leaf surface, or die in place from desiccation or ultraviolet radiation. Of those that remain only the subset that encounter the appropriate environmental conditions will germinate (often a particular range of moisture or temperature is critical).

BOX 2

Isolating microorganisms from plants

Many fungi and bacteria are easy cultured from plant material, and the techniques involved are simple and inexpensive.

1. Surface sterilization. Plants are covered with fungal spores and bacterial colonies unrelated to the disease of interest, or the microorganisms living inside healthy tissue. The first step is to sterilize the surface of the plant to allow the microorganisms inside the plant to develop in a less competitive environment. First, simply wash the plant material under running water to remove loose dirt. Then, take a small piece of plant tissue (generally no more than a few mm in any dimension) and immerse it in 70% ethyl alcohol for 1-2 minutes, followed by 1-2 minutes in 0.525 % sodium hypochlorite (10% household bleach), followed by a 1-minute rinse in 70% alcohol. Shorter times may be required for delicate plant material, longer times for woody plant material.

2a . The simplest way to induce microbial growth is to place the sterilized plant pieces into a sterile Petri dish with a piece of moistened sterile filter paper. The high humidity will permit the growth and reproduction of fungi inside the leaf. Daily observation of the leaf tissue with a dissecting stereomicroscope permits the observation of the development of fungal structures. It is important that all the components of the system, water, dishes, filter paper, forceps, are sterile when the plant piece is added. They can be sterilized by autoclaving or in a simple pressure cooker for 20 minutes.

2b. Fungi can be isolated onto microbiological media by placing the surface-sterilized plant pieces onto malt extract agar (2% malt extract, 1.5% agar) or potato dextrose agar (boil potatoes and mixed the filtered broth with 1% dextrose and 1.5% agar) in Petri plates. Fungal growth should visible within several days. Using sterile needles, transfer a tiny piece of the developing colony onto a fresh culture plate to grow in isolation. Bacteria can be similarly cultured using 10% Trypticase Soy Agar (commercially available). This medium supports a wide diversity of bacterial species.

Penetration.

Among the spores that do germinate on a leaf surface only a small fraction are likely to be able to breach the first formidable host defense: the waxy leaf cuticle. Pathogens may take three routes to penetrating the cuticle of a host: first, direct penetration, by excreting enzymes or by physical entry via a modified hypha called a "penetration peg"; second, entry through leaf stomata; and third, entry through existing wounds in the leaf (Mendgen & Deising 1993). The third route, and probably the most important in tropical forests, is entry through wounds from herbivores or perhaps other pathogens. Garcia-Guzman and Dirzo (1991) showed that for both understory and canopy leaves in forest at Los Tuxtlas, Mexico, natural or simulated herbivory was required for nearly all foliar pathogens to cause disease. Similarly, many fungi that attack the woody parts of trees require wounds to gain entry into the host (Blanchard & Tattar 1997).

Host response.

Once the fungus penetrates the plant a dynamic interplay between the host and symbiont begins. The host may have many defenses, or resistance mechanisms, against infection by microbes. Some of these defenses are constitutive, that is they are a constant feature of the plant's anatomy or physiology. Constitutive defenses include tissue toughness, phenolic compounds and other resins, and nutritional quality of the host, and often are effective against a very broad range of organisms. The

other class of resistance mechanisms are induced defenses, which are produced only in response to damage or infection by a particular agent (the elicitor) and include the production of a wide array of toxic secondary compounds (see Bennett & Wallsgrove 1994 and Krischik 1991 for reviews). When induced defenses lead to a broad, physiological immunity from further pathogen attack it is known as systemic acquired resistance (SAR) (Ryals *et al*. 1994). Induction of defenses often requires that the host recognize the invading microorganism; in many cases plants have specific resistance genes which confer the ability to recognize and prevent infection from fungi unless the fungus possesses a particular corresponding gene that allows it to evade the host response, making it virulent on that host. This correspondence between genes for resistance and virulence is called the Gene-for-Gene hypothesis, and is of fundamental importance in understanding the evolution of plant-pathogen interactions as well as in breeding for disease-resistant plants (Flor 1942; Lawrence *et al*. 1981). Other forms of disease resistance are more complex, involving multiple loci or with strong interactions between environmental conditions and expression. Such "quantitative disease resistance" is only now yielding to study through molecular methods, but may ultimately prove to be more important than traditional gene-for-gene interactions (Young 1996). Differences exist among plant species, among populations, and even among individuals in the resistance mechanisms they posses. Similarly, a particular pathogen may be able to infect a wide range of host species, or be restricted to one or a few closely related hosts. Degree of host specificity is a key issue in determining the impact of a pathogen on a forest community.

Pathogens that evade initial host defenses may cause disease. Disease symptoms are host responses to the pathogen, but may be either host attempts to restrict the pathogen or manipulations of the host by the pathogen. Necrosis of plant parts results in the loss of photosynthetic tissue, usually considered a negative impact on the host. But localized killing of host tissue (called a hypersensitive response) can actually limit the spread of a biotrophic pathogen through the host by cutting the pathogen off from necessary nutrients: thus necrosis may in some cases be a type of defense. In other cases some pathogenic fungi that are unable to efficiently colonize living tissue will instead secrete toxins that kill plant cells in advance of the growing hyphae, allowing the fungus to invade poorly defended necrotic tissue (Scheffer & Ullstrup 1965). Pathogens may spread through the host by colonizing living tissue, killing tissue and growing into it, or by using the host vascular system as conduits for rapid movement.

Not all fungi that penetrate a leaf or twig will cause disease. Nearly every healthy-appearing leaf in a tropical forest is infected with a diversity of fungi, although the number of infections per leaf may vary widely (Dreyfuss & Petrini 1984; Petrini & Dreyfuss 1981; Rodrigues 1994). Fungi that live inside a plant without causing disease symptoms are called endophytes. These endophytes may be participants in various kinds of symbioses. Some fungi are known to provide benefits to the host plant through protection from herbivores or diseases (Clay 1990). Others may be commensals that remain quiescent within the host until it dies, and then consume the dead material as a saprotroph (Bills & Polishook 1994). Many pathogens may pass a long, latent endophytic phase before causing disease in response to host stress or senescence (Sinclair & Cerkauskas 1985). For instance, the Ascomycete *Botryosphaeria dothidea* may pass many months in a latent phase after infecting stems of *Tetragastris panamensis* (Burseraceae); however, cankers develop rapidly on infected individuals soon after the onset of drought (Gilbert & De Steven 1996). Other factors such as nutrient deficiency, air pollution, attack from insects, or light stress could similarly induce disease development (Manion 1991).

Reproduction.

In all cases the microbial symbiont must eventually reproduce in order to colonize a new host individual. Reproduction for fungi involves first developing sufficient mycelium and energy reserves to support reproduction, and then producing spores. Spores may be produced and liberated either from a living host

providing inoculum for infecting other parts of the same plant or other individuals, or may be produced only after the death of the host. How quickly a pathogen reproduces and how much inoculum it produces are key determinants of the rate a disease spreads through a host population and evolves responses to host defenses. The life cycle of many microbes is much faster than the life cycle of the plants that they infect; in some cases a pathogen can pass through the cycle from infection to reproduction in only days to weeks, or even pass through several generations on a single host individual. This provides much greater opportunity for the pathogen to evolve mechanisms to overcome host resistance than for the host to overcome new virulence factors.

IMPACTS OF PLANT DISEASES IN TROPICAL FORESTS

Plant diseases in tropical forests are much less well studied than are diseases of agronomic crops or of temperate zone forests. Nevertheless, it is apparent that crop plants suffer a substantially greater number of diseases in the tropics than in the temperate zone (Wellman 1968) and we should expect tropical forests to be similarly strongly affected. Whenever possible examples of diseases in old-growth or secondary tropical forests will be used to illustrate important principles or concepts, but when such studies are lacking, temperate zone examples or studies from tropical agricultural systems will be used instead. There are several recent general reviews of the impacts of diseases in natural ecosystems, with primary focus on temperate ecosystems (Castello *et al*. 1995; Jarosz & Davelos 1995). Disease ecology in tropical forests is perhaps the most open and rapidly developing field of research in tropical ecology.

Population dynamics

Plant diseases can regulate the dynamics of host plant populations in natural tropical forests by reducing reproductive output of diseased plants or by killing seeds, seedlings, saplings, or adult plants. Host reproductive output may be reduced indirectly when weakened by disease, its growth is impaired, or scarce resources are diverted away from reproduction toward disease defenses. The loss in yield in agronomic crops is the basis for the science of plant pathology, but there are no clear data for reproductive loss for tropical forest plants. However, two Panamanian studies show clearly how diseases can directly reduce reproductive output by causing fruit loss. Among fertilized ovules of *Anacardium excelsum* (Anacardiaceae), 34% were lost to fungal infection, principally *Cladosporium* sp. (Sánchez-Garduño *et al*. 1994). Even more dramatic, infection of developing ovaries of the understory tree *Faramea occidentalis* (Rubiaceae) by the rust fungus *Aecidium farameae* led to nearly 100% fruit loss in heavily infected individuals (Travers *et al*. 1998).

Seeds in the soil seed bank are also susceptible to microbial attack. Fungal attack was responsible for the loss of up to 39% and 47% of buried seeds of *Cecropia* and *Miconia*, respectively, after 5 months in a lowland tropical forest (Dalling *et al*. 1998).

By far the most disease-susceptible stage in a plant's life is the seedling stage. Tender, rapidly-growing tissues are particularly susceptible to damping-off fungi, particularly soilborne Oomycetes like *Phytophthora* and *Pythium*, but also Fungi such as *Fusarium* and *Rhizoctonia*. Damping-off is the rapid, dramatic mortality in seedlings of many tropical forest trees, usually characterized by a collapse and discoloration of the young shoot around the soil level soon after emergence of the seedling. In a study of seedlings of nine tree species on Barro Colorado Island, Panama, damping-off was the primary cause of death for six species, killing up to 74% of a parent tree's seedlings (Augspurger 1984). In greenhouse studies more than 80% of 16 host species tested were susceptible to damping-off (Augspurger & Kelly 1984).

Once tree seedlings begin to become woody, their susceptibility to damping-off fungi decreases, but other pathogens may become increasingly important. More than 30% of 1 to 7 year-old seedlings of *Tetragastris panamensis* suffered from die-back of the growing tips or cankering of stems caused by the

broad-host-range fungus *Botryosphaeria dothidea* (Gilbert & De Steven 1996). Experimental inoculations indicated that infection by *B. dothidea* could reduce plant growth to 59% that of control plants. Such growth inhibition may make the plants less able to compete for light or other resources in the forest understory, and thus more susceptible to mortality from other factors. Growth-chamber experiments with boreal conifers suggest that susceptibility to diseases under low-light conditions may be largely responsible for the "shade intolerance" of some species (Vaartaja 1962). This idea has yet to be tested in the tropics, but could play an important role in successional processes.

Understory saplings that survive these "childhood" diseases are still susceptible to a wide range of diseases. For example, foliar diseases may lead to substantial losses of photosynthetic material - more than one-fourth of all leaves of red mangrove juveniles (*Rhizophora mangle* (Rhizophoraceae)) suffered leaf necrosis from fungal pathogens (Gilbert and Mejía, manuscript in prep). Foliar diseases may be particularly important in the tropics. In a study of a number of species in moist tropical forests, up to 33% of leaf damage on saplings was caused by diseases, but only 11% in dry forests (J. Barone, pers. com.). Esquivel and Carranza (1996) showed that the petiolar pathogen *Phylloporia chrysita* caused 52% reduction in growth rates on infected individuals of *Erythrochiton gymnanthus* (Rutaceae) in Costa Rica. However, canker diseases may be much more important to the growth and survival of saplings in tropical forests, because of they can impede the main stem's water conductivity and its structural stability. On Barro Colorado Island, 9 of 10 species of Lauraceae are susceptible to a stem canker associated with *Phytophthora* sp. (Gilbert *et al*. 1994b), with more than 70% of the individuals of some species affected (Gilbert *et al*. 1994a).

Although mature canopy trees are also susceptible to foliar and canker diseases, they are less likely to suffer serious impacts than are smaller saplings. Instead, root-rots and heart-rots are the principle diseases of large trees. Most of the fungi associated with these wood-decay diseases are Basidiomycetes in the Aphyllophorales - commonly called shelf fungi. They spread from tree-to-tree by root grafting or by air-borne spores, often infecting through branch or trunk wounds (Tainter & Baker 1996). Some may aggressively attack the roots and bases of trees, weakening them and increasing their susceptibility to falling over in heavy wind or rain. Many such fungi attack principally the inner heartwood of large trees - hollowing out the trunk, but without affecting water or nutrient transport of the tree. For some tree species nearly all mature trees are hollowed out by wood decay fungi; for instance, in Panamanian mangrove forests *Avicennia germinans* is usually hollowed out by *Phellinus swietenii*, and in lowland moist forests the heartwood of *Ocotea whitei* (Lauraceae) is commonly decayed by *Phellinus apiahynus* (G. S. Gilbert, personal observation). Such hollow trees may live for many years without showing external signs of disease (see Wildlife Habitat, below).

Host population genetics

Because there is variability within host populations for resistance to pathogens, diseases can have a significant impact on the genetic structure of a host population by selectively killing the most susceptible individuals. A severe epidemic disease might quickly eliminate the susceptible component of a population, leading to a narrowing of the genetic diversity of the population. In a temperate-zone example, a rust epidemic swept through a population of *Salix viminalis* (Salicaceae) that was comprised of subpopulations resistant or susceptible to the pathogen . The disease reduced the height of the susceptible subpopulation compared to that of the resistant group, providing the rust-resistant subpopulation with a competitive advantage and leading to lower survival in the susceptible group (Verwijst 1993).

Disease-resistant trees in natural communities may be important sources of genetic resistance for the improvement of crops or plantation-grown trees. However, it is important to recognize that individual hosts showing disease symptoms are not necessarily the most susceptible individuals in the population, nor are disease-free individuals always disease resis-

tant. Healthy individuals may have simply escaped infection by chance or microclimatic conditions were not conducive to disease development. On the other hand, because disease symptoms are a host response to the pathogen, only those individuals sufficiently resistant to mount a response will be symptomatic - the most susceptible individuals may be killed outright. One example of this is Botryosphaeria canker on *Tetragastris panamensis*; infection with *Botryosphaeria* was strongly associated with plant mortality, but most mortality in infected plants occurred before the host could mount a defense response and develop the cankers characteristic of the disease (Gilbert & De Steven 1996). In fact, individuals that developed cankers actually had lower mortality rates than did infected canker-free plants.

Diseases can also drive evolution in the host population, providing an important selective force for developing resistance to diseases, and potentially altering the genetic structure and diversity in the host population. Because pathogens may evolve virulence factors that allow them to overcome new resistance traits in the host population, there is a constant 'evolutionary arms race' between pathogens and plants (Jarosz & Davelos 1995; Van Valen 1973). Pathogens face an evolutionary dilemma because greater virulence (greater use of the host as resources) can translate into greater reproductive potential, but if it kills the host plants it risks losing its food source and ultimately a drop in fecundity. As such, pathogens with efficient mechanisms for transmission to a new host when the old one dies are likely to evolve to become particularly virulent. Among those pathogens with a high capacity for transmission, those which most rapidly consume the host, reproduce, and disperse to new hosts will have the highest fitness, leading to a spread of virulence in the population. Conversely, pathogens that rely on an intimate association with a healthy host for successful reproduction, and pathogens without efficient transmission to new hosts may evolve towards a more benign association with their host (Jarosz & Davelos 1995). Microbes may have more opportunities to win this race because the life cycle of many microbial pathogens can be much faster (often days to weeks) than the life cycle of the plants they infect (many years for trees), providing the pathogens with more opportunities to evolve virulence mechanisms. Additionally, this arms race suggests that parasites can drive the maintenance of sexual reproduction in hosts, which is the primary mechanism for maintaining population diversity and permitting development of resistance in host populations (Hamilton 1980).

Extensive stands of genetically similar plants are often an invitation for serious disease problems. Once established in a host population, a pathogen may quickly spread through the remaining genetically similar individuals, causing dramatic reduction in growth, stability, or even population size. Such genetic uniformity is a common feature of monocultural agricultural systems and plantation forestry. For instance, *Acacia mangium* has been widely planted throughout the humid tropics in reforestation and agroforestry systems. However, fungus-induced heartrot and canker diseases have caused up to 50% losses in both Costa Rica (Nichols & Gonzáles 1992) and Malaysia (Ito & Nanis 1997). Similarly, extensive plantations of *Pinus caribbea* in Venezuela became infected with the fungus *Botryodiplodia theobromae*. The fungus does little more than stain wood in vigorously growing trees, but the added stress of a drought induced rapid disease development, leading to the death of entire stands of trees in just a few months (Holmquist 1994).

Density-dependent disease development

Whereas the development of disease on an individual plant requires the congruence of a virulent pathogen, a susceptible host, and conducive environmental conditions, the density of the host population is often a key determining factor in the incidence and spread of plant diseases. Density-dependent disease development is nearly a universal rule for fungal diseases: that is, as the density of a host plant increases, the proportion of the population affected by a particular disease also increases (Burdon & Chilvers 1982). This pattern would be expected because at higher densities conspecific hosts are closer together, increasing the probability that pathogen propagules

dispersed from an infected host will encounter a susceptible host. A number of vector-borne viral diseases show negative density dependence - decreasing disease rates with increasing host density - probably due to saturation of how many plants a finite number of insect vectors can visit (Burdon & Chilvers 1982). In tropical forests Phytophthora canker on *Ocotea whitei* (Gilbert *et al*. 1994a), and damping-off on *Platypodium elegans* and *Tachigalia versicolor* (Fabaceae) (Augspurger 1983; Kitajima & Augspurger 1989) all show clear density-dependent disease incidence. However, the apparently commensal symbiosis between the leaf-inhabiting fungus *Scolecopeltidium mayteni* and *Trichilia tuberculata* (Meliaceae) showed no evidence of density-dependent colonization; data suggest that the environment may be saturated with fungal propagules and that abiotic factors regulate the development of the symbiosis (Gilbert *et al*. 1997). In general, we would expect diseases that are spread by contact among individuals (such as by root grafts) or through short-distance aerial or aquatic dispersal of spores to demonstrate density-dependent development. Insect-vectored pathogens, and fungi that produce resting spores with low dispersal ability (those that wait for a new seedling to grow by it) and are less likely to produce density-dependent diseases.

The Janzen-Connell hypothesis

In natural forests, density of juvenile plants is usually highest close to parent trees. Gillett (1962), Janzen (1970), and Connell (1971) independently recognized that this correlation, in combination with density-dependent pest pressure, offers a mechanism that can both determine the spatial distribution of plant populations in rain forests, and as we will see later, maintain the diversity of tropical forests. This model, now commonly called the Janzen-Connell hypothesis, postulates that when there are host species-specific pests, one would expect greater pest-induced mortality among plant offspring close to the parent tree than among offspring dispersed to greater distances. There are two reasons for this. First, the parent tree can act as a reservoir for pests that are then transmitted to nearby offspring. Of particular importance would be pests that utilize adult hosts without inflicting great harm on them, but which can kill affected seedlings or saplings. Second, higher densities of offspring will result in greater pest pressures near to adults than farther away due to density-dependent attack. Taken together, successful recruitment of offspring will be greatest at some distance that represents a balance between decreasing seed density and increasing survivorship with distance from the parent. As such we would expect adult trees in tropical forests to be significantly less clumped than we would predict based on seed dispersal patterns alone (Clark & Clark 1984). Many tropical tree species do show spatial patterns consistent with Janzen-Connell effects (Wills *et al*. 1997).

Early consideration of the Janzen-Connell model was largely limited to insect herbivores; Augspurger (1983, 1984) brought plant diseases into the discussion when she demonstrated that damping-off diseases of seedlings of several tree species showed the Janzen-Connell pattern in lowland Panamanian forests. Similarly, canker diseases of older seedlings (Gilbert & De Steven 1996) and saplings up to 8 cm diameter (Gilbert *et al*. 1994a) follow the model's predictions. The relative importance of microbial and insect pests in producing Janzen-Connell mortality patterns is yet to be critically tested (but see Barone and Coley, this volume). There may be, however, a strong interaction between insects herbivores and diseases; the damage caused by greater herbivore pressures near to parent trees may be magnified if the herbivory facilitates the development of plant diseases - a common phenomenon in tropical forests (Garcia-Guzman & Dirzo 1991). Evidence is accumulating that plant diseases may be a major determinant in the spatial distribution of plant populations in tropical forests.

Community diversity

The Janzen-Connell model offers much more than an explanation for the spatial distributions of tropical trees: it provides a mechanism for the maintenance of high species diversity in tropical forests. In the absence of distance- and density-dependent pest pressures, we

would expect that by sheer force of numbers the seedlings growing beneath a parent tree would outcompete rare seedlings from heterospecific neighboring adults. This would first lead to a forest comprised of low diversity patches, and then the most fecund and competitive species would increase in frequency until patches coalesce, eliminating less competitive species and ultimately producing a low diversity forest. The Janzen-Connell hypothesis predicts that by selectively increasing the mortality of conspecific juveniles growing at high densities near to a parent tree, host-specific pests and pathogens can locally reduce interspecific competition and provide an advantage for heterospecific seedlings that are not susceptible to those diseases. In this way density- and distance-dependent diseases not only can reduce the degree of clumping of individual populations and prevent their unchecked growth, but permit a greater interspersion of tree species in the forest. Most tree species in tropical forests are randomly distributed or clumped (Hubbell 1979), but Janzen-Connell effects are probably responsible for reducing the degree of clumping of individual species and for maintaining a high species diversity, based on relative susceptibilities to the assemblage of plant pathogens present in the forest community (Wills *et al*. 1997).

A temperate zone example illustrates how an endemic forest disease (native diseases with moderate impacts on host population dynamics) can locally increase tree species diversity. In the Cascade Mountains in the northwestern United States, Mountain hemlock trees (*Tsuga mertensiana*) often dominate the forest structure, outcompeting Pacific silver fir (*Abies amabilis*). However, when laminated root rot (caused by *Phellinus weirii*) spreads through an area, many of the large *Tsuga* die, allowing the less susceptible silver *Abies*, as well as other rare trees, to exploit the resources made available by the death of the hemlocks, substantially increasing stand diversity (Dickman 1992).

Nevertheless, diseases may enter epidemic phases, with rapid spread and dramatic declines in the host population. Endemic diseases may be induced to epidemic levels by changes in environmental conditions or host density, but most often introduced pathogens are the cause of devastating disease epidemics in forest ecosystems. There are numerous examples of how disease epidemics affect the composition of temperate forest ecosystems (Anagnostakis 1987; Dickman 1992; Hibben & Doughtrey 1988), but few in the tropics. An outbreak of Phytophthora canker on *Ocotea whitei* (Lauraceae) in Panama, possibly induced by a very wet year followed by a severe drought, was associated with a dramatic decline in the population of this once common species (Gilbert *et al*. 1994a), and perhaps of other susceptible Lauraceae. In contrast, *Beilschmedia pendula* is the only non-susceptible Lauraceae in the forest, occupies the same habitat type, and has a similar spatial distribution to *Ocotea whitei* (Gilbert *et al*. 1994a). During the period of rapid decline of *O. whitei*, the population of *B. pendula* actually increased (Condit *et al*. 1995), perhaps indicating a disease-mediated release from competition for *B. pendula*.

The effect diseases have on community composition and structure depends on the host-specificity of the pathogens, but one can not generalize about whether pathogens are specialists or generalists. Some pathogens, such as rusts, usually infect only one or a few closely related plant species. Others, such as species of damping-off fungi *Pythium* and *Fusarium*, can attack hundreds of host species from many families. However, for the most part, assessing host specificity from records in the literature or patterns of association with particular hosts is unreliable, and in tropical forests the degree of host specificity is still largely unknown. There are two primary reasons. First, many species of common plant pathogens such as *Pestalotiopsis* and *Phomopsis*, were described based on spore morphology from dried herbarium specimens. When spores were larger, smaller, or differently pigmented than previously named congeners, or were simply isolated from a new host species, they were given a new name, usually the name of the host plant, implying host specificity. Such species descriptions are unreliable because the host often greatly affects spore morphology - spore morphology must be compared under identical growing conditions, genetic comparisons of collections is strongly advised, and host specificity is best

determined through cross-inoculations of hosts, not by observed patterns of association. Appropriate modern analyses of problematic genera support the need for caution in assuming host specificity (Rehner & Uecker 1994). At the other extreme is the second problem; some pathogens are morphologically indistinguishable but show strong geographic divergence in genotypic and virulence attributes (Balardin *et al*. 1997). Assuming that a species of pathogen that has been found on many host species world-wide lacks host specificity may be a mistake: traditional morphological classification may be a poor reflection on the underlying biology, and even pathogens that truly do have a broad host range may function as specialists in a local setting, if only a small subset of the plant species in the community are among its potential hosts. In short, understanding the specificity of plant pathogens in tropical forests is perhaps the most pressing disease-related research issue to be addressed in coming years.

Community disturbances

Endemic diseases may thus play significant positive roles in the structural dynamics of tropical forests, and may affect ecological processes by locally increasing soil nutrient levels and light availability due the death and decomposition of trees. In many tropical forests, frequent but small-scale disturbances, such as tree-fall gaps, are a key characteristic of forest structure and dynamics (Brokaw 1985). Pathogens may in part be responsible for creating these gaps. Canopy trees may be killed directly by pathogen attack, but equally important is the structural weakening of trees by heart-rot, root-rot, or canker -causing fungi; such weakened trees are much more susceptible to falling or snapping in the wind or from excess weight of heavy rains. In the temperate forests of the northeastern USA up to 40% of canopy gaps were associated with root rot and butt rot diseases (Worrall & Harrington 1988). In Panama, nearly one-quarter of dead *Avicennia germinans* mangroves were associated with *Phellinus swietenii* (G. S. Gilbert and W. P. Sousa, manuscript in preparation). Tree-sized branches of massive *Dipteryx panamensis* (Fabaceae) fallen to the forest floor at the La Selva Biological Station in Costa Rica were usually associated with damage from the canker-forming Ascomycete *Nectria* sp. (G. S. Gilbert, personal observation).

Rapid, extensive mortality caused by disease epidemics on dominant forest species may have significant impacts not only on community composition but on forest structure and function. Such epidemics are especially likely to occur with introduced diseases. Until the turn of this century, the American chestnut (*Castanea dentata* (Fagaceae)) was the dominant component of the hardwood forests of the eastern USA. In 1904, the fungus *Cryphonectria parasitica* was introduced to New York from Asia (Anagnostakis 1987). This canker-causing fungus spread quickly throughout the range of the host, leading to the ecological extinction of American chestnut, and dramatically changing the structure of the forests. Similarly, the Oomycete *Phytophthora cinnamomi* was introduced into south Western Australia near the turn of the century, and subsequently spread throughout the native jarrah (*Eucalyptus marginata*) forests on logging roads. Not only is jarrah and many other *Eucalyptus* species extremely susceptible to *P. cinnamomi*, but this aggressive pathogen infected and killed a wide range of other forest community members; the epidemic caused by this introduced pathogen led to the destruction of vast tracts of forest in Western Australia (Dickman 1992; Shearer & Tippett 1989). Elsewhere in Australia *P. cinnamomi* caused dramatic declines in the dominant tree and shrub species in a shrubby woodland, allowing a resistant shrub species to become dominant, and fundamentally changing the structure of the ecosystem (Weste 1981). However, in South Africa where *P. cinnamomi* is endemic to undisturbed native vegetation, it causes only occasional death of scattered individuals from a variety of host plant species (Von Broembsen & Kruger 1985).

Finally, forest disturbances may affect the fungal community itself. Canopy gaps and storm damage can affect fungal community composition in tropical forests (Carranza 1996; Lodge & Cantrell 1995). The subsequent effects of such changes in the fungal community

on forest vegetation or nutrient cycling are unknown. A critical issue yet to be addressed is whether the ecological roles of different members of a fungal community is sufficiently redundant to prevent fungal community changes from having an effect on ecological processes (Lodge *et al*. 1996).

Wildlife habitat

Many forest animals depend on tree diseases for their survival. Raccoons, bats, and many other mammals frequently use disease-hollowed trees for nests or roosts. Anteaters and woodpeckers search decaying logs for insect prey, many of which depend on decay to gain entry to the wood. Trees with heartrot are important for providing nesting cavities for a number of bird species (Daily *et al*. 1993), including endangered species (Hooper *et al*. 1991; Jackson 1977). Diseased and standing dead trees are increasingly valued for their importance in maintaining habitat diversity for wildlife in forest ecosystems, and forestry practices now often call for leaving such trees in place after timber harvests to ensure minimal impact on the wildlife community.

However, forest tree diseases can also have important negative effects on wildlife populations. Epidemic diseases of keystone species such as palms, figs, nectar producers, or other trees that produce important food sources (Terborgh 1986) could have dramatic impacts on the animal populations that depend on them. An emerging disease of uncertain cause is currently threatening the wax palm, *Ceroxylon quindiuense*. The fruits of this palm are an important source of food for toucanets and many other animals in the montain forests in the central Andes of Colombia (Madriñán & Schultes 1995). Additionally, plant populations not susceptible to the disease could suffer damage as hungry animals search for alternate food sources (Foster 1992).

Wood decay

One of the most important roles of fungi in forest ecosystems is the decomposition of wood. Fungi are joined by protists and bacteria in the decomposition of cellulose, but are nearly unique in their ability to break down lignin. Fungi that break down only cellulose cause the wood to become darker during decomposition because the relative concentration of the dark-colored lignin increases; this kind of decay is called <u>brown rot</u>. However, other fungi, most notably certain groups of Basidiomycetes in the Polyporales (shelf fungi, or Aphyllophorales) degrade both cellulose and lignin. As they break down the lignin the wood becomes lighter in color, and is called <u>white rot</u>. In tropical forests most wood-decay polypores are white rotters (89% of the 72 genera know from Costa Rica (Carranza 1996)). Brown rot fungi are much more common in northern conifer forests, 82% of known brown-rotters occur primarily in conifers (Gilbertson & Ryvarden 1986). Fungi that decompose wood may first attack and kill living trees, or may be heart-rot fungi associated with internal dead wood in living trees for many years, or saprotrophs that colonize and decay only dead woody material. In addition to Aphyllophorales, a number of Ascomycetes, particularly in the Xylariaceae, are important in wood decay in tropical forests. The inside of a fallen log is a complex mosaic of numerous fungal species and genetic individuals competing for a finite and transient resource; this competition is often evidenced by the visible black "zone lines" of interaction where two fungi meet and wall off their territories in decaying wood (Rayner & Boddy 1988).

Based on collection records, most Aphyllophorales are able to colonize a range of host plant species, although several seem to be highly host specialized (Carranza 1996). Although most of the Aphyllophorales known from tropical America are cosmopolitan or pantropical (Ryvarden 1996), it is not yet clear whether morphologically similar collections from different parts of the world share similar host ranges, or whether populations have evolved distinct host preferences according to the local plant community composition. Probably more important than host range in determining where particular wood-decay fungi live are climatic factors, with numerous species restricted to particular elevation zones (Carranza 1996).

Perhaps the most striking feature of wood decomposition in tropical forests is its great rapidity. Fallen trees in temperate forests often

take many decades to decompose, whereas in moist tropical forests fallen trunks of even large individuals of palms and Bombacaceae may vanish in only a few years (G. S. Gilbert and A. Ferrer, manuscript in preparation). Estimates from Puerto Rico (Odum 1970) and Panama (Lang & Knight 1979) suggest trunk decomposition in less than 10 years for a variety of tree species. There are exceptions, however; individuals of *Tabebouia* spp. killed during the flooding of the Panama Canal in 1913 are still standing today (G. S. Gilbert, personal observation).

What determines the rate of wood decay is complex. Much decay takes place in living trees, in the form of heart-rot. Living trees have an array of responsive defenses against wood-decay fungi, including mechanical compartmentalization of fungal infections through formation of callus tissue or resinous boundaries (known by its English acronym CODIT, for Compartmentalization of Decay In Trees) (Shigo 1984). Additionally, trees possess a wide variety of constitutive and induced chemical defenses (Pearce 1996). Production of resins and callus in response to wounds are often effective physical barriers to prevent infection of susceptible tissue (Pearce 1996), and vary greatly among species of tropical trees (Guariguata & Gilbert 1996).

Dead wood cannot actively respond to fungal infection, but intrinsic characteristic of the wood may make it more or less susceptible to decay. Based on the literature from temperate-zone studies, wood density is not thought to be a good predictor of wood decay rates (Rayner & Boddy 1988), and data on the antimicrobial effectiveness of the important constitutively-produced aromatic terpenes are equivocal (Pearce 1996). However, a series of laboratory studies from early this century (Dickinson *et al*. 1949; Hess *et al*. 1950; Wangaard and Muschler 1952; Wangaard *et al*. 1954; Wangaard *et al*. 1955) on the decay rates of 95 species of tropical American woods by white-rot and brown rot fungi provide data for an assessment of the importance of wood density and terpenes on tropical wood decay rates. Specific gravity of the woods ranged from less than 0.3 to more than 1.1 g/cm^3. I divided the species into two groups, low-density and high-density wood, using the median wood density (0.635 g/cm^3) as the dividing value.

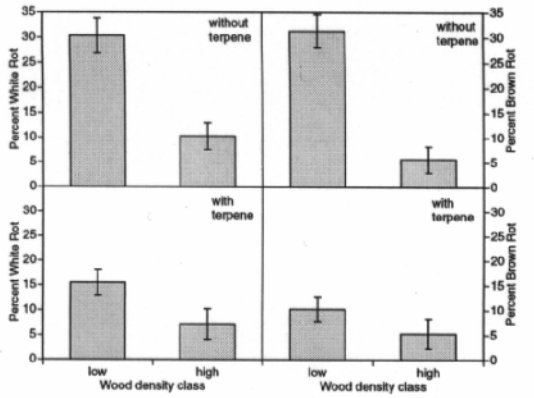

Figure 5. Relative decay rates of wood from 95 tropical American tree species, by white and brown rot fungi. Both increase wood density and the presence of terpenes slows wood decay rates. Data are from sources as described in the text.

Based on a variety of literature sources I determined whether the analyzed species was known to produce aromatic terpenes, and analyzed how density and terpenes influence wood decay rates using a two-way analysis of variance, separately for white rot and brown rot (Figure 5). Both wood density and terpene production have strong effects on decay rates ($P \leq 0.003$ for combinations), and there was a significant interaction between the terms for brown rot ($P = 0.0005$) and a nearly significant interaction ($P = 0.054$) for white rot. Low-density wood without terpenes is extremely susceptible to rapid decay, whereas high-density wood with terpenes is extremely resistant (Figure 5). High-density wood without terpenes and low-density wood with terpenes were intermediate. Although these laboratory tests suggest a wide range in decay rates influenced by wood structure and chemical composition, comparative decay rates of trunks of different tree species in forest environments is much needed.

Litter decomposition

Likewise, fungi are the primary decomposers of leaf litter and small woody debris. Over

500 species of fungi were isolated from leaf litter on Barro Colorado Island (Cornejo *et al*. 1994), and about 200 from litter samples in a similar lowland humid forest in Costa Rica (Bills & Polishook 1994). Mushrooms (Agaricales) are also important saprotrophs in tropical forests (Ovrebo 1996), both decomposing organic matter and keeping it from washing away on steep slopes (Lodge & Asbury 1988). A number of fungi produce hardened bundles of hyphae called rhizomorphs (often used by birds to construct nests), or stringy spreading mycelia that catch, colonize, and bind together leaf litter before it reaches the forest floor (Hedger 1993). In many tropical forests it is common to find clumps of dead leaves and small twigs bound together and decorated with small basidiomata of *Marasmius* or other mushrooms. By colonizing the leaves before they hit the soil, such fungi may gain a competitive advantage over the myriad fungi in the soil and leaf litter. But these "canopy decomposers" must still compete with many fungi that colonize the living leaves but live as quiescent endophytes until the leaf senesces; such fungi may have a substantial competitive advantage over other litter decomposers (Bills & Polishook 1994; Cornejo *et al*. 1994)

MUTUALISMS

Endophytes

Nearly every apparently healthy leaf and stem in a tropical forest has fungi living within it. For the most part, the ecological function is not known, although as discussed above some may be latent pathogens, neutral commensals, or beneficial mutualists (Carroll 1988). The endophytic mutualists obtain nutrition from the host and provide some ecological benefit in return. The best studied mutualists are those that infect grasses (Clay 1990). These Ascomycetes produce toxins that reduce feeding or survival of herbivores, providing a competitive advantage over non-infected plants. These endophytes may also suppress host reproduction, directing the host energy into an increased vegetative growth rate. The diversity of endophytes of *Quercus* and conifer leaves have also received much attention, (Wilson and Carroll 1997; Faeth and Hammon 1997; Carroll and Carroll 1978; Espinosa-Garcia and Langenheim 1990), but the importance of these endophytes in regulating insect pest or disease damage is still unclear. There are few studies on tropical endophytes in the literature (Dreyfuss and Petrini 1984; Laessøe and Lodge 1994), with the most intense work done on endophytes of palms (Rodrigues 1994). For no tropical system, however, is the ecological importance of endophytic fungi known.

Mycorrhizae

Mycorrhizae are mutualistic symbioses between fungi and host roots, where the fungus receives carbon from the plant in return facilitates that uptake of nutrients and water from the soil, increasing host growth (Janos 1980b). In tropical forests, most trees enter into an Arbuscular Mycorrhizae (AM) symbiosis with fungal symbionts in the Glomales (Zygomycetes) (Connell and Lowman 1989). The symbiosis is obligate both for the fungus (they do not grow saprophytically and have never been grown in culture) and for the host (who have much reduced growth in the absence of the fungi due to poor nutrient uptake) (Lovelock *et al*. 1996). These mycorrhizae are sometimes called Vesicular-Abuscular Mycorrhizae or Endomycorrhizae, because the fungus penetrates the host cell wall and produces bush-shaped hyphae with a large surface area of contact with the cell membrane, allowing efficient mineral and carbon transfer between the symbionts. The fungus also produces mycelium in the soil for the capture of water and nutrients. The AM fungi tend to have broad host ranges, leading to the suggestion that different host individuals, even of different species, could share below-ground resources through a common mycelial network (Grime *et al*. 1987). Just how diverse is the AM fungal assemblage, and how strong host preferences might be, is still unfolding (Morton 1990; Morton *et al*. 1995). Arbuscular Mycorrhizal symbiosis may facilitate successional processes (Fischer *et al*. 1994; Janos 1980a), modify host competitive ability (Hetrick *et al*. 1989), and provide protection from pathogenic fungi (Newsham *et al*. 1995).

Some tropical trees form Ectomycorrhizal symbiosis with Basidiomycete fungi. In these

cases the fungus forms a sheath around the root with mycelium extending into the soil. These fungi tend to have much greater levels of host specificity, grow more aggressively through the soil, and are better able to obtain nutrients from soil organic matter than are AM fungi (Connell and Lowman 1989). Although widespread in temperate forests, Ectomycorrhizae in the tropics are largely restricted to monodominant forests, communities dominated by single tree species (Connell and Lowman 1989). Dipterocarpaceae, Fagaceae, Myrtaceae, and the Caesalpinoid Fabaceae commonly have Ectomycorrhizae. Connel and Lowman (1989) proposed that the greater efficiency and host specificity of Ectomycorrhizal fungi may facilitate the formation and maintenance of low-diversity tropical forests by offering ecological advantages over species in AM symbioses.

Rhizobia and Actinorhizae

Nitrogen is often a limiting element in plant communities, and plants are unable to acquire nitrogen directly from the atmosphere. Papilionoid and Mimosoid Fabaceae have evolved mutualistic symbioses with the bacterium *Rhizobium*, where plants provide carbon sources in exchange for nitrogen fixed by the bacteria. Rhizobia fix as much as 0.27 kg N ha^{-1} yr^{-1} in Puerto Rico (Edmisten 1970), and probably much more under certain conditions. This fixation is the basis for using legumes as 'green manure' and for providing nitrogen in intercropping schemes. The bacteria infect root hairs and induce the formation of nodules on the plant roots, within which the bacteria can fix atmospheric nitrogen gas to a more biologically available ammonium form. The symbiosis may be host specific either at the time of root infections and nodule development, or at the time of nitrogen fixation, when the efficiency of nitrogen fixation varies among host-bacterial combinations. Other tropical trees, like *Alnus* and *Myrica*, form root associations with Actinomycetes, slow growing bacteria that are also able to fix atmospheric nitrogen. Because many soils are nitrogen poor, plants with nitrogen-fixing symbioses often have competitive advantages over non-fixers in establishment on disturbed sites, even helping introduced leguminous plant species to invade natural plant communities (Vitousek and Walker 1989).

RISKS, CONSERVATION, AND FOREST MANAGEMENT

Microorganisms thus play critical roles in the dynamics, diversity, and processes in tropical forests. Mutualistic symbioses between bacteria or fungi and plants can provide competitive advantages to the hosts and alter community composition. Endemic diseases may play crucial roles in maintaining community species diversity and provide habitat heterogeneity for wildlife. However, human intervention can often lead to the development of devastating disease epidemics (Gilbert and Hubbell 1996). The introduction of pathogens from other ecosystems can have dramatic impacts on native vegetation (Shearer and Tippett 1989; Anagnostakis 1987). Forest disturbance, through logging or other human activities, can stress plants and create wounds leading to large increases in disease levels. Forest fragmentation can change microclimatic conditions in the forests, leading to stresses that can incite disease development. Forestry practices such as plantations that result in high-density stands of very low diversity are especially susceptible to disease epidemics, through density-dependent disease development (Burdon and Chilvers 1982). Plantation forests and monocultural agriculture may pose threats to nearby natural forests by permitting the increase in pathogen inoculum that, given susceptible hosts in the native vegetation, may infect the natural stands from an inoculum reservoir (Gilbert and Hubbell 1996).

Plant diseases must be taken into consideration in the planning of forest reserves. Because diseases are contagious and the spread from individual to individual is dependent on the proximity between individuals, density, diversity, and connectivity are all important elements in determining if particular forest areas are vulnerable to catastrophic epidemics. Small forest reserves are subject to dramatic changes should a pathogen be introduced or conditions change to promote disease development. Much of the remaining tropical forests are in

discontinuous fragments, and there are efforts to provide biological corridors to connect them, permitting gene flow among populations and reducing the dangers from stochastic effects. Although these are important benefits, the corridors also increase the risk that a disease that develops in one fragment can spread along corridors to other forest fragments. Such risks have been discussed for animal diseases (Dobson and May 1986), but to date there are no data on the risks from plant diseases that corridors carry (Gilbert and Hubbell 1996).

The diversity of plant-microorganism interactions in tropical forests may provide economic incentives for forest conservation, as well. Biological prospecting for fungi with industrial uses in biocontrol, cosmetics, and pharmaceuticals may be even more promising than plant-centered bioprospecting. Fungi have traditionally been sources of particularly creative secondary chemicals, and because many fungi can be grown in culture, they are well-suited for industrial exploitation (Chapela 1997). Fungi collected in initial sampling expeditions can be maintained in collections and grown quickly and inexpensively through fermentation technology, eliminating the need for repeated expeditions to collect additional materials. Fungal collections can be especially rich source of information because many more samples of fungi than plant materials can be collected and processed with the same effort, easily providing thousands of different fungal types for automated industrial screening. Industrial interest in access to diverse tropical ecosystems for microbial bioprospecting may be one tool for leveraging resources for forest conservation. Merck, Inc. in Costa Rica (Bills and Pollishook 1994) and Novartis, Inc. in Panama, Mexico, and India are among the current partnerships between pharmaceutical companies and tropical nations using fungal bioprospecting to help promote forest conservation.

FUTURE DIRECTIONS FOR RESEARCH

The importance of microorganisms, and especially diseases, is one of the newest and most exciting fields of research in tropical forest ecology. Even on Barro Colorado Island, arguably the best studied tropical forest from a disease perspective, we have data on diseases for less than 10% of the tree species – and for even fewer species do we have any assesments of the impact of those diseases on host dynamics. There are several key areas of research for the next decade. At the microbial level, we need better understanding of the diversity and geographical distribution of microbial species, and an assessment of the degree of host specificity for important groups of pathogens and mycorrhizal fungi. Epidemiological processes in high-diversity communities may differ fundamentally from what we know from agricultural and managed-forest situations, and need to be explored both empirically and through modelling. And most critically, we need to incorporate both the beneficial and detrimental impacts of diseases into the management of tropical forests, and the design of forest preserves. No longer can we afford to view plant diseases as simply economic problems in agricultural systems, and plant-microbe mutualisms as alternatives to fertilizers. Microorganisms are the driving force behind too many important processes in tropical forest dynamics to stay invisible to ecologists.

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GLOSSARY

ascoma - sexual reproductive structure of Ascomycete fungi; produces ascospores borne in asci (see Fig. 1)

ascospore - sexual (meiotic) spore of Ascomycete fungi (see Fig. 1)

autotrophic - able to derive energy the photosynthesis or chemosynthesis

basidioma - macroscopic sexual reproductive structure of Basidiomycete fungi; produces basidiospores borne on basidia; examples are mushrooms and shelf fungi (see Fig. 1)

basidiospore - sexual (meiotic) spore of Basidiomycete fungi (see Fig. 1)

brown rot - fungal decomposition of wood where cellulose is degraded faster than lignin, increasing the relative concentration of dark-colored lignin in the remaining wood

clone - genetically identical individuals; in fungi may be derived from growth of hyphal fragments or germination of asexual conidia

coenocytic - property of hyphae that allows the movement of cellular contents, including organelles, among compartments; in True Fungi, crosswalls are semipermeable; in Oomycetes, truly coenocytic.

colony - collection of bacterial cells or fungal growth that originated from a single cell

commensalism - symbiosis where neither partner suffers or benefits from the association

conidia - asexual (mitotic) fungal spore produced by Deuteromycetes and during asexual reproduction of Ascomycetes and Basidiomycetes. (see Fig. 1)

constitutive - for plant defenses against disease, those defense always present, regardless of challenge by pathogens.

decomposition - microbially-induced breakdown of plant or animal material into simpler organic components

disease - negative disruption of the normal functions or form of a plant or animal, caused by a persistent agent (e.g. a parasite), leading to the impairment or death of the affected organism

endemic - native to and naturally limited to an area; for diseases, refers to the background levels of disease in an ecosystem when not undergoing rapid, epidemic disease spread

endophyte - fungi living inside apparently healthy living plant tissue; may be mutualists, commensals, or latent pathogens

epidemic - rapid increase in the incidence and severity of disease in a particular host population

facultative pathogen - a microorganism that can complete its life cycle as a saprotroph without causing disease, but which can cause disease under appropriate conditions

germination - growth of hyphae from a fungal spore; spores may require signals from the host plant or particular environ-

mental conditions to induce germination

heterotrophic - acquires energy through the consumption of other organisms or organic material.

host - the larger partner in a symbiosis with microorganisms, usually a plant or animal; in the case of infection by pathogens, the organism that suffers the diseases

hypha - fundamental tube-like unit of growth for fungi; functionally coenocytic for true Fungi; fully coenocytic for Oomycetes (see Fig. 1).

induced - disease defenses in the host plant or animal that are activated in response to infection by a pathogen or some other cue.

inoculum - source of infection of hosts plants by microbes; usually spores or hyphal fragments.

mutualism - a symbiosis that is mutually beneficial for the host and the microbial symbiont

mycelium - network of fungal hyphae (see Fig. 1)

mycology - study of fungi and fungus-like organisms such as Oomycetes.

mycorrhiza - a generally mutualistic symbiosis between plant roots and fungi; fungi receive carbon and the plant receives improved water and nutrition uptake.

obligate pathogen - a pathogen that can only grow or only complete its life cycle as a pathogen.

oospore - sexual (meiotic) spore of Oomycetes (see Fig. 1)

opportunistic pathogen - a pathogen that generally only causes disease on hosts that are already weakened or diseased from other agents.

parasitism - a symbiosis where the microbial symbiont benefits and the host is harmed.

pathogen - a microbial agent, usually a parasite, that can cause disease on its host.

phytoplasma - a group of small, wall-less bacteria that live in the vascular systems of plants and can cause diseases

resistance - defenses in plant hosts that prevent a pathogen from causing diseases; only definable with reference to a particular pathogen

saprotroph - a microbe that acquires its nutrition through the consumption of dead organic materials.

septa - cross-walls in fungal hyphae; they are not complete walls, and thus the hyphae are functionally coenocytic (see Fig. 1).

sign - in plant diseases, reproductive structures or other visible structures of the pathogen.

spore - primary dispersal and resting structure of fungi; one or more cells in a discrete unit - may be mitotic or meiotic products (see Fig. 1).

substrate - the organic material that a microbe lives in and consumes.

symbiont - may be applied to both members of a symbiosis, but usually refers to the smaller, microbial member of a symbiosis

symbiosis - intimate association between two species; "living together"

symptom - the expression by the host of a disease; can be necrosis, chlorosis, stunting, reduced yield, hypertrophic growth, etc.

thallus – the vegetative mycelium of a fungus

true fungi – organisms in the kingdom Fungi, including Ascomycetes, Basidiomycetes, Deuteromycetes, and Zygomycetes. Does not include the Oomycetes (water molds) which belong to the kingdom Protista.

vector - an animal, often an insect, that carries a pathogen to a new host

virulence - the relative ability to cause disease on a host plant; must be discussed in terms of the resistance of the host.

white rot - fungal decomposition of wood where lignin is degraded as fast or faster than the cellulose, decrease in the relative content of dark-colored lignin in wood through the decomposition process.

zoospore - motile, flagellated, asexual spore of oomycetes; swims through free water or water-filled pores in the soil.